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Contributors

Davidson, Andrew, 1836-1918.
London School of Hygiene and Tropical Medicine

Publication/Creation

Edinburgh : Pentland, 1893.

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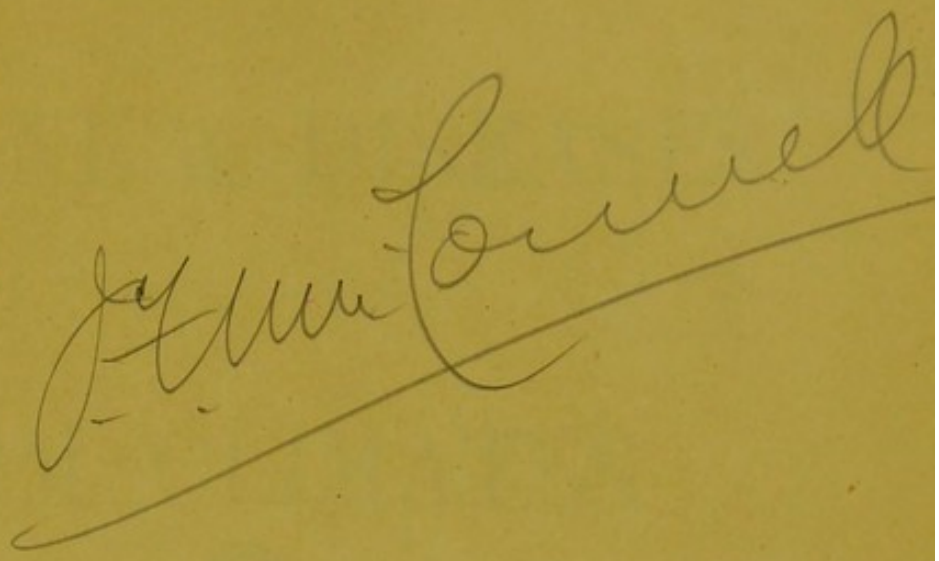
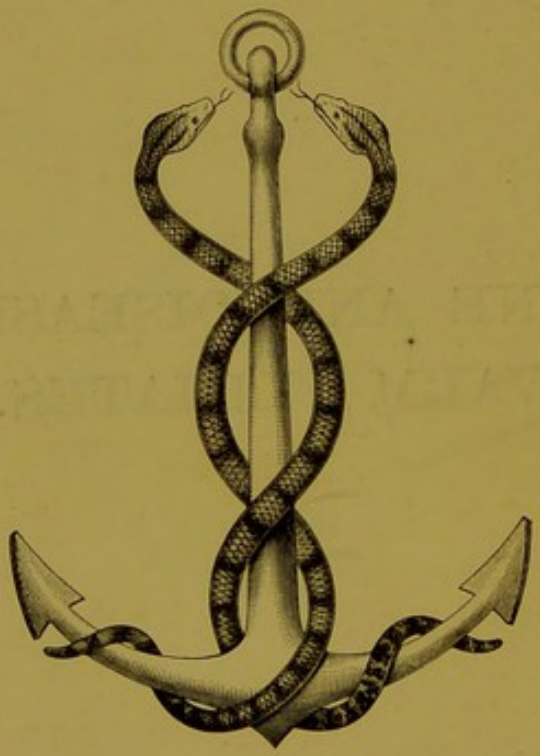


HYGIENE AND DISEASES OF WARM CLIMATES.



NUNQUAM ALIUD NATURA, ALIUD SAPIENTIA DICIT.

HYGIENE & DISEASES

OF

WARM CLIMATES.

EDITED BY

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ILLUSTRATED WITH ENGRAVINGS AND FULL-PAGE PLATES.

EDINBURGH AND LONDON:
YOUNG J. PENTLAND.
1893.

EDINBURGH: PRINTED FOR YOUNG J. PENTLAND, 11 TEVIOT PLACE,
AND 38 WEST SMITHFIELD, LONDON, E.C., BY MORRISON AND GIBB.

PREFACE.

THE design of this work is expressed in its title. The need for it is proved by the fact that no work professing to cover the whole field of tropical hygiene and pathology has hitherto appeared in our language. The classical treatises of Annesley, Morehead, Martin, and Chevers remain of permanent value, but they do not adequately represent the science and practice of the present day.

How far I have succeeded in filling up the blank that exists in this department of medicine, must be left to the judgment of the profession; but the co-operation of so many distinguished men, recognised as authorities on the subjects which they have undertaken, justifies the hope that this work will not disappoint the expectations of the teacher, student, or practitioner.

In selecting the subjects, and deciding upon the space to be allotted to each, I have been guided by their relative importance to the medical practitioner, rather than by considerations of their scientific interest, although these have not been ignored. The three dominating endemic maladies of warm climates—malarial fever, dysentery (including tropical diarrhœa), and hepatitis,—and the three great pestilences—cholera, yellow fever, and plague—have been treated in that detail which their importance demands. Leprosy and beriberi have also seemed to require particular attention; the former from its wide diffusion in the East; and the latter, not only on account of its gravity, but from the numerous interesting and, as yet, unsolved problems connected with its etiology, pathology, and treatment. All of these diseases are more or less unfamiliar to those educated at home, and most of them will probably be seen for the first time by the medical officer when he commences to practise in the tropics, and it may be safely said that they will form more than three-fourths of the cases he will be called upon to treat.

It is only within recent years that the *rôle* of parasitic diseases in tropical pathology has been fully understood. The responsibility for

this important class of diseases has been devolved on Professor Sonsino of Pisa and Dr. Manson of London. Those who read the articles on filaria disease, Bilharzia disease, and anchylostomiasis, will require no apology for the space which has been allotted to them.

I have gratefully to acknowledge my obligations to those who have rendered me their invaluable assistance in this undertaking. Some of them have done so under the pressure of other duties, in order to place their experience at the service of their younger professional brethren. The heavy labours devolving on an editor have been lightened by the uniform courtesy and consideration I have received from the contributors.

ANDREW DAVIDSON.

LASSWADE, *October* 1893.

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CONTENTS.

HYGIENE.

CHAPTER I.

CHAPTER II.

CHAPTER III.

GENERAL DISEASES.

CHAPTER IV.

MALARIAL DISEASES.

By Andrew Davidson, M.D., F.R.C.P.Ed.

CHAPTER V.

TROPICAL TYPHOID FEVER.

By H. R. Whitehead, F.R.C.S.

CHAPTER VI.

MALTA FEVER.

By David Bruce, M.B.

CHAPTER VII.

YELLOW FEVER.

By George M. Sternberg, M.D.

CHAPTER VIII.

DENGUE.

By Andrew Davidson, M.D., F.R.C.P.Ed.

CHAPTER IX.

PLAGUE.

By Montagu Lubbock, M.D., F.R.C.P.

CHAPTER X.

CHOLERA.

By N. C. Macnamara, F.R.C.S.

CHAPTER XI.

LEPROSY.

By N. C. Macnamara, F.R.C.S.

CHAPTER XII.

BERIBERI.

By Patrick Manson, M.D., M.R.C.P., LL.D.

CHAPTER XIII.

NEGRO LETHARGY.

By Patrick Manson, M.D., M.R.C.P., LL.D.

CHAPTER XIV.

FRAMBŒSIA OR YAWS.

By William T. Prout, M.B., C.M.

LOCAL DISEASES.

CHAPTER XV.

TROPICAL DIARRHŒA.

By Sir Joseph Fayrer, K.C.S.I., LL.D., M.D. Q.H.P., F.R.S.

CHAPTER XVI.

DYSENTERY.

By D. M. Davidson, M.B., C.M., and Andrew Davidson, M.D., F.R.C.P.Ed.

CHAPTER XVII.

TROPICAL DISEASES OF THE LIVER.

By Henry Cayley, F.R.C.S.

CHAPTER XVIII.

TROPICAL LIVER ABSCESS.

By Sir Joseph Fayrer, K.C.S.I., LL.D., M.D., Q.H.P., F.R.S.

CHAPTER XIX.

SUNSTROKE.

By Sir Joseph Fayrer, K.C.S.I., LL.D., M.D., Q.H.P., F.R.S.

CHAPTER XX.

GOITRE.

By Francis Nottidge Macnamara, M.D.

PARASITIC DISEASES.

CHAPTER XXI.

THE *FILARIÆ SANGUINIS HOMINIS* AND FILARIA DISEASE.

By Patrick Manson, M.D., M.R.C.P., LL.D.

CHAPTER XXII.

DISTOMUM RINGERI VEL PULMONALE.

By Patrick Manson, M.D., M.R.C.P., LL.D.

CHAPTER XXIII.

INTESTINAL, HEPATIC, AND PORTAL ENTOZOA, AND ASSOCIATED DISEASES.

By Prospero Sonsino, M.D., Pisa.

SKIN DISEASES.

CHAPTER XXIV.

DISEASES OF THE SKIN IN TROPICAL CLIMATES.

BY PATRICK MANSON, M.D., M.R.C.P., LL.D.

APPENDIX.

LIST OF ILLUSTRATIONS.

I. PLATES.

II. FIGURES IN TEXT.

HYGIENE AND DISEASES OF WARM CLIMATES.

HYGIENE AND DISEASES

OF

WARM CLIMATES.

CHAPTER I.

THE INFLUENCE OF WARM CLIMATES ON THE CONSTITUTION.

BY EDWARD A. BIRCH, M.D., F.R.C.P.

Evidences of the Influence of Warm Climates on the Constitution.—The evidence that residence in warm climates produces very appreciable effects upon the human constitution is ample. A study of the comparative mortality which prevails in temperate and tropical climates renders it certain that residence in the tropics has an adverse effect upon Europeans, under the conditions of ordinary life. Guy's oft-quoted table of the death-rate at the soldiers' age brings this out very prominently. Selecting men of various occupations, he showed that while only 7 per 1000 of the members of the London Fire Brigade succumbed annually, the British soldier in Bengal died at the frightful rate of 63 per 1000. He also instituted a number of intermediate comparisons; for instance, the London Police force yielded 7·6, and agricultural labourers of similar age in England, 8·0 deaths per 1000. It is unnecessary to quote all his figures, especially as they apply to a bygone time, when the mortality of the soldier, and, indeed, of all classes in India, was very much higher than it now is. Since Guy wrote, the Indian death-rate has declined by quite two-thirds; yet this enormous reduction still leaves a balance against India of more than double as regards the soldier. This point is shown very plainly by a study of the statistics of later periods.[1] Thus, taking

[1] Parkes and Notter, *Practical Hygiene*, 8th ed.

the ten years ending 1880, the gross male civil mortality at the soldiers' age in England was—

From 20 to 25 years = 7·32 pro mille.
„ 25 „ 35 „ = 9·30 „ „

If we confine ourselves to the still later year 1888 alone, these rates are altered to 5·4 and 7·2 respectively; further, we find that the soldiers' ratios in England stood in that year at 4·70 and 8·11 for these ages, thus presenting a favourable comparison. Turning next to the soldiers' death-rate in India for 1888, the mortality was as follows in Bengal:—

20 to 25 years = 14·75 } or, excluding { 11·32
25 „ 35 „ = 16·07 } cholera, } 9·74

The mortality among the women of British regiments is even greater than that of the men, especially from fevers, dysentery, cholera, and phthisis. It has not been found possible to discover any method by which the mortality or sickness of the civil European population of India can be compared with that of England. It may, however, be remarked that the general health among the indigo planters of Tirhoot, which is a dry, though at times very hot, climate, is good. They expose themselves at all seasons; and, although they take a great deal of exercise in the sun, they retain more of their healthy European appearance than the settlers in other parts of India. On the other hand, the tea planters of Bengal (excepting those resident in the hills) can but be characterised as sickly in appearance. Tea, however, requires a humid and hot climate, one where malaria is usually rife, and all the surrounding influences are adverse to health.

We possess data of a valuable kind regarding children. It may be briefly stated, without going too minutely into figures, that under 5 years of age soldiers' children die in India at the rate of something like 140 per 1000, while the English death-rate for this period is about 58 per 1000, or less than half; and under 15 it is about one-third of the Indian death-rate. The accuracy of the following table, drawn up by Dr. Townsend, has never been impugned, though it must be admitted that later years exhibit better results:—

	England. The Mean of 29 Years.	Bengal. Soldiers' Children, 1870.
Under 5 years, . . .	67·58	148·10
From 5 to 10 years, . . .	8·80	17·73
„ 10 „ 15 „ . . .	4·90	11·51

In 1878 the death-rate of soldiers' children under 1 year in India was 314 per 1000, while in London it was 185.[1]

Viewing the mortality in relation to the birth-rate, we find that in England about 15 per cent. of all infants born die within the first year of life. There is but little evidence as regards children in India in this particular, but the following may be quoted from the *Calcutta Review* of 1851: "Taking the returns of two regiments which reached India last year, we find that in one there have been born 44 children, of whom at the end of the fifteenth month there are only 29 surviving, showing a loss of 27 per cent. within the first year. In another regiment 52 children have been born within 14 months, of whom 32 have died within the same period, giving a ratio of mortality equal to 33 per cent. during the first twelve months of their Indian life. In another case, taking the children born in England or on board ship who arrived with the regiment in India eight years ago, out of 159 (the original number) no less than 110 have perished. Of the remaining 49, how few in all probability will grow to manhood! Hence we see that whether we take 100 children imported from England, born of healthy parents, or 100 children born of the same parents within the first year of their arrival in India, still the melancholy fact remains the same." The same writer quotes the following table, exhibiting the respective ages of the survivors of 261 children born in one regiment since landing in India eight years previously:—

From 7	to 8	years,	. .	4
„ 6	„ 7	„	. .	8
„ 5	„ 6	„	. .	13
„ 4	„ 5	„	. .	15
„ 3	„ 4	„	. .	20
„ 2	„ 3	„	. .	15
Under 2		„	. .	38

Died, 148; survivors, 113. Total in 8 years, 261.

It may be true that the management of soldiers' children is bad; but have we any approach in India to the want, privation, and exposure to which multitudes of the children of the poor in London are subjected? Surely not.

[1] The death-rate of soldiers' children in India has declined very considerably since 1878. In 1888, ten years later, the death-rate of children under 1 year was 189·64. This very marked decrease is the result of circumstances which add weight to the author's reasoning and conclusions. They are—(1) short service in the country; (2) rapid means of transit to the hills, and a larger proportion of men quartered there; (3) houses established in the hills for the reception of children; (4) the larger number of married families sent to the hills during the hot season. In other words, the diminished mortality is the result of withdrawing soldiers' children from the tropical influences which prove so injurious, and rearing them in a temperate climate.—EDITOR.

The foregoing considerations would seem to lead to a perfectly reasonable deduction that, under the circumstances of ordinary life, a tropical climate (of which India is a type) is inimical to the European constitution.

But it will not be altogether fair to leave this subject without one or two further observations. It should be noted that it is very questionable how far any of the results above quoted are due to climate *per se*. The enormous and progressive reduction which has been effected in the mortality of soldiers in India has been alluded to; unfortunately we cannot boast that the efforts to ameliorate the condition of their children have been attended with similar success; but the investigations of Macpherson and Fayrer have proved that, under carefully considered hygienic conditions and physical training, the European child may live and thrive in the plains of India "almost as well as in its native country." Sir R. Martin's opinion was that, though with care the European child may be reared in India up to 5 or 6 years, beyond those ages a physical and moral degeneration occurs; the child then "exhibits the necessity for change of climate by emaciating and outgrowing its strength"; and he considered the attempt to rear children up to and past youth as an "altogether cruel and impracticable endeavour." Even those who are kept in India only till 5 or 6 "exhibit a restlessness and mobility of the nervous system—a busy idleness—beyond their age, as compared with the habits of children of the same ages born and bred in England. There is also a marked disposition to relaxation, and to a loose, relaxed state of the joints in such children, and to consequent lateral curvature of the spine." Fayrer sums up the whole question plainly and correctly. He says, "I have no desire to prove too much, as I certainly should appear to attempt to do were I to advocate the theory that Calcutta, or any other part of the plains of India, is a *desirable* locality for the training and nurture of European children; such, indeed, would be a theory as dangerous as false. For although the exceptionally favourable circumstances of the European Female Orphan Asylum prove that the European child may thrive, yet it is certain that without favouring influences it will not; and the statistics of infant life in the British army in India prove not only that such is the case, but that the obstacles to success in the rearing of children are very great. It has long been known to the English in India that children may be kept in that country up to 5, 6, or 7 years of age without any deterioration, physical or moral, and in the higher classes of life with probably as little, if not less, danger to life than in England; for most assuredly in some respects—as, for example, scarlatina,

measles, whooping-cough, thoracic complaints, and even dentition—they suffer less in India than in England. But after that age, unless a few hot seasons spent in the hills should enable parents to keep their children in India until a somewhat later age, to do so is always a doubtful proceeding. The child must be sent to England, or it will deteriorate physically and morally,—physically, because it will grow up slight, weedy, and delicate, over-precocious it may be, and with a general feebleness not perhaps so easily defined as recognised, a something expressed not only in appearance, but in the very intonation of the voice; morally, because he learns from his surroundings much that is undesirable, and has a tendency to become deceitful and vain, indisposed to study, and to a great extent unfitted to do so,—in short, with a general tendency to deterioration which is much to be deprecated, and can only be avoided by removal to the more bracing and healthy (moral and physical) atmosphere of Europe." Infant mortality is small among the children of the civilian Europeans of Calcutta, large among those of the Eurasians, and very large among those of the natives, being higher among the Mohammedans than the Hindus. "The treatment of the child in the first twelve months either destroys his life or leaves indelible traces on his future existence," observed Farr. That European children die in Calcutta in small numbers, means simply that they are subjected to exceptionally watchful care; and the mixed races hold an intermediate place because of the admixture of native habits which prove so disastrous to the Bengalis, and concerning which much that is sensational but absolutely true has been written.

Taking the percentages of all deaths of soldiers' children from 1872 to 1876, the causes were as follows:—

Under 6 Months.	6 to 12 Months.	1 to 2 Years.
Convulsions, . . 29	Bowel Complaints, . 27	Bowel Complaints, . 31
Anæmia, . . . 25	Dentition, . . 21·5	Dentition, . . 16·5
Bowel Complaints, . 18	Convulsions, . . 16	Convulsions, . . 11
Croup and Bronchitis, 7	Anæmia, . . . 8	Anæmia, . . . 9
Fevers, . . . 5	Croup and Bronchitis, 7	Croup and Bronchitis, 8
Etc. etc.	Fevers, . . . 6	Tabes mesenterica, . 6·5
	Etc. etc.	Fevers, . . . 4
		Measles, . . . 5
		Etc. etc.
2 to 3 Years.	**4 to 7 Years.**	**Above 7 Years.**
Bowel Complaints, . 24·5	Cholera, . . . 18	Cholera, . . . 31
Cholera, . . . 12	Fevers, . . . 16·5	Fevers, . . . 17·5
Fevers, . . . 11	Croup and Bronchitis, 16	Croup and Bronchitis, 10
Croup and Bronchitis, 10	Bowel Complaints, . 10	Bowel Complaints, . 8·5
Measles, . . . 8·5	Convulsions, . . 10	

The only conclusion that can be arrived at from these facts is that extraordinary care is required in India to attain results which are obtained in England under conditions of life largely vitiated by neglect and poverty; and that, if the want of care which prevails in England be imported into India, a very terrible European child-mortality becomes inevitable. A tropical plant may be reared and even thrive in England in a hothouse, but it will die if placed under the ordinary conditions of plant life in that country.

The adult cannot be treated or treat himself with a total disregard to all circumstances, save those of health, throughout each hour of the day and night. Even were this possible, statistics, observations, and authoritative opinion justify the conclusion that, "after making fair deduction for the effects of errors of life, there remains a certain proportion of disease, by no means a small one, occurring among persons of well-regulated life, which can only be attributed to the effects of climate" (C. T. Williams).

A tropical climate, foreign to another race also of tropical origin, frequently has very marked effects upon the imported people. Of this we have numerous instances. Bengal Sepoy regiments are recruited in the North-Western Provinces and Punjab, which possess climates very different from that of Lower Bengal; the men invariably become sickly and more or less scorbutic when serving for any length of time in the latter province. On this point the late Dr. Bryden wrote: "The native soldier of India is a foreigner in many districts of the Bengal Presidency in which he is called on to serve. Stationed beyond the Indus, his admission-rate and death-rate much exceed the average for the European army as a body; and at the opposite extreme of the Presidency the ratios are not less than on the North-Western Frontier. From Behar to the Indus may be regarded as the limits within which the Sepoy finds a climate suited to him, or approximating to what is natural to his requirements as a native of Upper India." About 8 per 1000 may be taken as the normal annual death-rate for this area; in Bengal Proper and Assam it reaches 18, and trans-Indus it is about 17, per 1000. The Bengali civilian who migrates to Assam suffers more from malarial diseases and anæmia than in his own malarial climate. The African ceases to thrive in India. In the latter instance the contrast of the climates and surroundings is particularly great. Henry Marshall, writing in 1821 of the Africans who composed part of the troops in Ceylon, says that that island "appears to have been extremely unfavourable to the health and propagation of the Caffres. Not a trace of the many thousands brought to it by the Portuguese Colonial Government is to be perceived. The

same may be said of a colony of Africans which was imported about the year 1782 by Governor Van de Graaf. . . . With a view to promoting the successive production of Africans in Ceylon, a large proportion of females was always imported with the males. The females were allowed a ration of provisions gratis, and each male child had a ration allotted to him from the day he was born. These prudential measures have, however, availed nothing; almost all the children born of African parents expire before they reach 10 or 12 years of age. During infancy they are plump and healthy; but by the time they reach 5 or 6 years of age they droop, become meagre, and generally die before the age of puberty. . . . The children of indigenous females, by Caffre fathers, are as liable to disease, and thrive as badly, as the descendants of Africans." And, further, it is pointed out that these results are due neither to neglect on the part of the parents nor to hardships. The curious thing here is, that the dangerous period of infancy would seem to have been successfully tided over, and that climate appeared to have had the predominating, if not exclusive, influence. Another remarkable instance is to be found in the fact that black troops in Jamaica suffer from a pulmonary phthisis mortality of 8·67 per 1000, when the whites are hardly at all affected by tubercular disease.

The effects of a tropical climate upon the European are so insidious that it is not from the death-rate that we glean the best idea of the general deterioration of health which is in progress. There are three climatic factors present, which tend to the production of a slowly advancing asthenia and cachexia which almost imperceptibly sap the constitution, even if the individual escapes the attacks of one of the acute diseases of which they are so often the exciting cause. These factors are—(*a*) a long continued high temperature; (*b*) the presence of malaria; and (*c*) the great diurnal variations at certain seasons. The operation of these influences is constant, producing an excessive cutaneous action, alternating with internal congestions; nor is it possible that any individual can altogether escape them. The natural pathological consequences ensue in the form of deterioration of the blood, and those degenerative and other changes, the usual resultants of hyperæmia; and the liver, kidneys, and intestines become incompetent to perform their eliminative functions adequately. Thus it is that actual disease, which in the asthenic is always obscure in its advance, frequently steals unawares upon the constitution.

Influence of Warm Climates on the Physiological Functions.—*Temperature.*—The influence of tropical heat and residence upon the natural temperature of the body is an important

point, which the accurate and extended observations of Dr. A. Crombie,[1] of Calcutta, have placed upon a certain footing. That observer found that the mean diurnal difference of temperature in the axilla, mouth, and rectum are practically the same in Europeans and Indians, and that they are in close accordance with English results (between the axilla and mouth the difference is 0°·24 F., and between the mouth and rectum, 0°·4 F.; wherefore the rectal heat may be said to be 0°·64 F. higher than that of the axilla). Accepting the observations of Ogle, Allbutt, Casey, and Rattray as being scientifically correct with reference to temperate climates, Crombie proceeds to compare their results with those obtained by himself in India. As these investigations extended over a period of eighteen months, were periodically made hourly and during each month of the year, under stringent precautions, they may be accepted as absolutely trustworthy and of great value. The diagram (Fig. 1) represents his conclusions, and their comparison with those of the English authorities named. The four lower curves depict the English daily range; the two upper show Dr. Crombie's own observations, the highest representing the average for natives, and the line below it the average at each hour for Europeans, the latter made under the tongue. It will be observed that the average temperature of the native is quite 0°·5 F., and, indeed, at noon, nearly 1° F. higher than that of the European. It is, however, necessary to mention that the Indians' temperatures were taken in the axillæ (of fifty-five healthy individuals), while those of the European are mouth records. This was necessary on account of caste prejudices, forbidding anything to enter the mouth which had been touched by a Christian, Mohammedan, or Hindu, as the case might have been; but a correction was made by adding 0°·2 F. to the axillary temperatures.

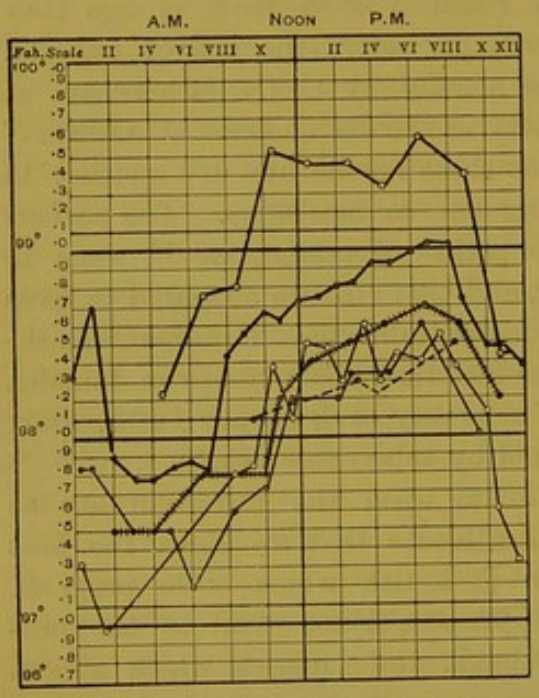


Fig. 1.—Showing the Daily Range of Temperature of European Observers,[1] and of Europeans living in Bengal (●——●), and of the Natives of India (⊙——⊙).

[1] European Observers—
Ogle, ●——●. Casey, ⊙——⊙.
Allbutt, ●·····●. Rattray, ●-·-·-●.

[1] *Indian Annals of Medical Science*, No. xxxii.

The mean mouth temperature of the European (calculated from 1288 observations, the average of each hour of the twenty-four) was found to be 98°·49 F.; and Crombie, comparing his deductions with those of the four selected English physicians, obtained the following ratios:—

	Mean A.M. Temperature.	Mean P.M. Temperature.	Mean Temperature.	Max. Daily Range.
English averages,	97°·763	98°·341	98°·084	1°·41
Crombie's Indian averages, .	98·21	98·77	98·49	1·31

These figures show that the body temperature of the European living in Bengal is about 0°·41 F. higher than the average of healthy persons in England; and they confirm Rattray's observations in the tropics and England, as well as the law laid down by Becher, that the body heat increases in the proportion of 0°·05 F. for every 1° F. increase of the air. Similarly, according to Wunderlich, Brown-Séquard, on a journey from Mauritius to France, found that eight healthy people, when the external temperature was 46°·4 F., had a mean temperature under the tongue of 97°·9 F.; but a week later, when the air had risen to 77° F., the body heat was 99°·4 F.

It would seem to be well established that the European newly arrived in the tropics exhibits a somewhat higher temperature than he who has been exposed to the debilitating influences of climate for a year or two. In the former case the vital functions may be presumed to be working vigorously, but more or less after the European model, the skin not having had time to assume to the full its vicarious functions, and not being yet capable of counterbalancing the great additional external heat. Indeed, it is a common observation that the old resident perspires more freely than the new arrival.

Alluding to the peculiarities of the highest curve in the diagram, Crombie remarks that "the high average temperature of natives between 10 and 12 A.M. is probably the consequence of their habit of taking one of their two enormous meals between 9 and 10 A.M.; and the absence of a marked rise in the evening, which gives a somewhat different character to their mean daily curve, is the consequence of their temperatures having always been taken while they were at rest. These observations were made in August 1873," and he concludes that the temperature of natives may therefore be considered to lie between 98° and 99° F. from 10 P.M. to 10 A.M., and between 99° and 100° F. between 10 A.M. and 10 P.M. It may,

however, be remarked that during August in Lower Bengal the humidity of the atmosphere is almost at saturation point, and evaporation nearly suspended; and the writer has met several observant physicians who question the universal applicability of Crombie's conclusion on this particular point. More extended observations, and at varying periods of the year, are required.

Dividing the day into four natural periods, the daily fluctuations of the temperature of the European in Bengal show remarkable uniformity with the English averages, except that the curve for temperate climates occupies about half a degree lower. In Bengal Crombie found them to be—

From 2 A.M. to 7 A.M. = 97°·83 F.
„ 8 „ to 1 P.M. = 98°·61 „
„ 2 P.M. to 8 „ = 98°·92 „
„ 9 „ to 1 A.M. = 98°·54 „

The effects of food and exercise in the tropics are the same as in temperate climates, with the exception that the latter seems to act more immediately and powerfully, a very slight amount of exercise producing a marked elevation, which the body is prone to maintain for a space of some hours.

Sleep invariably depressed the temperature, whether during the day or night, or at the period of normal increase or otherwise.

Respiration.—All authorities now admit the correctness of Rattray's deductions, that (*a*) the capacity of the chest for air is considerably greater in the tropics than in temperate climates; and (*b*) that the frequency of the respirations is diminished. Furthermore, it has been fully established (*c*) that the respiratory act as a whole, notwithstanding the contending nature of the above physiological facts, is lessened; "the increased quantity of air and oxygen inspired in the tropics does not make up for the diminished number of respirations in supplying the same amount of air and oxygen for blood purification as in cold climates, though doubtless a requisite quantity is inspired, less probably being needed there to carry on the vital processes."[1] The spirometric increase is considerable, amounting to 7 or 8 per cent. or more. Assuming that 15 cubic inches are utilised in England at each respiration, and that there are seventeen respirations per minute, the total consumption will be 255 cubic inches; in a warm climate the amount used at each respiration will be greater, say 16½ cubic inches, but the number of respirations will be lessened, say to fourteen, and this would yield a total of 231 cubic inches, that is, twenty-four (or over 9 per cent.) less than were required in England. But there remains yet another factor to be

[1] *Proceedings Royal Society*, London.

taken into account: (*d*) a reduction, averaging 3½ or 4 per cent., has to be applied to meet the obvious fact that heated air contains less oxygen per cubic foot (air expanding $\frac{1}{480}$ of its volume for each rise of 1° F.), so that the number 231 is diminished to 222½ or thereabouts.

Finally, as the amount of carbon thrown off by the lungs in any climate bears a ratio to the quantity of air inspired, it follows (*e*) that the lung elimination of carbon undergoes a considerable diminution in the tropics; something in excess of an ounce will, under the ordinary circumstances of either climate, represent this loss (10 oz. daily being about the English standard).

How are we to explain the increased capacity of the chest in the tropics, and its decline upon return to a cold climate? The idea that it is caused by the efforts of the chest to accommodate the respiration to the lessened quantity of oxygen in a given volume of air cannot be entertained, because we know that the amount of oxygen obtained is actually less. The explanation which is stated in detail by Rattray, and now generally accepted, lies in the fact that the lungs contain less blood, and that therefore more room is allowed for air. The capacity is really the same; it is merely a matter of the relative quantities of blood and air occupying the space at disposal, and this is regulated by the diversion of the blood to the surface or internal organs, as the case may be, under the influence of the external temperature to which the body is subjected. An accommodatory relative fluctuation, essential to the perfect working of the cooling apparatus on the one hand, and of the respiration on the other, is thus maintained in accordance with the requirements of the varying climates. Rattray estimates that 23 oz. represents the total withdrawal of blood from the lungs under the influence of an average temperature of 80°–83° F.

Francis has recorded the fact, derived from numerous autopsies of European bodies in India, (*f*) that the lungs are lighter than the European standard. This would now appear to be accounted for by the alteration in the relative quantities of blood and air contained in these organs.

With a lessened supply of blood and air it is obvious (*g*) that the watery vapour discharged from the lungs is diminished in the tropics; but the proportion, of course, depends largely upon a variety of coincident circumstances, the chief of which is the humidity of the atmosphere. Rattray sums up thus: the vascularity of the adult lungs is reduced by 12 or 13 fl. oz.; their spirometric measurement by an average of 32 inches; their function by 18·43 per cent., that is, they use 36·85 cubic feet less of air daily; the excretion of 1·84 oz. less of carbon and 6·7 less of watery vapour.

Pulse.—Formerly it was thought that the pulse-rate was accelerated by tropical heat, but the statement is incorrect; it would seem rather that it is somewhat slowed by heat, and that the cardiac pulsations are probably less forcible. We should expect that the latter view would be correct, and that with a slower respiration we should have a slower pulse; but it does not seem that there is that proportionate slowing which might be expected. The difference, however, if any, is very slight. Talking of the West Indies, Armstrong[1] stated that Europeans are capable of more work there than the muscular negro; "in the intervals of rest, the pulse was repeatedly examined in twenty or thirty black men and the same number of seamen and marines, and in the former it was invariably slower and weaker;" but probably the Europeans were full of fresh British energy, or were putting on a "spurt," a point not alluded to.

Urine.—The urine diminishes greatly in a warm climate; but its quantity is largely influenced by the degree of heat, the amount of fluid ingesta, and the humidity of the air. "A sudden increase of the moisture of the atmosphere seems to produce as considerable a modification in the bodily functions as a sudden increase of temperature. Less moisture being lost by the skin and lungs, more will have to be given off by the kidneys and intestines. Polyuria and diarrhœa are, therefore, of by no means infrequent occurrence (Stewart, Hirsch, Thomas, Rohden); and if a sufficient quantity of water be not removed in this way, a transitory increase of the liquid constituents of the blood seems likely to result. Rohden ascribes to this circumstance the more frequent occurrence of pulmonary hæmorrhage at times when the moisture of the air is suddenly increased."[2] In this way, too, is accounted for the otherwise curious absence of thirst during those days in the tropics when the air is saturated and still, the heat great, and the sky cloud-laden. Under these conditions the cold bath has lost its charms; it brings no relief beyond the temporary removal of the greasy perspiration. A slightly-heated bath is more desired and desirable; the feeling of the return of the blood to the damp chilled skin is enjoyed. A proportionate increase and decrease is carefully observed by these organs and the lungs. Any sudden chill, or the unguarded effects of cold from sudden change of climate, may be attended by grave consequences in the form of a disturbance of this equilibrium, and terminate in serious congestions, to which the kidneys and the liver more especially are liable. Rattray calculates that nephritic vascularity and secretion are reduced by 17½ per cent. as a general average in

[1] *The Influence of Climate on the Human Constitution*, 1843.

[2] Hermann Weber, *Ziemssen's Handbook of Therapeutics*, vol. iv. p. 38.

a warm climate; but that notwithstanding their greatly reduced function, they still always remain the chief eliminators of watery fluid. He argues that the diminished kidney excretion (17½ per cent.) and loss of watery vapour by the lungs (4½ per cent.), almost exactly balance the amount of blood withdrawn from the lungs on the one hand, and the increased skin secretion (24 per cent.) on the other.

Blood.—The effects of a warm climate upon the blood cannot be so easily gauged. The general tendency, especially among the young, is towards anæmia; but accurate observations are still wanting. In attempting to form an opinion, it is difficult to eliminate the part played by that blood-destroyer, malaria. Perhaps it is natural that we should expect some development of anæmia, because the organs are not so well stimulated by bodily exercise as formerly; and it may be urged that neither are they so well physiologically nourished, the appetite being diminished and metamorphosis slower, the blood itself undergoing deterioration as well as the tissues. Repeated estimations of the corpuscles have led the writer to no other conclusion than that anæmia is much more common in India than in England, but it is impossible to eliminate numerous extraneous causes. Martin held that the excessive perspirations have the effect of rendering the venous blood unnaturally dense; that "the systemic circulation becomes decidedly venous" because the blood is less arterialised, and that thus resulted imperfect oxidation and elimination. This really amounts, when viewed by the light of modern physiology, to asserting that the vital functions are less vigorously performed, which is admitted by all.

Yet it must be allowed that if the tendency be towards anæmia, this condition will impede oxygenation, nutrition, and sanguification as secondary results.

Nervous System.—The older observers maintained that the nervous system is at first, and for a short period, exalted in function, but subsequently depressed during tropical residence. It is probable that this is true. The increase of bodily temperature, which, as already stated, affects new arrivals, may account for the exaltation, the depression being coincident with the acquirement of the permanent standard; and these again possibly have an intimate relation to the periods before and after the attainment by the system of that power of vicarious blood distribution which is essential to the maintenance of healthy functions in a warm climate. The vasomotor system will share in the general depression or relaxation, and the deeply seated viscera, which are protected, will therefore be specially liable to congestions when cold affects the surface of the body. The disturbance of sleep in very

hot weather cannot but seriously interfere with the recuperation of the nervous system after a long and weary tropical day's work.

It would not appear unreasonable to attribute to the chemical and physiological effects of sunshine some of the part played in the earlier exaltation of nervous function, before there has been time for the depressing effects of constant heat to develop the opposite condition; but nothing is known upon this point.

Digestion.—The digestive powers are lessened, the process is slower. There is less desire for animal food on the part of the adult, but the child develops an abnormal craving for an animal diet. The reason of this is not very apparent. It would appear to be natural that the carbohydrates should be less desired in the effort to avoid further heat formation, and the albuminous constituents are not essential in large quantity because muscular action is so greatly in abeyance. Parkes observes that it is not, in fact, yet known what amount of lessening of food, or what kind of lessening, the increased heat of the tropics demands; all will, however, agree with his remark that spirits are most hurtful, and that even wine and beer must be taken in great moderation. In very hot weather total abstinence is the best rule, at all events no alcoholic beverages should be tasted till after the evening meal and the sun has disappeared. The use of vegetables and fruits is most essential, more so than in temperate climates, to counteract the tendency to scurvy, which is frequent. The notion, promulgated by the older writers, that the biliary secretion is augmented, is now held to be erroneous.

Nutrition.—As a rule, the bodily weight is diminished by tropical residence; and the muscular system becomes deficient in tone, as the result partly of the withdrawal of blood by which its nutrition is rendered slower, and partly because it is less exercised. With a slower digestion, a lessened appetite, and a feebler circulation, diminished lung work and blood oxygenation, and therefore less perfect nutrition, a generally relaxed state, a diminished necessity for surplus fat which is absorbed, and a tendency to poverty of blood, we have the natural consequences of lessened bodily vigour and loss of weight. Rattray has shown that in youths a tropical climate interferes with the normal development of the body, though growth in height is rather augmented.

Menstruation.—The advent of menstruation is largely influenced by warm climates. The following table exhibits the results of 561 observations concerning European and Eurasian girls who were either born in India, or who, having come to the country during their early childhood, were exposed to the full influence of climate; and these again are compared with the observations of Dr. Thomas More Madden in England. Twenty-seven of the number were investigated

by Fayrer in the European Orphan Asylum of Calcutta, and the remainder (534) have been collected, at the writer's instance, by Dr. K. N. Das, registrar to the Medical College Hospital, Calcutta, with scrupulous care, availing himself of the ample opportunities afforded him in his connection with the Eden Hospital:—

Class.	Observer.	Ages.									Totals.
		10	11	12	13	14	15	16	17	18	
English girls in England, . .	Madden,			17 3·6	50 10·4	94 19·6	138 28·8	105 21·9	65 13·6	10 2·1	479 %
European girls under Indian climatic influences,	K. N. Das,	1 ·9	8 6·9	15 12·9	21 18·2	35 30·2	15 12·9	15 12·9	5 4·2	1 ·9	116 %
Ditto.	Fayrer,			4 14·8	8 29·6	9 33·3	5 18·5	1 3·8			27 %
Eurasian and East Indians, . .	K. N. Das,	2 ·48	23 5·5	78 18·6	117 28·0	112 26·8	62 14·8	17 4·07	4 ·95	3 ·72	418 %

In addition to the above, Madden tabulates fourteen as having commenced to menstruate "above 18" years of age, and four "below 12."

It will be observed that Dr. K. N. Das' examples are divided into two groups, viz. those of European and those of mixed parentage. Comparing Madden's figures with Dr. Das' first group, some remarkable points are brought out. It appears that in England by far the largest percentage (50·7) commence to menstruate during their fifteenth and sixteenth years, and that in India nearly the same average (48·4) is attained during the thirteenth and fourteenth years by English girls who have been brought up in that country. The highest point is reached by the former in their fifteenth, and by the latter in their fourteenth year.

The proportion who attain physiological puberty in England during their seventeenth year is nearly the same as that of those who menstruate in India for the first time at 15, while at the latter age the figure for India is less than half that for England, and at 17 it is below one-third. Nearly 7 per cent. of the Anglo-Indian girls menstruated during their eleventh year, while Madden shows less than 1 per cent. "below 12." Although Fayrer's cases number only twenty-seven, his results bear a very close resemblance to those of Dr. Das, the maximum in either case being attained at 14, after which there is a sudden decline. It further appears that the children of Eurasian or East Indian [1] parents closely follow the

[1] These terms are used conventionally to signify the less and the darker-skinned respectively. The term "Anglo-Indian" is used to designate children of European parentage domiciled in India from birth or early childhood.

Anglo-Indian model, though their maximum average is attained somewhat earlier, and the twelfth year of the Eurasian corresponds with the thirteenth of the Anglo-Indian, while the decline of the fifteenth year is maintained, after which it is very much more sudden, being one year in advance of the latter class.

In the following table Dr. K. N. Das tabulates the results of 913 investigations regarding children of purely Indian parentage, as well as a few of Jewesses:—

		Ages.										Totals.
		9	10	11	12	13	14	15	16	17	18	18	
Natives of India, Hindus and Mohammedans,	No.	10	74	296	271	144	56	34	3	2	1	2	893
	%	1·11	8·29	33·15	30·36	16·13	6·27	3·81	·33	·22	·11	·22	
High class Indian girls,	No.			10	8	2							20
	%			50·0	40·0	10·0							
Eastern Jewesses,	No.			1	2	8	14	5	2				32
	%			3·12	6·25	25·0	43·75	15·62	6·25				

There are some very remarkable differences between this and the former table. The highest percentage occurs during the eleventh year among Indian girls, against the fourteenth year for Anglo-Indian children, and the fifteenth for the English child in its native land. The Indian girl at 12 menstruates in the same proportion as the Anglo-Indian at 14; and another prominent feature of this table is the sudden decline which takes place at and after the thirteenth year,—a decline which finds its analogue at the fifteenth for the Anglo-Indian, and not until the seventeenth year in Madden's table. The twenty unimpeachable records from among the higher class of natives tend merely to confirm the accuracy of the larger numbers. A few authentic figures regarding Jewesses were obtained; so far as they go they show that these girls follow the Anglo-Indian type. Concerning these Barnes has written: "Certain races preserve the menstrual type proper to them in the country of their origin, even when transplanted. Jewesses, whatever be their habitat, menstruate generally somewhat earlier than girls of Saxon origin"; and this would seem to be so.

The tabulated facts have been represented graphically for facility of comparison. The parallelism between Madden's line for European and Das' line for Anglo-Indian children with the one year's space uniformly maintained is very striking, and the contrast between these and the black continuous line, which represents the course followed by girls of purely Indian extraction, is indeed remarkable

(Fig. 2). This diagram also shows the ages and percentages of the thirty-two records for Jewesses. The other diagram (Fig. 3) contrasts Fayrer's and Das' observations regarding Anglo-Indians, and shows

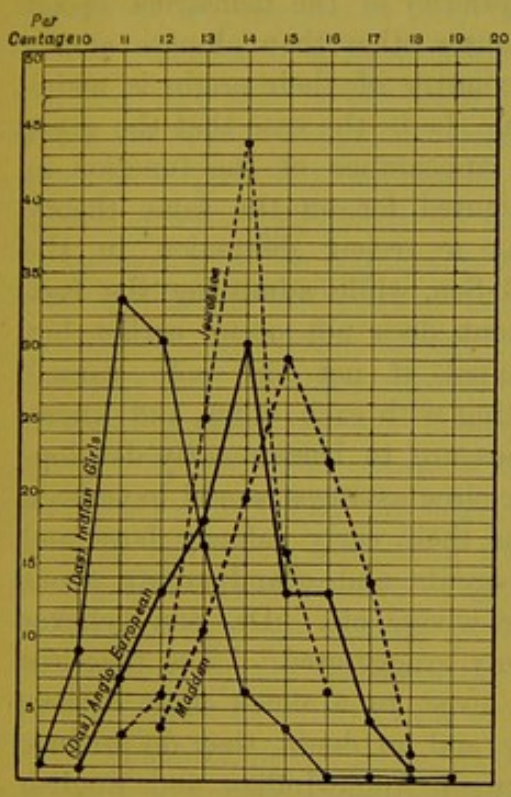


FIG. 2.

——— Europeans born in India and menstruating for the first time under its climatic influences.—K. N. DAS.[1]

- - - - - Thomas More Madden's observations in England (taken from Quain's *Dictionary of Medicine*).

..... Jewesses in India.—K. N. DAS.

——— Natives of India (Hindus and Mohammedans taken collectively).—K. N. DAS.

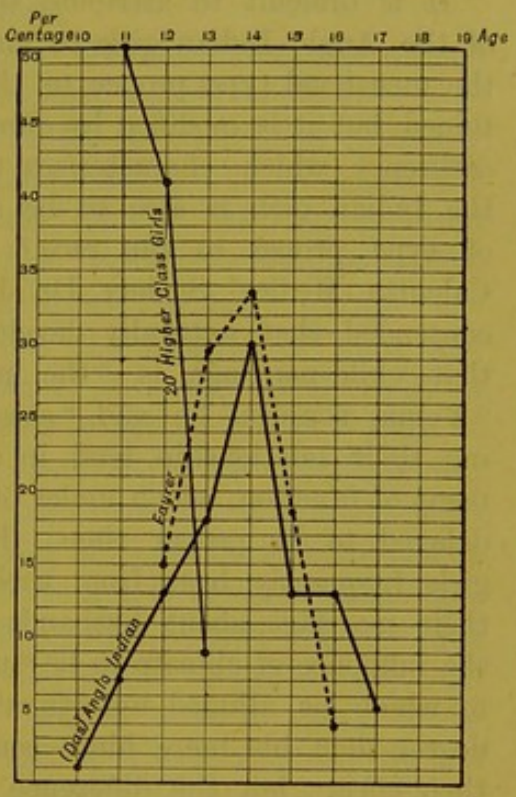


FIG. 3.

——— Europeans born in India and menstruating for the first time under its climatic influences.—DAS.

- - - - - Fayrer's observations in the European Asylum in Calcutta.

——— Respectable Bengali ladies.—K. N. DAS.

their general coincidence; and these are compared with the few records for girls of the higher class.

From these observations we may conclude that European girls born in India, or who have lived for a prolonged period under its climatic influences, menstruate about one year earlier than girls of English birth and rearing; that with this exception the physiological function pursued by either group is, on the whole, similar; that children of mixed parentage who have lived under tropical climatic influences, imitate generally the Anglo-Indian type of menstrual function, but still manifest a distinguishable priority; that children of Indian blood and rearing menstruate between two and three

[1] For *Anglo-European* in Fig. 2, read *Anglo-Indian*.

years earlier than Anglo-Indians, and between three and four years earlier than English girls in England; and that Jewesses in India follow the Anglo-Indian type.

It is difficult to attribute the priority of the menstrual epoch of the Anglo-Indian girls to any causes other than climate alone; the menstrual type proper to the country of their origin is maintained, but it is modified by climate. Is it the case that the great difference which characterises the appearance of the function in the Indian girl, is also to be accounted for on the same theory of type proper to the race? At a recent discussion in the Calcutta Medical Society, Dr. B. C. Sen, while admitting the fact, contended that naturally development does not occur earlier, but that child-marriage is "the principal, if not the sole cause." "Young couples," he said, "associating with each other, and knowing their relationship, tend in a measure to hasten early development of function, which under other circumstances would have been delayed to its proper time. From their early infancy boys and girls frequently hear from their parents and grandparents about their marriage, about husbands and wives," etc. Dr. Sen believes the influence of climate to be "unduly exaggerated." The practices to which he alluded unfortunately exist to a much more serious degree than his mere hints convey, and no doubt they do produce their effect; but the difference between Anglo-Indian and English girls is almost conclusive that climate *per se* has a marked effect. Evil customs may, and probably do, hasten the maturation of the menstrual type proper to the Indian native, but it is difficult to define the limits of their operation. That climate alone should be capable of producing the type, even in the course of generations, seems doubtful, especially in view of the remarks which have been made concerning Jewish girls of Eastern, but pure Jewish, origin.

It is pretty certain that abortions and miscarriages are more common among Europeans in India than in their native countries; and European women at the climacteric are unduly liable to hæmorrhages.

Theory of Acclimatisation.—Viewing the subject of this chapter as a whole, it may be affirmed that certain physiological changes are inevitably consequent upon transfer to a tropical climate; and the evidence is strong that under the ordinary circumstances of life, even with accompanying prudence, a certain proportion of constitutions, unfitted to encounter a warm climate, *i.e.* to accept these changes, are unable to cope with the new order of things, and so become liable to pathological changes, which gradually merge into disease. The vast majority suffer, to

some extent, by prolonged residence. But if the inevitable physiological changes be warred against by carelessness or habits inimical to the climatic demands, these pathological changes become also inevitable, and disaster is likely to ensue.

The diversion of a large proportion of the blood from the normal channels to which it has been accustomed, and in conformity with which the whole system had been educated to work, must assuredly produce a profound impression, which cannot but influence the general nutrition adversely for the time; and it has long appeared to the writer to be a question whether "acclimatisation," so-called, does not lie in the gradual adaptation of the body to the new circulatory distribution which elevates the activity of some and depresses that of other organs, combined with the acquirement of habits of general hygienic management suited to the new state of affairs. The effort of nature is to accommodate the constitution to the newly-established physiological requirements; the accomplishment of its object represents the limit of possible acclimatisation. If this be the true theory of acclimatisation, it is conceivable how it happens that the dweller in the temperate climate is better able to encounter great heat than the dweller in the tropics to bear cold. In the former case the beam of the balance is, so to speak, at the level; whereas, in the latter, at one end it is well weighted by one of the outcomes of heat (exaggerated external circulatory functions), and lightened at the other (diminished vital activities). The former is ready to adapt itself in either direction to the requirements of heat or cold, the latter is adapted markedly in the direction of heat: to encounter cold it would first have to gain the equipoise, and then, having reached the starting-point of the other, gradually regulate itself in the opposite direction, thus undergoing a double movement. Thus, too, do we see that great caution should be exercised in advising sudden changes of climate for those who are suffering from tropical congestive diseases. The teaching of experience, that such patients become worse by a sudden transfer to the cool and bracing Himalayas, yet improve immensely by the slow and graduated change to a very similar climate in Europe, is explained; and, further, it becomes apparent how greatly it may be in the power of the physician to benefit others by the like facility, judiciously exercised, of stimulating sluggishly working organs by regulating their activities in accordance with the law of their vicarious working, under the influence of climatic variations. While the organs act reciprocally for one another, there are no congestions. Even among the fairly healthy, the ascent of the hills is often attended with suppressed perspiration, a quickened pulse, polyuria,

and smart diarrhœa. A sudden increase of moisture in the atmosphere, which has the effect of lessening the moisture lost by the skin and lungs, is frequently attended by like results, the mucous membrane of the intestine acting vicariously for the skin. When organic disease is present, perfect compensation is impossible, and the patient's troubles are only aggravated by change to the hills. When the humidity is high, the temperature suddenly lowered, and the facilities for chills increased at the beginning of the rains, the sickly fail; then we get pulmonary hæmorrhages, dysenteries, and hepatitis. Those "delicate" persons who have braved the hot weather and rains often yield at the onset of the cold season in the same way.

The length of time occupied by the process of acclimatisation has not been ascertained, probably it varies individually. Bryden[1] says of the European army in India, "The invaliding of the first four years of residence represents the loss sustained while the European constitution is being subjected to that process of adaptation to the climate of India which we assume to occur in the case of the newly arrived. That men who have broken down during the process have furnished three-fifths of all the invaliding of 1871, is a fact which cannot be too seriously contemplated, whether it be regarded from a political or sanitary point of view."

Influence of Warm Climates on Pathological Processes.—If the vasomotor system participate in the general nervous depression, and undergo relaxation throughout, the internal organs will be preternaturally ready to receive too large a supply of blood (congestion) when the surface of the body is subjected to chill. We have seen that in the tropics the power of generating animal heat is diminished, because so much is not required, and because the system has become asthenic. It follows that the tropical resident is unable to resist an amount of cold which would be inadequate to hurt more robust constitutions in temperate climates; and when we further recollect the frequent and immense diurnal fluctuations, combined with high humidity, common in the tropics, it is evident that the body must be exceedingly liable to chills of which the factors are present, and to the ill-effects arising therefrom. Probably many of the ephemeral fevers of India are to be accounted for solely through the agency of the nervous system. On the one hand, the central nervous tension may be so deranged or modified by heat as to cause it to lose its restraining influence over the oxidation of the tissues; or, on the other, the new

[1] Report on the influence of age and length of service in India, as illustrated in the European army during 1871.

distribution of the blood, under the influence of chill, may so affect the chemical changes (nutrition and destruction) in the tissues, as to influence the nerve terminations, and thus again, from this opposite direction, produce the same effect of overpowering the inhibitory nerve tension. If this view be correct, the distinction between sun-fever—fevers due to chills—and true malarial fevers becomes pathological, practical, and rational. The first, due to primary nervous shock, might be designated "ardent" or "nervous" fever; the second would be correctly defined as "congestive" fever, the nervous participation being secondary; and the third as specific malarial fever.

A tropical climate suits some enfeebled people admirably—their appetites improve, and they become capable of greater muscular exertion. The external warmth seems to be necessary to stimulate their functional energy. Such persons seem not to be capable of manufacturing sufficiently bodily heat in a temperate climate; but the warmth removes the great tax which was formerly placed upon their organism in the endeavour to do so, while the skin, which was before inactive, perspires freely, relieving the digestive and other organs from their condition of apathy. To old tropical residents, to whom anæmia or general cachexia has become a chronic state, removal to the hills is hateful; while they remain on the plains they are perfectly happy, and consider themselves to be in good health. The weak-chested and scrofulous, in Martin's opinion, benefit by residence in the tropics; and the experience of others confirms this statement. On the other hand, it should be stated that cases of developed pulmonary phthisis succumb rapidly.

It may be accepted almost as a law, that once the constitution has received a severe shock in a tropical climate, a prolonged period is required for recovery; a change to a temperate region is usually essential to recovery.

A class of persons who must be regarded as bad lives in India, are those who are overburdened with fat. They suffer much from heat, but their risk lies in the consequences of the severe climatic fevers, and early heart failure is then the immediate danger to be apprehended.

Bryden points out, in his admirable *Army Statistics*, that newly-arrived young soldiers die in "a strikingly larger proportion" than men of older residence—48 per 1000 was the death-rate of 1864–69, and this had been reduced to half, or 24 per 1000, as the ratio of 1871–75; while the rate for the army of India, excluding cholera and violent deaths, for the latter period, fluctuated between 11 and 15 per 1000; and 48 per cent. were under 5 years' service. "We may

expect to find in the statistics of the same body of men," wrote Bryden, "after twelve months' residence in India, a considerable decrease in the diseases which show themselves in an exaggerated form in the first hot season. These are the severer class of fevers, heat apoplexy, and bowel complaints; and the death-rate from these diseases may be expected to show a corresponding diminution. This expectation is borne out by the figures which follow. Among the admission-rates, enteric fever goes down from 13 in the first year to 5 in the second; remittent and continued fever, from 315 to 150; heat apoplexy, from 5 to 1·7; and bowel complaints, from 156 to 112." The death-rate diminishes from 19·3 to 11·5; the fever ratio of 7·96 becomes 2·8; heat apoplexy is diminished by one-half, and the dysentery mortality is less.

The following extract illustrates the different modes in which climate affects different nationalities: "The death-rate of the native army is chiefly influenced by cold, and by the prevalence of diseases of the anæmic type. This is followed by diarrhœa, spleen enlargement, dropsy, scurvy, and atrophy; and when the system is thus debilitated, exposure to cold readily determines a fatal pneumonia. It is remarkable to note how, as we proceed northwards, the relative components of the death-rate alter their positions, bowel complaints diminishing, and respiratory affections gradually predominating in the ratio." The native succumbs to fever during the colder months of November, December, January, and February (60 per cent.); the European during the hot months, from May to October (70 per cent. of total fever deaths); and these periods may be taken to represent the dangerous seasons to either class, —63 per cent. of the native mortality falls in the cold months, and 37 in the hot season; while in the European army 62 per cent. falls in the hot months, and 38 in the cold. Dysentery, fever, and pneumonia make up three-fourths of the mortality of the native army. Sixty per cent. of all deaths from fever and dysentery occur between November and April, and 80 per cent. of all deaths from respiratory diseases are shown in the same months. Respiratory diseases represent 20 per cent. of the mortality of the native, and 5 per cent. of that of the European; but hepatitis, which causes 1·59 per cent. of native mortality, brings about 17 out of every 100 deaths of Europeans; and heat apoplexy is shown with a ratio of 11·51, as compared with 1·88 in the case of the natives. These facts regarding different classes, of different nationality, serving in a climate for the most part foreign to both, are very significant of the effects of a hot climate upon varying types of constitution, and the same features are exhibited when we

examine the statistics of other large bodies of persons of whom accurate records are made, such as jail populations.

When we consider the physiological peculiarities which must be inherent in either race with regard to blood distribution, the consequences thereby brought about by climatic changes and other facts concerning the excretions and effects upon the nervous system, it does not become a difficult task to surmise the directions which disease will naturally tend to assume under the special conditions of a tropical climate. Certain organs of the imported European are manifestly called upon to exert themselves less than formerly, while others are worked beyond their wont. The thoracic viscera are spared, but the liver, spleen, intestines, and skin, which have functions to perform in excess of those thrown upon them in a temperate climate, are particularly liable to undergo pathological changes, and to suffer from actual disease upon the occurrence of any exciting cause. This remark applies especially to the European; with the native, who is simply exposed to new, but still tropical climate influences, the order is somewhat reversed, in the directions which have been already indicated. His constitution is set for warmth, and a change to the north of his own vast country affects him in a reversed order; or, when he migrates to the east, he is subjected to somewhat the same effects in consequence of the excessive humidity of the climate, and the consequent chills and congestions. The nervous system may be included among the tissues, which are unduly ready to yield, but apparently for an opposite reason; here we have exhaustion caused by continuous heat, impoverishment of the blood, and possibly also altered thermotaxis due to diminished supply of blood,—causes all of which tend to increase nervous irritability, and lessen its controlling influence over nutrition and the mechanism of the vital functions. The type of nervous diseases met with in India among its own population presents some remarkable peculiarities; locomotor ataxy, for instance, is never, or almost never, met with; and tetanus is very prevalent, both among infants and adults. Of the latter disease, if we are to admit the theory of its telluric origin, which has received such strong support, it would seem that the soil of the country and the climate must be well suited to the vitality of its bacillus. Most of the cases seem to be idiopathic, though the tide of opinion goes strongly with Verneuil, that idiopathic tetanus is non-existent. Persons with any hereditary taint of insanity should never migrate to the tropics, and those European residents who develop insanity seldom recover till removed to Europe. The same applies to epilepsy. The hope of recovery by change to a temperate climate is great; it is small otherwise.

During the hot tropical season, delirium tremens is very fatal; but these are subjects which, together with other peculiarities of diseases in tropical climates, such as the immunity from scarlet fever, the extreme rarity of enteric fever among native races, and its great frequency among newly-arrived European youths, the singular difference in the type of diabetes met with in England and India, and so forth, do not come within the scope of this article, and some of these will be treated fully elsewhere.

Certain indirect effects of heat may produce disease. Children, for instance, often suffer severely and even fatally from bowel complaints produced by fermentative changes set up in milk; and, similarly, adults may be affected by ptomaine poisoning, the result of early and undetected putrefactive changes in animal food.

CHAPTER II.

THE HYGIENE OF THE TROPICS.

BY J. LANE NOTTER, M.A., M.D.

THE climate of a country has a most important influence on the health and character of its inhabitants. It therefore requires careful study by those who, having previously lived in temperate zones, are suddenly transferred to a tropical country: and whose very change of environment necessitates a perfect knowledge of many rules and precautions, if they desire to maintain a standard of health at all commensurate with what they naturally expect to enjoy at home: as well as to ward off those diseases, the result of deteriorations of the functions of the body, which are the effect produced by long residence in tropical countries.

It is proposed in the following pages to consider the clothing, food, exercise, and, briefly, the habitations which experience has shown to be best suited for the maintenance of health in the tropics; to give an outline of the sanitary arrangements best adapted to preserve the health of an army in peace and war; the effects of change of climate on Europeans generally, and the results of residence in tropical climates usually frequented as health resorts.

Clothing.—The object of clothing is to protect the body against cold and heat, wind and rain; and it is the power man possesses of modifying the character of his clothing, and adapting it to the climate in which he is placed, that enables him to accommodate himself to dwell in any part of the world. The same holds true, as regards season, even within the temperate and subtropical zones, and therefore the selection of clothing is of the first importance.

In the tropics, where the external temperature generally exceeds that of the body, there is increased action of the cutaneous circulation, and by rapid evaporation and radiation the normal temperature of the body is maintained. The clothing in this case serves to

protect the body from the effects of external heat and the direct rays of a tropical sun.

The subject of clothing naturally divides itself into two parts, viz. (1) the materials, and (2) the make of the garments worn.

The following are the materials of clothing usually adopted:—

Cotton. This is the fine downy hairs surrounding the fruit of a shrubby plant of the species *Gossypium*, cultivated in the tropics and Southern States of America for textile manufacture.

The quality is judged by its smoothness and by the length of its fibres, which vary from half an inch to one inch in length. When examined under the microscope, cotton fibres exhibit a diaphanous membrane with an internal tube closed at both ends. When the material is old it is said to be flatter, more twisted, and riband-like. When cotton and linen fibres are closely interwoven, there is great difficulty in detecting this by the microscope, and the following chemical test may then be used. The linen containing cotton fibre is placed in a bath of sulphuric acid for one to one and a half minutes, the cotton fibre is immediately affected, the sulphuric acid acting on it more rapidly than upon the linen. The fabric upon being dried has a shrivelled appearance.

Linen is from the fibre of the flax plant *Linum usitatissimum*. The flax is treated by chemical and mechanical means to fit the fibre for textile purposes. When examined under the microscope the fibres are seen to be finer than those of cotton, diaphanous, cylindrical, and presenting transverse striæ at regular intervals.

As an article of clothing, cotton, not being so good a conductor of heat, is warmer than linen; it also absorbs moisture better, not becoming wet so soon. Both are conductors of heat, especially linen; but they are very non-absorbent of moisture, whether into the substance of the fibres or into their interstices. Linen is much smoother and more lustrous than cotton cloth, and presents a less "woolly" surface. It does not soil so readily, nor absorb or retain moisture so freely as the more spongy cotton. Cotton clothing is, therefore, well adapted to warm climates, except when the body is perspiring, in which case a dry woollen inner garment is preferable. It is also a cool and healthful material for bed-sheeting.

Cellular cloth, a material recently brought into use for under-clothing, is made principally from cotton. The fibres are so woven as to have large interspaces in the fabric, and the air contained therein renders it an excellent non-conductor of heat. Its warmth depends on its porous character.

Jute, a fibre obtained from the *Corchorus capsularis*, is mainly cultivated in Bengal. It is used in India to a limited extent for

the manufacture of native clothing, and is said to be used as an adulteration in some fabrics in this country. The fibre is very brittle and hygroscopic.

Silk is a fibre produced by the silk-worm (*Bombyx mori*). The silk fibre is smooth, cylindrical, devoid of structure, not hollow, and equally broad. The surface is glossy, and irregularities are seldom seen on it. Silk is very hygroscopic, taking up as much as 30 per cent. of water and not feeling damp. It is also a non-conductor of electricity.

Wool is a modification of hair, being distinguished from the latter by its fineness, its being curled, not straight, and generally of a white colour, as it contains less pigment. The epithelium of wool consists of thin plates which overlap each other; the fibres are round, transparent, and colourless, unless dyed. They have on their surface imbricated scales, all of which run in one direction. Wool is obtained from the sheep, the Cashmere goat, and the llama. Alpaca and mohair, or so-called camel's wool, from the Angora goat.

Shoddy is made by tearing up woollen rags, and is used in very considerable quantities both along with and instead of fresh wool.

As articles of clothing, silk and wool are bad conductors of heat, silk being more so than wool. Both silk and wool are very hygroscopic, absorbing moisture so completely that they feel dry when cotton and linen would be damp and cold. A fabric made from either of these will thus absorb a large quantity of moisture, as well as hold water in its interstices. Owing, therefore, to their large hygroscopic power, evaporation is exceedingly slow from the surface of the skin. When perspiration is excessive, the silken or woollen garment does not get thoroughly wet, and evaporation being gradual, the body gets less rapidly chilled. On the other hand, when cotton and linen garments are worn next the skin, these speedily get damp, especially when exercise is taken in hot climates, and, adhering to the surface of the body, induce rapid evaporation, which lowers the temperature and produces chill when exercise ceases.

Much of the good effects of clothing depend on the porosity of the material. Air is one of the worst conductors of heat, and the warmth of a garment is to a large extent due to the amount of air entangled in its interspaces. Woollen materials being, as a rule, loosely woven, are more porous than linen and cotton, and are on this account warmer. It is for this reason that cellular clothing has been introduced and recommended. Wool is less easily penetrated by cold winds, and the same layer of warm air remains in contact with the body. Wind tends to reduce the temperature and

produce chill by increasing evaporation from the surface, as well as replacing the warm air in the interspaces; therefore, when the air is cold and there is much wind, furs are worn to keep off the wind. India-rubber garments produce the same effect; but they not only prevent the entrance of wind, but also, by checking evaporation, allow moisture to collect, which by its power of conducting heat reduces temperature and causes chill. This material, being itself non-porous, favours the escape of heat from the body.

The clothing introduced by Dr. Jaeger is one of the best materials for warm climates, excepting the hot season in the tropics—it is too thick and warm for wear at that period. The fabric is so made that it does not irritate the skin. This is accomplished by a careful selection of the wool, which is of the best quality procurable. The shape and arrangement of the constituent hairs of the material also provide for the escape of moisture, while air permeates freely through the texture. It is one of the best materials for underclothing.

In the hot season in the tropics flannel is frequently found to be too heavy a material for comfort, and cotton and linen underclothing are used; these are most uncomfortable, and ill adapted for the purpose. It is said that the Chinese adopt a plan of wearing a fine net next the skin, and a thin silken garment over the net. The net, without increasing the heat, prevents the perspiration soaking into the upper garment, and the latter from adhering to the skin. Cellular underclothing is well adapted for this purpose.

In the choice of woollen underclothing we have largely to depend on the touch as being the best guide. Smoothness and softness of texture are required; the hairs on the surface should be even, not straggling, and the material should possess weight. When viewed by transmitted light, the cloth should be of uniform structure; and it should not tear easily, but have a certain resisting power. Microscopic examination will at once detect any admixture of cotton, linen, or jute fibre.

The disadvantages of woollen clothing are that the fabrics deteriorate by washing,—shrinking and becoming harder. With many persons in the tropics they cause irritation of the skin, so that these garments cannot be tolerated. To avoid shrinking, the material should be well soaped on both sides and rinsed in cold water, and this afterwards allowed to drain from them.

In India, woollen garments are frequently hung up, exposed to the sun, and well beaten.

Colour influences the absorption of heat, therefore the colour of the material worn is an important factor in warding off the effects of solar heat. White garments absorb the least heat, and are

best for the tropics during the hot season. White cotton being taken as 100, pale straw will be 102; dark yellow, 140; light green, 155; dark green, 168; turkey red, 165; light blue, 198; black, 208. Experiments made at Aldershot some years ago showed that the absorbing power of grey was next to white and less than yellow. White and grey are the best reflectors of heat, and therefore the coolest; while blue and black are the worst colours, as absorbing the most heat.

From this somewhat brief and general description it will be comparatively easy to determine the articles of clothing best suited for the different parts of the body.

A proper head-dress is absolutely necessary; this should be light, have a free circulation of air around the head, and afford protection to the eyes, temples, and nape of the neck. In India a good thick solar "topé" is perhaps the best, and when well fitting and ventilated is an effective guard against the sun's rays. The "Tuson" helmet is highly spoken of in India. It consists of two bodies, one within the other, forming a complete air chamber, not only in the crown but in the brim, thereby completely protecting the temples and the nape of the neck. The weight is about 10½ oz.

If much exposed to the sun, it is advisable to wear a light wadded curtain of white drill, which will afford further protection, if attached to the brim of the helmet; this will also shield the side of the head and back of the neck from the horizontal rays of the sun. The temples are the parts requiring attention when the sun is low and not directly over the head.

Straw hats are unsuited for hot climates; they are made too low in the crown, which is generally in contact with the top of the head, and thus offer little protection.

It is a question well worthy of consideration whether it would not be possible to adopt the universal custom of Asiatics in wearing so-called turbans or thick folds of linen round the head. It is possible, if racial prejudices could be overcome, the adoption of this head-gear by Europeans, and assimilation to native customs in this respect, would materially conduce to the prevention of heat-stroke.

During the cold season in the tropics, a grey felt "wide-awake" which has ventilation round the rim is perhaps the most comfortable head-dress; but the rim must be broad, so as to give protection from the sun. "Puggarees" are frequently worn with such hats. In the hot weather a light, white-covered umbrella will be found useful by the protection it affords.

It is desirable to protect the eyes from the direct rays of the sun in the tropics. The eyes really offer very little resistance to the

passage of the sun's rays, as the optic nerve is fully exposed to light rays. Neutral tinted glasses give an enormous amount of relief to the eyes, as well as afford a sense of coolness. They also confer mechanical protection to these delicate organs from dust and flies.

With reference to the clothing of the body, the indications to be carried out as regards dress in hot climates are the following:—

1. To maintain an equable temperature of the body by reducing the absorption and conduction of heat.

2. To allow muscular action the greatest freedom, and so to limit the fatigue due to mechanical work.

3. To protect from wind, cold, and damp, and thus to avoid those diseases induced by chill.

4. To allow evaporation from the skin to proceed with as little hindrance as possible.

Such being the objects to be fulfilled, the underclothing, for reasons already stated, should be of wool, its thickness depending on the season of the year,—from stout flannel in the cold weather, to the thinnest gauze in the hot. If cotton underclothing is used, that in the form of cellular clothing is the best, as it entangles a maximum quantity of air in its meshes. Underclothing should be made to follow the outlines of the body, and sufficiently loose so that it will not compress the thorax, abdomen, or limbs. The band of the drawers should always be made of flannel, as the perspiration is greatest at this part when exercise is taken. On this account, and to avoid the risk of chill from evaporation, a "Kummerbund" or binder is worn over the parts to afford additional protection to the abdominal organs. The most rational shape of the dress in warm climates would seem to be that known as "combinations," which may fall loosely over the abdomen; and by not compressing it in any way, excessive perspiration over that part is avoided.

In the hot weather, the coat is generally made of cotton or some washing material. It is advisable that it should not be too highly starched, as this hinders evaporation. But thin flannel and serge are also used; both are excellent materials for the purpose. The clothing for the legs should be trousers; they are cooler than knee-breeches or knickerbockers, and have the advantage of allowing the greatest movement of the leg. Braces are better than a belt, inasmuch as they do not compress the abdomen, and cannot be said to interfere with respiration.

As regards the foot-gear, during the hot season shoes are preferable to boots; the leather should be soft and pliable; tanned leather

shoes are most comfortable when properly made. Great care should be taken in getting the correct measurement for boots and shoes. As a rule, the foot is measured when raised from the ground, no allowance being made for the natural expansion of the foot in walking. Every time one foot is raised, the other expands one-twelfth of its length and one-eighth of its breadth at the base of the toes. In taking the measurements, therefore, the foot should be firmly planted on a sheet of paper, and the sole described with a pencil. The absurdity of pointed toes need only be referred to here.

Boots may be made impermeable to wet, as follows:—

Take half a pound of shoemaker's dubbing, half a pint of linseed oil, half a pint of solution of india-rubber. Dissolve with gentle heat and rub on the boots. It is well to renew the application of this to the boots every three months.

Cloth may be made waterproof by the following simple method. Dip the material alternately in a solution of sulphate or acetate of alumina and of soap.

Clothing may be rendered incombustible by being steeped in a solution of tungstate of soda—one pound to two gallons of water, or, if starched, by adding one part to every three of starch.

Food.—As usually understood by the word, food includes everything taken into the body which goes to the growth or repair of its structure, or which can be made use of to promote the functional activity of the system.

As this varies considerably with the climate, the nature of the food will also depend on the requirements of the body under such altered conditions as are to be found in tropical climates.

The classification of foods necessary to support life in its highest state of vigour is based on the fact that milk, which is the staple food of the infant, contains all the proper aliment necessary to the support of life, namely, nitrogenous or proteid substances, fats or hydrocarbons, carbohydrates and salts: and experience has shown that these classes serve different purposes in nutrition, and all are required to maintain health, and must be given in due proportion.

The purposes served by food are—

1. The maintenance and repair of the body.

2. The production of energy as manifested by (*a*) exercise; (*b*) animal heat.

A large part of the solid constituents of the body consists of proteid substances; these are constantly undergoing oxidation, and the oxidised products are daily excreted in the urine in the form of urea and uric acid, so that the body is constantly using up

nitrogenous matter. To supply this waste, nitrogenous food is taken somewhat slightly in excess of the amount required, and in this way an equilibrium is established. Voit's experiments show that proteids taken in the food accumulate in the general fluids of the body, which draws on them for the amount required for growth and repair; and it is probably to this excess of nitrogenous food that functional activity, and possibly also the power of resistance to fatigue or exposure to disease, is due. But if the ingestion of this class of food-substances is carried to excess, an excess is excreted, the amount of urea passed in the urine being dependent on the quantity of proteid ingested, and not on muscular exertion.

It will be seen that the quantity of nitrogenous substances is almost a fixed one; for the repair and waste of the tissues, under ordinary circumstances, is almost a constant quantity, and far less than was formerly laid down. Only when a great increase in muscular work takes place, or in the repair of waste after a severe illness, is there a need for an increase in nitrogenous food.

Excess of proteids cause more rapid metabolism (tissue change), and if not counteracted by active exercise, the result is that in the fluids of the body there will be formed an excess of proteid material; the blood becomes loaded with unoxidised products, an excess of urea and uric acid appear in the urine, and possibly albumen.

When additional work has to be performed, it is the fats and carbohydrates, which are the real sources of energy as well as of heat, that have to be increased.

In hard work as well as in extreme cold, fat is largely made use of on account of its ready oxidation. Carbohydrates differ from fat, in that the amount ingested is in proportion to the carbon dioxide excreted. In oxidation carbohydrates require only sufficient oxygen to unite with the carbon, since the hydrogen and oxygen are in proportion to form water; but fats require oxygen to oxidise their hydrogen as well as their carbon. Whether fats and carbohydrates can replace each other is a moot point; but the weight of evidence is decidedly against their being able to do so. An excess of fats and carbohydrates lessens metamorphosis of tissue, and thereby fat is stored up in the body; if not digested, symptoms known under the names of biliousness, acidity, dyspepsia, etc., follow.

Salts are as essential as the proteid substances. Sodium chloride is easily decomposed in the stomach by the action of the gastric juice, and hydrochloric acid is formed; this keeps in solution the globulin of the blood. A deficiency of this salt leads to dyspepsia and gastric derangement. Its use appears to be more necessary

with vegetable than with animal food, as it is present in considerable amount in the latter, but only exists in very small quantities in the former. In India the Hindu recognises this, and consumes large quantities of salt with his food. Phosphates have the property of readily combining with the alkalies, sodium, calcium, etc., and are essential to life, particularly in the young, since they constitute the solid parts of bone. Salts which form carbonates in the system, such as lactates, tartrates, citrates, etc., maintain the alkalinity of the blood and other fluids of the body. If they are absent from diets, the condition known as scurvy follows. They are quite as essential to health as any of the other classes. These salts are supplied in fruit and fresh vegetables, and, although their nutritive power is small, they are never absent from a well-arranged dietary.

The above brief description of the general principles on which a dietary is constructed, will indicate the chief points to be attended to in the tropics.

To maintain health, a diet containing proteids, fats, carbohydrates, and salts, with a certain quantity of water, sufficient for the solution of the juices and for other work, is necessary. It is evident that no two persons will require or consume exactly the same diet; but from a number of careful observations made on healthy individuals, a *standard* diet has been fixed, which for all practical purposes may be accepted.

The quantity of food ingested will be influenced by the age, sex, and work of the individual. Aged persons living in warm houses require a very small amount of food compared with children, when the period of growth and vital energy is at its highest. Of the standard diets that have been proposed, that given by Moleschott may be accepted as being approximately true; it is—

Proteids,	4·59 oz.
Fat,	2·96 „
Carbohydrates,	14·26 „
Salts,	1·06 „
Total water-free food,	22·87 „

This amount of food is sufficient for a man in ordinary work (300 foot-tons) weighing 150 lbs.

This diet in terms of carbon and nitrogen is nearly as follows:—

Nitrogen,	321 grains.
Carbon,	4737 „
Hydrogen,	179 „
Sulphur,	28 „
Salts,	464 „

This is water-free food or dry food; but in ordinary food there is from 50 to 60 per cent. of water, so that this diet is equal to about 50 oz. of the so-called dry food.

In addition, from 60 to 80 oz. of water are necessary, and are taken in some liquid form, as tea, coffee, beer, etc. Climate and temperature seem to exercise little influence on the quantity of food required. In cold countries generally more exercise is taken, and perhaps more food is required. Certainly exercise and an open-air life is necessary to digest and assimilate a large nitrogenous diet.

Fats are more digestible in a cold climate than carbohydrates, which are more easily assimilated in hot climates.

In India, although carbohydrates form the chief food of the native population, yet, wherever and whenever it can be done, this is supplemented by the use of fats in the form of ghee, which is largely consumed in that country. Animal food possesses the following properties. When well cooked it is easy of digestion; the proteids average in beef about 20 per cent., the fats about 5 per cent., and 1 per cent. of salts. These are more easily assimilated when supplied from animal than from vegetable sources; it is necessary that the cooking should be well done, not only to render food as digestible as possible, but also to ensure the total destruction of parasites. Beef is more digestible than mutton, and this again more so than fish and poultry.

Milk, which is an animal food, is generally taken in this country from the cow, but in India milk from the buffalo and goat is also used; the specific gravity of these three kinds of milk is about the same, generally 1032. Buffalo milk contains much more total solids than cows', 18·6 per cent. against 12·6; it is also richer in fat, 7·5 per cent. against 3·7, while the proteids are double. It is not, therefore, as digestible as cows' milk, and is not as pleasant to take. Goats' milk is in common use in India, especially among the natives. It contains usually 4·2 per cent. of fat, 4 per cent. of milk sugar, and 3 per cent. of casein. As a rule, it is richer in cream, but poorer in proteid matter than the milk of the cow. It is somewhat objectionable for general use, as goats are foul feeders, and in India are usually allowed to roam anywhere in search of food.

In India and in the tropics generally, cows are often fed on the litter from stables, and the places in which the animals are kept are filthy; their limited supply of food leads them to feed on any sort of garbage. It is wise, therefore, to use no milk which has not been previously boiled; if it is possible to do so, the feeding of the animals and the milking should be under supervision, but the use of

raw milk should not under any circumstances be permitted. Milk which has been boiled should be so guarded from contamination by keeping it in a perfectly clean and well-ventilated place, that there can be no danger of its becoming infected by dust or flies. In parts of India where cholera is endemic, special attention should be paid to this point, as it has been shown that the danger is greater when boiled milk is used: as boiling delays the acid fermentation of the milk, which of itself helps to inhibit the growth of the cholera organism. Milk that has been boiled should be kept in a perfectly clean place, and guarded against access to dust or contamination by foul air.

The flesh of the ox, sheep, calf, and pig is that usually eaten by man.

Bone forms about 20 per cent. of the meat as sold in this country. In India the proportion is somewhat higher. In that country a full grown gram-fed sheep seldom weighs over 35 lbs., and yields about 18 lbs. of available food; an ordinary country sheep will not weigh more than 25 lbs. The amount of fat depends on the feeding of the animal. In the tropics sheep, as a rule, are badly nourished, and consequently their yield of fat is extremely small. All meat, especially in India and other tropical countries, requires to be well cooked. Oxen, particularly in the Punjab, are very commonly infested with the cysticercus of beef, which gives rise to *tænia mediocanellata* in man. Sheep are liable to be infested with *tænia tenella*.

To destroy these parasites the meat should be thoroughly cooked; underdone it is not safe.

The flesh of the pig is comparatively little used in India, notwithstanding that it is the staple food of the working classes at home. The pig is liable to be affected with the cysticercus of the *tænia solium*, constituting the affection known as "measles," and this "measly" pork is the chief source of a tape-worm—the *tænia solium* in man. The pig, further, is peculiarly liable to be affected with another parasite, the *trichina spiralis*, causing the disease known as trichinosis. Human beings who consume trichinous pork readily contract the disease, which usually runs a chronic course, but in some 25 per cent. of cases ends fatally.

The system of conservancy, or rather the want of any system in Eastern countries, renders the flesh of animals liable to be infested with many parasites; the difficulty of obtaining proper food for animals, combined with the carelessness and indifference of the natives, adds to the danger of infection, and renders it all the more necessary to exercise strict supervision, not only in the cooking of meat, but in the selection of supplies tendered by native butchers.

The natural tendency of the native cooks to overdo all meat in the process of cooking, is no doubt partly the reason so few Europeans suffer from parasitic diseases in that country. Raw vegetables, such as salads, etc., should be well washed before use. During the hot weather any vegetables, except potatoes, are difficult to obtain in India. With the present excellent supply of preserved vegetables (best when preserved in bottles), this difficulty has been overcome. Their place in a dietary has hitherto been taken by fresh fruits, etc.

Cooking vessels should be inspected regularly. In India these are made of copper, and are usually tinned inside. It is important that this should be well done, as native cooks are in the habit of allowing food to remain many hours in such vessels, and the copper is liable to be acted upon; pure tin should be used, and not an amalgam of lead and tin. The tinning material is commonly known in Indian bazaars as "Kalai," and some supervision is needed to see that it consists almost entirely of pure tin and not an amalgam containing an excess of lead.

The water supply in India is taken generally from canals or shallow wells; both sources are liable to pollution. In some few of the larger cantonments a regular water supply is laid on, the source generally being from a mountain stream; but this is the exception, and wells furnish the chief source from which water is taken.

Canal water must be regarded with suspicion; the water is usually sluggish, and fouled by the natives. It should never be used if another source of supply is available.

Wells usually furnish a good water if the surroundings of the well are clean and all dangers from surface pollution guarded against. The practice natives have of washing, not only their clothes, but their persons in the immediate vicinity of wells should never be permitted. In India, water derived from wells in cantonments is generally of good quality, but considering the danger to which it is exposed by surface pollution, as well as from the method adopted to bring it to the surface (draw-wells), it is advisable not to use such water for drinking purposes without previously boiling it. The experience of the writer of this article is against the use of any form of domestic filter hitherto adopted. Under the conditions of life in that country it is not safe to rely on any filter; but for the purpose of aerating the water, after boiling, it may be passed through sand in a "chatti" or earthenware vessel of native make, care being taken to select clean sand and to protect the water and filter from dust. These chatties or earthenware bowls need to be regularly inspected, and periodically destroyed and replaced by new ones.

Animal charcoal, so largely adopted for domestic filters in this country, should be abandoned; while, too, the routine use of filters generally in tropical countries needs to be judiciously watched and discouraged, as experience goes to show that their general use gives rise to a sense of security which is not warranted by the only too common conditions of the filters employed in most European establishments. Filters, unless clean and constantly kept clean, in place of purifying the water passed through them, merely render the last condition of the water worse than the first.

As regards the use of alcohol in the tropics, the question is a debated one, and is unlikely to be ever more definitely settled. In the extremes of heat and cold men have maintained perfect health, and are certainly better without it, if provided otherwise with proper food. Its universal use during long ages seems to indicate some necessity for its continuance. In the tropics the stronger forms of alcohol are injurious unless freely diluted with water. The physiological effects of alcohol on the system are now fairly well established. It certainly does not raise the temperature of the body, and whether it lowers it is still a disputed point; it dilates the cutaneous vessels, and thus more blood is brought to the surface of the body, so that it lessens body heat by radiation; it causes increased action of the heart, followed by a period of depression, and in small quantities acts on the nervous system by increasing the quantity of blood sent to the brain, and with it mental activity. If, however, large quantities are ingested, the nerve centres become paralysed and depression follows. In moderate doses it induces sleep, and possibly to those accustomed to its use assists digestion, though probably it may retard it also. It must be clearly understood that alcohol is not necessary for man in any climate, and should only be taken when the day's work is finished. In the tropics, spirits should be avoided while the sun is above the horizon; but small quantities of alcohol taken with food in the evening seem to check waste, and to remove a sense of fatigue incidental to life in hot climates, where the appetite is never very vigorous, even with those in health, and where all Europeans suffer more or less from anæmia. It also aids digestion and produces sleep; but those who can do without it are strongly recommended to abstain from it altogether. There is a universal consensus of opinion that alcohol taken in excess is most injurious. To its indiscriminate use are due those diseases of deterioration, such as heat-stroke, hepatitis, heart diseases, and dysentery, so frequently seen in old soldiers, not to mention delirium tremens, which is an acute poisoning induced by it. There is also less resisting power to all diseases, especially

those of an epidemic form; this is shown by the fact that cholera more frequently attacks those accustomed to take spirits in excess, and these seldom recover. The habitual use of alcohol between meals is the very worst form of its abuse, although this may not have any intoxicating effect. After prolonged fatigue its value is unquestionable, provided rest can be enjoyed afterwards.

The quantity a man may take with comparative safety is important. Dr. Parkes, by experiment, showed that a man who was accustomed to the moderate use of spirits, could not take more than $1\frac{1}{2}$ to 2 oz. of absolute alcohol daily; if this quantity was exceeded, alcohol appeared in the urine. Ordinary whisky contains about 50 per cent. of absolute alcohol, so that about 3 oz. may be considered the maximum quantity which a man in good health should take in twenty-four hours. Such amount should never be taken at one time, and should always be largely diluted with water.

The use of beer as a beverage is of very doubtful advantage in hot climates. It has of late years been introduced into India. In order to prevent the beer turning sour, the alcohol is in excess of that generally found in home-brewed ales, and reaches as high as 5 and 6 per cent. vol. in vol. One pint (20 oz.) would therefore contain 1 oz. of absolute alcohol. Its action on the system is certainly depressing; this is due to the lupulin it takes up from the hops. It undoubtedly checks metabolism by lessening oxidation in the body, and thus causes a deposition of fat.

Light wines, freely diluted with water, are perhaps the best when some form of alcoholic drink is required. The vegetable acids in light wines prevent scurvy; they aid in digestion, and being acid they are to a certain slight extent a prophylactic against cholera.

Tea, coffee, and cocoa are excellent drinks in hot climates. All act on the nervous system and are distinct restoratives. Both tea and coffee are said to have a protective influence against malaria. When tea is drunk in considerable quantities, it should be infused for only two or three minutes and then poured off the leaves. Cold tea is an excellent drink during the day and evening; if made strong it retards digestion.

Coffee and cocoa are the best drinks to take in the early morning; they are more satisfactory than tea, and hunger does not supervene so rapidly. Of these two cocoa is preferable. In the tropics the use of lime juice, when fruit and vegetables cannot be obtained, is necessary. Scorbutic dysentery following on an ill-arranged dietary is not an uncommon occurrence.

If fresh vegetables cannot be had, preserved ones (not compressed) may take their place. Compressed vegetables are of very little antiscorbutic value.

Such are the chief points which should guide the European as regards his dietary on taking up his residence in a tropical country; his chief rule should be "moderation in all things," and he should endeavour to proportion the food to the work which the body is called on to perform. Even without excessive exercise there is considerable waste of tissue, and therefore a certain amount of proteid food in the form of meat is necessary; but there should be no excess. On the other hand, the inability during the hot weather to take much exercise, and the enforced confinement in hot rooms, render the quantity of fats and carbohydrates needed less, neither an excess of bodily heat nor energy being required. Condiments and highly spiced dishes are for the same reasons to be avoided.

Alcohol should be used with the greatest moderation, and taken only with food, its use being restricted to the evening.

It should also be remembered that thirst and the habit of relieving it by fluids can be educated, and it is not wise to give way to this in the early period of residence in the tropics. Fluid drinks in excess tend to weaken the digestive processes, and, later on, spirits and condiments are required to stimulate the stomach, rendered weak by an over-indulgence in constant drinking indulged in to assuage thirst on first arrival. When at rest, occasional large drinks of cold, weak tea are very beneficial, as they reduce the high specific gravity of the urine resulting from excessive perspiration, and lessen its irritating properties.

Exercise.—The amount of exercise and rest which are best suited to maintain perfect health in the tropics depends much on the habits of the individual. No exact rules can be laid down, and in the majority of cases it would be unnecessary to do so. Life in the tropics depends on the occupation of those whom force of circumstances oblige to reside there, for few there are who would endure the heat of a hot season on the plains of India, unless compelled to do so.

A certain number of hours of sleep are necessary to restore exhausted nature, but in a large majority of cases this is overdone. Sleep should never be indulged in immediately after partaking of a heavy meal. During that time digestion is most active. Sleep also is generally "deep"; perspiration is profuse; and the supply of blood is liable to be drawn to the cutaneous surface, leaving a weak and anæmic stomach to perform its functions under the worst conditions.

More sleep appears to be necessary in hot climates than in temperate ones, and it is advisable to allow extra rest to the nervous system.

The time generally recommended for exercise is in the early morning, before sunrise. This is not always the best time to select, and in many cases it is advisable to choose another hour for this purpose. It is true that in the early morning hours the air is fresher and cooler; but when the greater part of the night has been passed without sleep, due to excessive temperature or some other cause, it is unwise to force extra work on the body when rest is necessary to restore equilibrium. It is only a poor repose that the early hours of night bring in the hot season, and it is an error to make any hard and fast rule on this point.

If, however, sound and refreshing sleep is obtained at night, an early morning ride will offer the best means of procuring exercise.

If sleep is required during the day, and it generally is necessary, the best time to take it is during the afternoon, about three hours after partaking of food. Rest at that time refreshes and invigorates the whole body.

For a man doing ordinary work, eight or ten hours' rest is not excessive, and many men require even more than this. It is said that one of the chief causes which contribute to the premature old age of soldiers in the tropics is night-guards, in which rest is broken, and which, owing to sickness and other causes, come round too frequently. More sleep is necessary for those who have much anxious work to do, especially brain work. The amount of sleep required by individuals in the tropics depends much on the same rules which guide us in temperate climates, and varies with the work done during waking hours. No condition is more trying than that produced by sleeplessness; and when this occurs it is necessary to cease work, and seek change of climate as soon as possible.

In India sleeplessness may be one of the first symptoms of a breakdown, and there is little to be gained by struggling against it. The climate and temperature are answerable for this condition; and ten days passed in the cooler surroundings of a hill station is the very best remedy. Narcotics should be avoided, although it is often better to have recourse to them than to pass a restless night, to be followed by a day of hard work. They should only be resorted to as a temporary measure, and with a knowledge that a complete change of scene and abandonment of work is the true remedy. Sleeplessness in the tropics is sometimes due to exhaustion; when such is the

case a cup of good beef-tea, with a little light wine added, will produce refreshing sleep.

The practice of turning night into day is an injurious one. Sleep during the day does not compensate for the loss of it at the proper time. It is a well-observed physiological fact that between 2 A.M. and 3 A.M. the heart's action is most feeble, thus clearly pointing to the necessity for rest.

Exercise is necessary in tropical climates. The tendency is to lead a sedentary life, although during later years the folly of doing so is better understood. Exercise increases the processes of assimilation by aiding digestion; absorption is more rapid, and the circulation through the liver more vigorous. The peristaltic action of the intestines is also increased; and constipation, so frequently experienced in tropical countries, to a great extent removed. During active exercise the quantity of carbon dioxide in the expired air is largely increased, and so the tissues of the body are relieved from any accumulation of that element. At the same time, it is not wise to overdo exercise in tropical climates.

In consequence of the effect which residence in a hot country has upon the physiological processes of man,—especially Europeans,—the metabolism or tissue change of the human body is exaggerated, if not actually excessive. The necessary effect of this is the throwing into the circulation of a large amount of the products of muscular waste. Though our knowledge at present regarding these products is limited, still we know sufficient about them to realise that they are in the main stimulants of the vasomotor nerves. Now, one of the most marked and deleterious effects of a prolonged residence in the tropics is enfeeblement of the vasomotor centres and nerves; and prolonged stimulation of their weakened centres by the circulation in the blood of bye-products of excessive metabolism is followed by their exhaustion, with the subsequent or consequential phenomena constituting fever. This train of events is not uncommon among soldiers on the march, and among others called upon to perform great exertions in tropical countries.

It is thus seen that no rigid rules can be laid down; it is, in fact, quite impossible to define what suitable exercise should be.

In India riding is the best exercise, and should always, if possible, be practised. The comparative cheapness of a horse or pony in that country makes it universally available. It is of great use in hepatic congestion and constipation, two of the chief ailments incidental to tropical life. Respiratory movements are also increased, the respiration being deeper, and as a result the blood is better purified. Cycling has recently been introduced into India, and promises the

best results. It is an exercise well adapted for those who suffer from hepatic disorders and constipation.

The Dwelling.—In the tropics the choice of a site is not always possible, except for a temporary hut or camp. As a rule, permanent habitations will be found in most countries where the exigencies of life oblige man to live and work. In India these are either in one of the large towns or in cantonments, as they are called.

The selection of a site in the tropics is of much more importance than in temperate climates. New country is being constantly opened up for new stations, either civil or military, and it is therefore well to consider what is the best site on which to erect a dwelling.

In former years nearly all Eastern cities were built on the banks of rivers or in their immediate vicinity, with the object of obtaining an easy and ample supply of water, and of getting rid, with the least labour, of the refuse of men and animals, regardless of the fact that by doing so they were polluting the water for those living on the banks below them.

As an example of this may be mentioned the town of Cawnpore in India, which is built on alluvium. Such places have always had a high death-rate, and have suffered severely from epidemics.

Granite, metamorphic, and trap rocks afford healthy sites; water runs off rapidly, and marshes are not frequently to be met with; but when they are weathered and disintegrated they are said to be unhealthy.

The clay slates are also healthy; these do not hold water, and are generally found at some height; water from these rocks is bright, sparkling, and clear.

Of the limestone and magnesium rocks the former are good, hard oolite being the best, and magnesium the worst. Marshes are, however, common. Water is very hard, clear, and sparkling. It is not, however, desirable to build on magnesium limestone if it can be avoided.

Chalk, unmixed with clay and permeable, forms a very healthy site; but if chalk is underlaid by fault clay, it is very likely to prove malarious. Gravels are always healthy, except when they are much below the general surface, and water rises through them. Sands are healthy if they are pure and of considerable depth. The unhealthy sands are those which are composed of siliceous particles and some iron, held together by vegetable *débris*. Sands are also unhealthy from underlying clay near the surface, or from being so placed that water rises through their permeable soil from higher levels;

or they may be unhealthy if they contain soluble mineral matter, as carbonate of magnesia and lime salts.

Clay, dense marls, and alluvial soil are always to be regarded with suspicion; they hold water which neither runs easily off nor through them. The air over these soils is generally moist, and they are subject to frequent mists; marshes are common, and the water supply is in most cases impure. Vast tracts of country in India are made up of soils of this description, and, unfortunately, are the sites of some of the principal cities in that country.

There is no evidence to prove that sites on cultivated land are unhealthy, except in the case of rice fields, which are not only damp in themselves, but send up a large amount of organic matter into the air.

Old burial-grounds should not in any case be occupied; however long they may have remained unused, they are always unhealthy. Made soils should also be avoided.

If a site on sandy soil is occupied, care should be taken to prevent surface pollution of any kind; the power of this kind of soil to oxidise any organic matter added to it is limited. During the hot weather it is dry on the surface and for some distance beneath, a condition which does not favour the nitrifying process, for the completion of which a certain degree of soil moisture is necessary.

Density of population is one of the chief factors which influences the health of a district, not only in temperate climates, but to a much larger extent in the tropics. A free movement of air around the dwelling is essential for the health of its occupants, and, to secure this, each house or bungalow is placed in its own compound, with a free access of light and air on all sides.

High walls or a superabundance of trees, except inasmuch as sometimes they afford protection from malaria, should not be permitted; nothing tends to cause greater heat than stagnation of the surrounding air.

The dwelling as usually erected in the tropics is raised on a solid plinth, leaving no direct communication between the subsoil air and the air in the rooms. This plan is a good one. An alternative plan would be, that floors should be raised, that there might be free perflation of air beneath them. Against this latter plan it is urged that these open spaces would harbour dirt, etc.; some method ought always to be adopted so as to completely cut off the entrance of ground air into the dwelling, either by a concrete basement, or by the free circulation of air beneath the floor. A damp-proof course should be carried round the entire basement a little above the ground, and also at all the piers of the arches, to prevent damp rising.

Every dwelling should have a verandah all round; this should not be less than 8 feet in width, and it should be ventilated so as to carry off the hot air. If the verandah is high, in order to shelter the rooms from the rays of the sun a series of louvres may have to be carried the whole way along between the pillars, sufficiently deep to afford the required amount of shade.

In the subtropics houses are generally of two or more storeys, and except having protection from the direct entrance of the sun's rays by means of Venetian shutters, they do not differ materially in detail from those in temperate climates. The roofs, however, are generally flat, and afford a ready means of enjoying the open air in the evening. In India, except in the large towns, such as Calcutta, Bombay, etc., bungalows are nearly always one storied. They are built of sun-dried bricks, covered with cement. It is not advisable to have very thick walls, as these absorb heat during the day and give it out at night. If possible, double walls with an intervening air space, ventilated by air bricks, through which the air circulates, should be used, as these are the coolest. The inner walls should be plastered; plaster is one of the best non-conductors of heat.

Every dwelling and its verandah should be supplied with eaves, gutters, and pipes to convey the rain water away from the foundation and walls of the buildings. Roofs are either thatched or of tiles. Thatched roofs are cool, but harbour vermin, and are dangerous on account of their liability to fire. Double roofs of well burnt tiles are very generally employed, and if there is sufficient opening to admit of a good current of air they answer every purpose.

Terraced mud roofs are in use in some stations; they are not so well adapted for ventilation, and are apt to crack during the hot weather; they frequently leak, and are difficult to keep watertight. The sloping roof is better adapted in every respect. The ceiling of the bungalow is usually made of canvas stretched across the room and lime-washed. There should be as many doors and windows as can be conveniently placed, and these should be opposite to each other, so as to permit free perflation of air. The ventilation of the rooms should be provided for; this is essential during the hot season. Much, of course, depends on the site occupied, both as regards its position and locality. Some stations are situated in close, sultry, damp positions, where there is slight movement of the outer air, and the temperature varies little between day and night. At others, cold nights succeed very hot days. Some are exposed to hot winds, and in these places it is necessary to close all openings to windward in order to keep the rooms cool and habitable, and to adopt artificial

means for cooling the air by the use of tatties or thermantidotes. Dust storms are frequent during the hot season, and means must be provided for closing all apertures while the storm lasts.

In military hospitals and barracks special means are taken to ensure thorough change of air in the rooms. In private dwellings, as a rule, no such provision is made, although it is desirable that there should be some plan adopted to secure a regular change of air besides the usual windows and doors.

The punkah, which is merely intended to agitate the air, really partly acts as a ventilator, as it displaces masses of air; it does not, however, insure thorough change, and may displace impure air only; it seems correct in principle, and is universally adopted in those parts of India where the heat is excessive. Tatties, which are made of kuskus grass, if kept wet, moisten and cool the air passing through them. They are, however, a frequent cause of chill, and although they are agreeable to many, are hurtful to others. The cooling of the air is due to evaporation from the surface of the tattie.

Thermantidotes are frequently used, but they are not of general application; they set air in motion, as well as introduce a supply of fresh air. The incoming air can be cooled by placing ice or damp kuskus grass near the fan wheel through which the fresh air enters.

Tatties are of no use when the air is stagnant or damp, and in such circumstances the ordinary punkah or thermantidote is the best means for securing the movement of the air. At some hill stations, and also in the plains during the cold weather, it is necessary to provide for fires to warm the rooms. Wood is generally used as fuel. Chimneys, as a rule, are very badly constructed; they are made too large and rectangular in shape, in place of being round or square; the result is that they nearly always smoke, the air in the chimney being chilled by the volume of damp smoke sent up by the burning wood. Fires are also necessary during the damp and rainy season for warming and drying the air in rooms. When coal is used as fuel, the ordinary slow combustion grate fulfils all requirements.

The system of conservancy adopted depends on the country and situation of the place. In many places the sewage is received into sewers which have an outfall to sea at some distance from the town, and so arranged that the sewage is carried away by currents, and cannot be washed back on the foreshore or remain impounded in harbours. In such cases all that is necessary is the complete disconnection of all house pipes from the main sewer—except the W.-C. pipe. This should be guarded by an intercepting manhole and

syphon trap at the sewer end, and open free above the house at the house end. The best method of preventing the entrance of sewer gas into the house is to provide for a current of fresh air to flow constantly through the soil pipe. Some form of wash-out closet, on the plan of the "Unitas" closet, is perhaps the best.

In India, Egypt, and some other places, the dry system of conservancy is carried out; the excreta are removed daily in closed carts, and buried in trenches outside the cantonments. This system answers well so far as the immediate vicinity of the houses are concerned, although the process of removal is often attended with an offensive odour. The great advantage of this system is the compulsory and complete removal of the excreta from the immediate neighbourhood of the dwelling. The carts empty their contents into trenches which are dug about 4 feet deep, and covered when within 1½ feet of the surface. There is great difficulty in disposing of the contents of the carts during the rainy season. This is a matter which requires great attention and supervision in the tropics. It is well to examine into the nature of the soil, and to be certain that it is in a condition fit to disintegrate and oxidise the filth deposited in it. Dry sandy soils are unsuited, as these do not contain the elements required to destroy organic matter, and there is danger of infected dust arising from these places. The native population do not use even trenches, or make the smallest attempt to bury or disinfect the excreta, and this may in some measure account for the fæcal odour so prevalent during dust storms in India. It shows the absolute necessity for guarding all food supplies against dust, and especially milk, which is a medium that can readily be seeded with specific infection.

Careful supervision over latrines cannot be too strongly advocated: during the hot weather these should be frequently lime-washed. All receptacles should be covered, and some disinfectant (lime) used which will prevent flies from collecting and diffusing any specific contagion which may be present. This is a possible source of danger, and should not be overlooked.

Water Supply.—The quantity of water required in the tropics is larger than that usually supplied for domestic purposes in this country. For a fairly cleanly household in England, an allowance of 25 gallons per head daily is usually the quantity consumed; this amount includes 3 gallons for a bath, which is too little for the purpose in hot climates. There is, however, a saving in the quantity used for water-closets and the removal of excreta generally, which, as stated, is in India carried out on a modified dry system. In the tropics, consequent on the increased action

of the skin, more water is required for drinking purposes than in temperate climates.

An adult requires daily about 100 oz. of water; about 20 to 30 oz. are taken in his so-called solid food, and the remainder in some liquid form as tea, beer, etc. During the hot weather in India, a man taking any exercise will consume a much larger quantity. The War Office Regulations lay down that 8 pints of water are to be provided on transports for each man in the tropics, and 6 pints out of the tropics, to be issued for drinking and cooking purposes. The following amounts may be taken as approximately near the quantities used:—

For drinking, cooking, etc., . .	5	gallons	per head daily.
Baths, ...	10	,,	,,
Household purposes, . . .	5	,,	,,
Total domestic supply, . .	20	,,	,,

No allowance is here made for the washing of clothes, which is never done in the house, but at some tank or stream generally set apart for that purpose. Water is also required for tatties, watering the verandah and footpaths round the dwelling, and for any animals that it is necessary to keep. An allowance has also to be made for native servants, who with their families generally reside in the compound, and who always consume large quantities of water.

It will thus be seen that the quantity required is very large. The following approximate estimate is not far from the actual amount generally used:—

Domestic supply, . . .	20	gallons	per head daily.
Add extra baths, . . .	4	,,	,,
For each pony or cow, . .	8	,,	,,
For tatties (during the hot weather), .	25	,,	,,
Watering paths, etc., . . .	20	,,	,,
For each servant, . . .	5	,,	,,
Total quantity required, . .	82	,,	,,

In large cities public water companies, as a rule, supply water. The quantity per head consumed in Calcutta is 37 gallons of filtered, and 5·8 gallons unfiltered. Here the weather is not so hot as in up-country stations; tatties are not used, and water is taken in addition from tanks for ablutionary and other purposes. Many wells are also used by the native population, although the water from these is often polluted.

In the larger military cantonments, water supplies from pure sources are being gradually introduced; much, however, still remains to be done. As a rule, the supplies are taken chiefly from shallow wells, varying from 18 to 36 feet in depth, depending on the level of the ground water; and this again is largely influenced by the system of irrigation which has during recent years been developed by the Government. Fortunately, owing to the system of conservancy adopted, there is a total absence of dead wells and cesspits, so that there is less danger from pollution than is usually found in the rural districts in England; but, on the other hand, there is no proper steining of the wells, and as the caste prejudices of the Hindu lead him to perform his ablutions at the well, there is always a danger from surface defilement. The wells are nearly all draw-wells and remain uncovered. In the bazaars and villages the immediate surroundings are often filthy, and these foul the wells during heavy rainfalls.

Ground water in the plains in India is almost stagnant; there is no perceptible flow, and it is unsafe to drink water from a well left unused for any length of time. The drawing of water insures a fresh supply entering through the sides and bottom of the well, and means filtration through the soil of the surface water.

As a rule, well water is fairly good if it is protected from surface pollution. Most of the wells receive their supply from side percolation, few having the masonry carried down to the *mota*, thereby shutting out from direct entry all water overlying it. Wells yield sufficient water generally, except during a very dry season. This is in a large measure the result of irrigation of the land by the vast canals which have been constructed in the country.

In some parts of India, such as the Terai, it is almost fatal to drink water in its natural state, as it produces malarial fever of the most intense character. Such waters, abounding in vegetable organic impurities, may be found in dense jungles where vegetation is abundant and rank. A safe rule to adopt is never to drink water which has not been previously boiled, except it is supplied by a public water company from a source which is above suspicion. This process should not be left entirely in the hands of native servants, as they are apt only to heat the water to a temperature insufficient to destroy specific germs. Water which has been boiled will taste flat and insipid, but habit soon gets over this difficulty. Boiled water can be used with perfect safety for drinking purposes.

The advantage gained by the use of filters is a moot point. The experience of the writer is against their being universally adopted; they are very difficult to keep clean in India, where there

is constant danger of their being fouled by dust, and they require more care and attention than the majority of people give to them.

Spongy iron is unsuited for India in the hot weather; it is impossible to be certain that the material is always covered with water, and if allowed to run dry the iron cakes and is useless; if taken into camp or elsewhere, the various filtering layers are mixed together, and the action of the filter is largely interfered with. Charcoal rapidly becomes foul, and gives out impurities to water, and under the conditions of heat and moisture micro-organisms develop in it. It is almost impossible, in many places, to obtain sand free from organic impurity, and there is much difficulty in properly washing and renewing it.

The widespread indifference and want of knowledge that exist about filters, not only in India but elsewhere, and the belief, which is almost universal, that once a filter is obtained, it may be used without reference to the character of the water passed through it, or to time, inclines me to believe that a greater danger arises from the false sense of security which filters give than would occur by their total abolition; but if they are used they should be carefully guarded from access of dust, and examined at frequent intervals to ascertain that their condition is satisfactory. The filter best adapted for India is, perhaps, the "Morris" filter, which is simple in construction, and can be easily cleaned. The Chamberland-Pasteur filter, lately introduced into this country, is also well worthy of trial. It is very portable, and seems at first sight to be admirably adapted in every respect for India. On no account should sponge or any organic substance enter into the construction of a filter. Water is sometimes turbid from finely-divided suspended matters, which do not subside on its being allowed to stand for several hours. Such water can, in most instances, be rendered clear by adding alum to it (6 grains to one gallon of water), stirring well up, and allowing the suspended particles to settle down.

In the hills of India a notable danger to water supplies exists in the fact that much of the water is conveyed for miles along open aqueducts, natural or artificial. This state of affairs renders them extremely liable to pollution, though the original source may be quite above suspicion. The greatest difficulty is also experienced in obtaining a good water supply. In many localities the fixed salts of lime and magnesia are very large, and goitre is common among the native population. In Dharmsala, in the Punjab, diarrhœa was formerly prevalent, and was caused by minute scales of mica which floated in the water. Water in India is distributed in mussucks by bheesties. Mussucks are made of the skins of sheep; it is well to

examine them and see that they are kept clean; water should not be allowed to remain in them longer than it is actually necessary.

In other tropical countries the same rules should be carried out, and the water intended for drinking purposes should always be boiled before use. Sand filters, which are in general use in some places, are, when the sand is clean, useful in removing suspended matters; but they cannot be relied on as having any permanent effect on the dissolved organic impurities present in water.

In camp and on the march the portable form of the Chamberland-Pasteur filter seems well adapted, and the advantages which are claimed for it are borne out by experience.

The Hygiene of Troops in Times of Peace and War.

The prevention of disease amongst troops in tropical climates is a subject of the first importance to the British army. Out of a total strength of 196,502 men (in 1890), 96,382 were serving abroad. Of this number 67,456 were stationed in India, 27,546 in the tropics or subtropics, and the remainder, 1381, were quartered at Halifax.

It is thus seen how large a number of soldiers are serving in hot climates, and how necessary it is for the medical officers of the army to be familiar both with the conditions of life and diseases incidental to residence in them.

The conditions under which the British soldier serves abroad are very varied. In some places, exposed to a tropical sun during the day and to almost a freezing temperature at night, he has been able to withstand the vicissitudes of climate, by adapting his food and dress to suit the varying states of the environment in which he is placed. Coming direct from an equable climate, and endowed with the vigour of a northern race, he appears able to resist the consequences of alternations of temperature, of intense heat and great humidity, and, at least for a time, to conquer their effects and adapt himself to the new life in which he is placed. As the sanitary conditions which tend to promote health in the tropics are better understood and more acted upon, it is satisfactory to find that both the mortality and invaliding have been reduced to no inconsiderable extent.

The factors which most influence the health of the soldier are: high temperature, with rapid variations and alternations; excessive rainfall and humidity. These tend to produce those diseases incidental to the climate in which he is serving, for each climate has its own class of special diseases, which only flourish within its geographical limits.

Thus in parts of the West Indies, epidemics of yellow fever occur;

in Bengal, cholera; on the coasts of Africa, malarial fevers prevail; and as we pass from the tropical to the subtropical zone, we find the class of disease changes from these acute and continued to the intermittent and less severe forms.

In the tropics too great care cannot be taken in the selection of sites for barracks. Independently of soil, unhealthy sites are: enclosed valleys—these are always damp, and interfere with the free movements of air; so, too, ravines, being subject to currents of air in one direction, combined with excessive vegetation; badly drained ground; proximity to marshes, especially if the prevailing winds blow over them.

The best site is near the top of a hill, where the crest protects from cold winds. As regards vegetation, herbage is always good if closely cut and carefully kept; rank vegetation is unhealthy, and when cut it should be burned as speedily as possible. Trees are useful as affording shade, and keeping cool the ground; they are also useful as barriers against malarial currents of air.

The chief points to be attended to in selecting a site are: to avoid damp soils, and exposure to malarial currents of air; to select a soil as dry as possible, and if necessary to drain it; to avoid the entrance of ground-air into dwellings by raising the buildings, or cutting it off by means of a concrete basement. Ample provision must also be made for the removal and disposal of surface waters, and for protecting the surface of the ground against pollution.

In India barracks are placed broadside on to the prevailing wind, *en échelon;* if the building is on a slope, it should be placed end on to the side of the hill, and not nearer the slope than 20 feet. Experience has shown the danger that arises from congregating too many men in single rooms, where they breathe a common air, which predisposes them to heat-stroke and epidemic diseases. Each man in the plains in India is allowed in barracks 90 feet of superficial and 1800 cubic feet of space. Many of the barracks are two-storied, with a verandah 10 feet wide.

In the hills, where the climate resembles that of summer in England, each man has 60 square feet of superficial area and 600 cubic feet of space allotted to him; the height of the rooms being 10 feet and the width 24 feet.

With regard to ventilation, no one plan will answer, as the conditions of climate vary in different stations. In some, the air is damp, close, and sultry, with very little movement; in others, hot days succeed to cold nights; while in many the hot winds which blow during the day necessitate all windows, doors, and outlets being closed.

Outlet shafts in barracks and hospitals are provided for: the

aggregate area of each shaft should be 1 square inch for every 15 to 20 cubic feet of room space; each shaft is louvred on the four sides above the roof. Both inlet and outlet openings should admit of being temporarily closed during the hot weather. Inlets are formed by a succession of openings placed round the rooms at the eaves. The plan is almost the same as that adopted for barracks at home; but the inlet and outlet area is doubled. In cooling the air, punkahs are used during the hot weather, supplemented by kuskus tatties as already described. The ablution and bath-rooms are placed in separate buildings, and generally connected by a covered passage, open at the sides; this plan prevents damp or impurities entering the barrack-rooms. Walls should, when possible, be built hollow, and ventilated by air-bricks, as they are cooler than solid walls; the inner surface is plastered and regularly lime-washed. Every building should be supplied with eaves, gutters, and rain-water pipes, to convey the rain-water from the foundations and walls of the buildings; the roof is made double, of well-burnt tiles, slanting and well ventilated between the spaces. Thatched roofs harbour vermin, and are dangerous on account of fire.

Terraced roofs are liable to crack during the hot weather, and are difficult to keep watertight.

The conservancy is on the dry earth system, and is generally well carried out in every cantonment.

In some of the hill stations wooden huts are used; but these are being gradually replaced by more permanent buildings; they are on the same plan as those in use on home stations.

The tents used in the tropics (outside India) are the bell tent and the hospital marquee: the bell tent has a diameter at the base of 12·5 feet, a height of 10 feet, and weighs when dry 72 lbs. It has a total cubic space of 492 feet, and is intended to accommodate at home 15 men; ventilation is very defective, the only outlet provided consists of a few small holes in the canvas near the pole. This tent is not adapted for service in tropical countries.

The hospital marquee has two poles, and is made of double canvas. It has the following dimensions:—length, 30 feet; breadth, 15 feet; height of sides, 5 feet; height to ridge, 15 feet; area about 385 square feet; cubic space, 3336 cubic feet; weight, 512 lbs. It is a good tent when not overcrowded, but is considered to be too cumbersome for service, and is being gradually replaced by tents of a smaller pattern. It is well adapted for sick at the base of operations when no buildings are available.

In India the following tents are used:—

European Private's Tent.—With two poles and ridge and double

fly. Length, 20 feet; breadth, 16 feet; height of walls, 5 feet 6 inches; height to ridge pole, 16 feet 6 inches; cubic space, 2373 cubic feet. This tent is used for inland service only, and accommodates 16 healthy men, or 8 sick.

Mountain Service Tent.—With two poles and ridge. Length, 12 feet; breadth, 8 feet; height of walls, 10 inches; height to ridge pole, 8 feet; cubic space, 544 cubic feet. This tent has hitherto been used for field hospitals, and is intended to accommodate 4 sick; but it is proposed in future to use one of lighter construction and less cumbersome.

General Service Tents.—These are of two kinds, a large one, with three poles and ridge. Length, 14 feet; breadth, 14 feet; height of walls, 1 foot; height to ridge pole, 7 feet; cubic space, 686 cubic feet. This tent accommodates on field service 16 British, or 20 native soldiers, or 25 camp-followers.

The smaller one has two poles and ridge; length, 8 feet; breadth, 14 feet; height of walls, 1 foot; height to ridge pole, 7 feet; cubic space, 392 cubic feet; weight, 80 lbs. It is intended for 8 British soldiers, or 10 native soldiers, or 12 camp-followers.

In the French army the *tente conique* is the one generally used; it is ventilated at the top. A galvanised iron ring 12 inches in diameter receives the canvas, which is sewn round it. An opening is thus left, which can be closed against rain by a wooden top which rests on the top of the pole, and this gives 113 square inches of outlet area. The weight is 129 lbs.; cubic space, 1059 cubic feet. It accommodates 16 men. In the German army a conical tent with a single pole is used. Diameter at base, 15 feet; height, 12 feet; weight, 83 lbs.; cubic space, 1050 cubic feet. It holds 15 men.

In the selection of a site for barracks and camps, the quantity and quality of the water supply is of first importance,—as a rule, a good site affords a good water, and therefore the geology and configuration of the country should be studied, as these will also largely influence the drainage of the site.

Reference has already been made to the effect which density of population has on the health of the inhabitants of a place or building, and it is necessary in the tropics to guard against evils arising from this cause. Buildings should not be erected so close to each other as to interfere with the free passage of air and light; there should be no obstruction to the free currents of air through the rooms at all times.

The rules for camps are laid down by Regulations; the following are the most important points:—

1. The length of time troops may be expected to occupy the camping-ground.

2. That order, cleanliness, ventilation, and salubrity are to be ensured.

3. That means of passing freely through the camp are essential.

4. That a straggling camp increases labour of fatigue duties, and impedes delivery of supplies and circulation of orders.

5. That the more compact the camp the easier it is to defend.

In the tropics, the more space, within due limits, that is allowed for ventilation, the more healthy are its occupants. The too close proximity of tents has always been a common cause of camp diseases. It is impossible to carry out surface drainage and cleanliness to such an extent as always to allow the maximum number to be placed on the ground, or even to approach the population of cities, where provision is made to provide against the risks of overcrowding by proper paving and a well-organised system of drainage. There are 3,097,600 square yards in a square mile, and assuming that there are 15 men to each bell tent, the following table shows the different densities of population per square mile:—

No. of Square Yards per Tent.	No. of Tents per Square Mile.	No. of Troops per Square Mile at 15 Men per Tent.
50	61,953	929,280
100	30,976	464,640
200	15,488	232,320
400	7,744	116,160
800	3,872	58,080
1000	3,092	46,464

In the following table, assuming the strength of the military unit to be as given in the first column, and the maximum and minimum superficial area laid down by Regulations for encampments to be as detailed in the second column, the respective densities of population on these surface areas will be as follows:—

	Strength.	Square Yards.	Acres.	Men per acre.	Men per sq. mile.
Infantry Battalion, full size, .	1011	21,600	4·46	226	144,640
,, ,, minimum, .	1011	7,800	1·61	628	401,920
Cavalry Regiment, full size, .	630	34,000	7·02	89	56,960
,, ,, minimum, .	630	15,000	3·09	204	130,560
Battery, ...	154	11,200	2·31	67	42,880
Field Company, R.E., . .	182	7,500	1·54	118	75,520
Bearer Company, . . .	66	8,400	1·73	38	24,320
Field Hospital, ...	145	11,200	2·31	62	39,680

For comparison, the densities of population in some of the most crowded localities in England are given as follows:—

St. Anne's, Soho,	.	196,800	persons per square mile.
Liverpool,	. .	72,833	„ „
Manchester,	. .	56,256	„ „
Birmingham,	. .	33,600	„ „

In camps, even in the most open order, there is considerable compression.

In the arrangement of a camp, short single rows of tents are better than tents in double lines, and the tents should be separated from each other by a space at least equal to one and a half times the diameter of the tent. The camp should be so placed that the prevailing wind may blow freely through it, and the tents kept open as far as possible, as they are not pervious to air, especially if they are wet.

It is not recommended to excavate the ground before pitching tents; the rule is to disturb the surface of the ground as little as possible, and the soil should be well beaten down so as to render it less permeable; if boards are used they should be frequently taken up, and the surface beneath aired and cleaned; the earth may be scraped if dirty, and the surface remade with wood ashes well rammed down. If boards are not to be obtained, a waterproof sheet should be substituted. If straw is used for bedding, it is best to have it made into mats of a triangular shape and 3 or 4 inches thick; these can be exposed to the sun and air, which insures dryness and cleanliness.

The doors of the tents should not be closed at night, and the camp should be visited to see this rule is enforced. The sides of the tents should be raised during the day, and at night, if convenient. A trench 4 inches deep and connecting with a surface drain, to carry off water, should be dug round each tent.

All refuse should be taken to leeward and burned or buried; latrines dug as close as consistent with sanitary requirements, and to leeward of the tents. When in use, dry earth should be added daily, and the whole trench filled in when within $1\frac{1}{2}$ feet of the surface and a new one dug. Lime may be sprinkled daily with great advantage; it prevents flies from accumulating about the trenches, and may obviate infection being disseminated.

It is desirable to change the camping-ground from time to time, so as not to occupy the same site for a lengthened period. Old camping-grounds in the tropics cannot always be occupied with safety.

The food supplied to the soldier in the tropics differs only to a very slight extent from that supplied to him in England.

The Government home ration consists of 12 oz. of meat, including bone (which must not exceed 20 per cent.), and 1 lb. of bread. The soldier is also provided with a "grocery ration," consisting of extra bread, potatoes, vegetables, tea, sugar, milk, etc., for which an average stoppage of 3½d. per diem is made from his pay. In addition, there are certain purchases he makes individually, which are not easy to determine; but generally these consist of cheese, bacon, preserved meats, and fish.

The following table gives the nutritive values of the "free ration" and "grocery ration":—

NUTRITIVE VALUE IN OUNCES (AVOIR.) OF THE FREE RATION AND OF THE GROCERY RATION.

Articles.	Quantity taken Daily in ounces and tenths of ounces.	Water.	Proteids.	Fat.	Carbo-hydrates.	Salts.	Water-free Food.
Meat, . .	12 oz., of which one-fifth is bone.	7·20	1·44	0·81	...	0·15	2·40
Bread, . .	24	9·60	1·92	0·36	11·81	0·31	14·40
Potatoes, . .	16	11·84	0·32	0·02	3·36	0·02	3·72
Other vegetables (taken as cabbage),	8	7·28	0·14	0·04	0·46	0·06	0·70
Milk, . .	3·25	2·82	0·13	0·12	0·16	0·02	0·43
Sugar, . .	1·33	0·04	...	...	1·29	...	1·29
Salts, . .	0·25	...	...	...	...	0·25	0·25
Coffee, . .	0·33	...	...	...	...	...	...
Tea, . .	0·16	...	...	...	...	...	...
Total quantity,	65·32	38·78	3·95	1·35	17·08	0·81	23·19

In this diet there is—

Nitrogen, . . .	276	grains.
Carbon, . . .	4592	,,
Sulphur, . . .	32	,,

This may be taken as the minimum quantity he receives, and really falls short of what he gets under the present excellent system of dieting in the army. It is altogether independent of his private purchases.

The new system of messing, as described in a pamphlet on *The Messing of the Soldier*, issued by the Army School of Cookery, Aldershot, 1892, gives the nutritive value of the "free ration" and "grocery ration" as follows:—Proteids, 5·06 oz.; fats, 2·23 oz.; carbohydrates, 17·64 oz.; salts, 0·94 oz.; total water-free food, 25·87 oz.

In India the ration differs slightly from this; more meat is issued, 16 oz., but its nutritive value is probably not greater than is

contained in the 12 oz. issued in England, as the animals are small and not so well fed. The constitution and nutritive value of this ration is as follows:—

	Quantity taken Daily.	Water-free.	Proteids.	Fats.	Carbo-hydrates.	Salts.
	oz.	oz.	oz.	oz.	oz.	oz.
Meat with bone, .	16	3·2	1·92	1·08	...	0·2
Bread, . . .	16	9·6	1·29	0·24	7·84	0·2
Potatoes, . .	16	3·72	0·32	0·02	3·36	0·02
Rice, . . .	4	3·6	0·20	0·032	3·38	0·032
Sugar, . . .	2·5	2·42	...	...	2·41	0·012
Tea, . . .	0·71	0·7	...	...	...	...
Salt, . . .	0·66	0·66	...	...	...	0·66
Total, . .	55·87	23·90	3·73	1·372	16·99	1·124

In this diet there is—

Nitrogen, . . .	261 grains.
Carbon, . . .	4523 „

The bread and meat are a free issue; the remainder are supplied by the Commissariat for a stoppage of 9 pies (= 1d.) a day. There are also individual purchases by the soldier of tinned meats and fish, eggs, etc. The deficiency appears to be in vegetables, which are difficult to obtain during the hot weather.

This diet is not excessive, and no ill effects have followed on its use. It is doubtful whether the time at which meals are issued is the best; the same rule in this respect is followed in India as in England.

In India, work during the hot weather is impossible in the middle of the day, and enforced confinement to barracks is necessary. Men sleep after their heavy mid-day meal, and wake up feeling languid and depressed. Dinner at a later hour might be introduced with good results.

Men should not parade in the early morning without having partaken of food. At that time the system is lowered by the excess of heat and often by unrefreshing sleep, and food is required to restore the vital processes. It is a well-recognised fact that one is more likely to take an infectious or other disease while fasting than while the digestive organs are performing their proper functions. A cup of coffee and a biscuit should always be partaken of before leaving barracks for any duty in the early morning.

Some persons have advocated a diet containing less meat and more farinaceous food; but those who have had considerable experience

deprecate any change in this direction. The constitution appears to have accustomed itself to a certain class of diet, and it is doubtful whether the digestive organs would tolerate any sudden and complete change.

Theoretically, it would appear that as the body requires to produce less heat in a tropical climate than in a cold one, less heat-producing food is necessary; and this is the case, for in a sedentary life in India less fats are needed and taken. But the soldier's life is not one of rest; it is more or less an active one under special conditions, which require food not so much to produce body heat as to support and maintain the tissues of the body in health. Food supplies the potential energy which is converted in the body into heat and mechanical force, and it must be taken in due proportion to the expenditure induced by exercise. In a hot climate less food is required to keep up the temperature of the body; and if the life of the soldier was one of idleness, his diet should consist of a larger proportion of fresh vegetables and fruits, and less meat. But his life is one of active exercise, particularly in the mounted branches of the service, and therefore an ample diet is required, and it has not been shown that there is any danger of hepatic disease following on its use. Great care should be taken in providing good meat; and as the Commissariat in India generally keep their own cattle, there is no reason why any animal out of health should be slaughtered for food.

There is a universal consensus of opinion that the issue of a spirit ration is injurious, and that beer and wines must be taken in the strictest moderation.

It is difficult to teach soldiers, who are accustomed to take strong beer, that it is advisable to substitute light wine for it when serving in the tropics.

Good light beer, having a low percentage of alcohol, should be provided, and even this should be taken in moderation.

The beer at present made for issue in the tropics is too strong; it contains in some cases as much as 5 and 6 per cent. of alcohol, volume in volume, and this class of beer is unfitted for use in hot climates.

Scurvy is sometimes seen among the men, due to an absence of fresh vegetables during the hot season; the use of fresh fruit should therefore be encouraged, and this is best partaken of in the early morning. When vegetables are scarce, lime juice may be issued.

The clothing should receive careful attention; it is an important factor in preserving the health of the soldier in the tropics. The object of suitable clothing is to maintain an equable temperature of the body, and thus to avoid those diseases induced by chill as

well as to lessen the absorption and conduction of heat, and so remove one of the conditions which favour the occurrence of heat-stroke. Flannel is the best material for undergarments, as it has the greatest power of absorbing moisture, and is a bad conductor of heat; the material should be light and soft, and should be made to fit loosely on the body, so as to give complete freedom of movement, and by doing so to diminish mechanical work; when underclothing is worn loose, a layer of air is interposed between it and the skin, and this prevents evaporation of heat from the body; the collar of the shirt should be of linen, which will not shrink in washing.

It is very necessary to protect the abdominal region, and for this purpose flannel belts are used; they should be made sufficiently wide to encase the whole abdominal region, and be worn loose; if the belt is narrow and tightly drawn round the body, perspiration collects on the upper surface, which is not the case if loosely made and fastened with tapes; it should be made narrower at the top, gradually increasing in size over the hips.

In India, trousers are made of white drill, or khaki cloth, or serge, —an excellent material, especially for Northern India. Drawers are not supplied to soldiers in the tropics, but the necessity for them is evident; and that they are appreciated, can be judged by the number of men who wear them, and who have purchased them at their own expense. Socks are made of wool, and if they are frequently changed answer very well.

White clothing is very largely used in India; it absorbs heat less than any other colour. The objection to white is that it soils very easily, and in many cases "khaki" is used; its absorbing power is very little greater than white. During the rainy season a light serge is better than drill; it makes an excellent and durable garment.

Helmets covered with cotton and provided with puggarees are issued for the tropics; they are light (13 oz.) and durable. The rim should protect from the level rays of the sun, particularly the temples and the nape of the neck. The Tuson helmet has lately been introduced into India; it consists of two bodies, one within the other, forming a complete air-chamber, not only in the crown, but in the brim, thereby completely protecting the temples and the nape of the neck. The weight with chin strap and buckle is 11⅓ oz., with chin strap and spike 14 oz. It has been very highly spoken of. Whether soldiers should not adopt the native head-dress—the "lungi"—is worthy of consideration; it affords excellent protection to the head, and is cool and smart looking.

Boots are supplied of the same pattern as worn in England; they are of excellent quality, and wear well, but their initial hardness

presses on the foot, which is tender and soft from perspiration. The edges are sharp and the seams rough; the result is a larger amount of foot-soreness than there should be. It would be very advisable to issue a softer and more carefully finished boot for use in tropical climates, especially seeing to the fact that length and breadth are allowed for so as to permit of the foot expanding during walking.

In India "putties" are used; worn over the trousers, they give protection and support to the leg, and are found most comfortable; they are better than leather gaiters, which frequently press on the ankles and instep.

The soldier's kit is divided into his "service kit" and his "surplus kit." The former (except in India) he carries on his person, the latter is carried for him.

The following table shows the weights of the present equipment, in different marching orders, which forms the infantry soldier's kit:—

1. *Service Marching Order.*

	lbs.	oz.
1 Valise,	1	8½
1 Pair of braces and chapes,	0	13½
1 Waist-belt,	0	12½
2 Pouches,	1	3
1 Haversack,	0	9½
1 Mess-tin, cover, and strap,	1	11
1 Water-bottle and carrier,	1	0
2 Coat-straps,	0	5
1 Frog for side-arm,	0	3½
1 Greatcoat,	4	6
1 Cape,	2	0
1 Rifle and sling (magazine),	9	8
1 Bayonet and scabbard,	1	1
1 Magazine pouch,	0	4
1 Spade,	2	9¾
Total,	27	15¼

2. *Home Marching Order.*

2 Pouches,	1	3
Haversack,	0	9½
Water-bottle and carrier,	1	0
Greatcoat and cape, with slings,	6	11
Mess-tin, cover, and strap,	1	11
Valise,	1	8½
Pair of braces and chapes,	0	13½
Rifle and sling,	9	8
Bayonet, scabbard, and frog,	1	4½
Waist-belt,	0	12½
Total,	25	1½

3. *Light Service Order.*

	lbs.	oz.
Same as service marching order, .	27	15¼
But without valise, braces, and chapes, .	2	6
	25	9¼
Add waterproof sheet, . . .	4	4
Total, .	29	13¼

Additional Weight carried on Service.

Field kit, ...	5	6
Rations, ...	2	0
Water (in bottle), . . .	1	8
Ammunition (magazine), . .	6	4
Waterproof sheet, . . .	4	4
Total, .	19	6

The valise holds the following articles:—Emergency ration; towel and soap; clothes brush; hold-all, complete; housewife, fitted; pocket-ledger; one pair spare boot laces; worsted cap; shirt, or boots, or trousers, as ordered; one pair of socks; canvas shoes; cape (carried under flap).

It is made of japanned canvas, and is carried on the back of the shoulders; and it is so arranged that it adapts itself to the width of the soldier's shoulders.

The ammunition is carried in two pouches; the right hand is the "expense" pouch, containing thirty rounds; the left being the "reserve," holding forty rounds.

The mess-tin is carried on the top of the greatcoat or under the coat, when this is carried on the shoulders, or it may be fastened to the waist-belt; in each case it can be detached without interfering with the rest of the equipment.

The great feature in this equipment is that each article can be taken off separately without interfering with the rest of the equipment; and, similarly, changes from one "order of dress" to another can also be effected with great speed, whilst the whole can be put on or taken off in a few seconds by the soldier without any extraneous assistance.

The hygiene of troops in war is of the first importance to the British army. Since the close of the Crimean War, when the first Royal Commission was appointed to inquire into the sanitary state of the army, England has been constantly at war, and these have all been undertaken—with one exception—in tropical or subtropical

countries. War in hot climates has been the normal condition of the British army, and still continues to be so.

Until the Egyptian War of 1882 the mortality and invaliding from disease were far more destructive to our soldiers than the deaths and wounds received from their enemies. It is now the great aim of the army surgeon to preserve the health of the army, so that the mortality from wounds on active service exceeds that from disease, thus reversing the history of all previous wars.

When war has to be undertaken in the tropics, the selection of the season is the first consideration. The effect of climate on the individual has been pointed out, and it has been shown that temperature and humidity are the two principal factors to contend against. As regards the former, the variations of temperature deserve more consideration than the degree of absolute heat.

The influence of rainfall in the production of malaria and other diseases is well known.

The geographical distribution of disease should also be considered, as this largely influences the precautionary measures necessary; and it is also well to make a careful study of the diseases peculiar to the country about to be occupied. In the tropics we find malaria to be of a severer type than is generally the case in the subtropics. Yellow fever prevails within certain well defined limits, while cholera is endemic in others. Conditions which in cold latitudes have no effect, in the tropics intensify the character of many diseases. An example of the good results attending the proper selection of season for campaigning is the Ashantee War of 1874. The troops employed were picked men who were not previously debilitated by climate; they were carefully fed, and provided with proper equipment, and the campaign was undertaken in the dry season—January and February. The result was, the expedition was a complete success; while the mortality of the troops disembarked was only 3·14 per cent.

Before proceeding on active service each man undergoes a medical inspection, and, as far as possible, those who are unfit are kept back and sent to their depôt. It is not possible to select only those whose temperament is suited for active service in the field—to eliminate all men of a nervous or lymphatic temperament, because their sustaining powers to resist and overcome hardships incidental to war are feeble, would be to deplete the ranks of an army unduly. These men must take their chance, and the hardships and excitements they undergo not infrequently improve their condition. Many believe that the army is composed of too young men, who are likely to break down under any continuous hard work. Lord

Wolseley does not share this opinion, and considers it is fully equal to any army in the past which was composed of older men. The truth perhaps lies midway. The soldier ages much more rapidly than his fellow countryman in civil life; the old soldier suffers from diseases of deterioration induced by climate, by night duties and fatigues, and to a large extent from his indifference in regard to his own health, and the latitude he takes in alcohol and sexual excess. A very large number contract syphilis, for which they are invalided; or, if they are permitted to serve on, they do so with an enfeebled constitution. The young soldier has only commenced his career; as a rule he is temperate, and, if he is lucky enough to escape from venereal disease, his physical condition improves under the regular life and discipline to which he is subjected.

Lord Roberts, whose experience of tropical warfare is unequalled, is against the employment of very young soldiers, and considers that only those who have reached 25 years of age should be sent on active service in the tropics; this is the age when the period of growth is completed, and the muscles attain their full development. Soldiers under 21 years of age fill the hospitals, as they are unable to bear any long continued exertion without taxing their strength beyond endurance; but, if only required for peace service in England, they can at this age be brought, by judicious training and by a proper amount of exercise and diet, to a state of development which will insure their efficiency in war. The experience of other armies is the same. Morarche, writing on the French army, states that the ages between 25 and 35 are the best for active service in the tropics. The younger the soldier the greater is the tendency to contract disease, and especially those diseases incidental to campaigning, such as dysentery and fevers.

In the tropics, also, partial acclimatisation plays an important part. Seasoned troops suffer less than those newly arrived in the country; this is partly due to weakly men, who are unable to stand the new conditions under which they are placed, being invalided or otherwise disposed of, as well as to the fact that they have learned intuitively how to live in the tropics.

On the other hand, too long residence induces tropical diseases, which are equally a cause of inefficiency. It is useless sending on service a regiment saturated with malaria from long residence in a malarious station,—the men have deteriorated, and are physically unable to stand the hardships of a campaign.

In the strict sense of the word, acclimatisation for the soldier is impossible; such as are said to be acclimatised have only accustomed

themselves to the environment in which they are placed; but, in doing so, they have probably contracted constitutional diseases which render them inefficient when that environment is changed.

The feeding of an army in war is one of the most difficult questions to decide and carry out. The quantity of food required depends on the amount of work which has to be done; and the training of the soldier for war demands an ample diet in peace. This has now been fully recognised and provided for. On taking active service in the field, a special scale is fixed by the Secretary of State for War, according to the climate and circumstances of the expedition. The following is adopted as far as possible, and may be taken as a type for a war ration:—1½ lb. of bread, or 1 lb. of biscuit, or 1 lb. of flour; 1 lb. of fresh, salt, or preserved meat; ⅙ oz. of tea; ⅓ oz. of coffee; 2 oz. of sugar; ½ oz. of salt; $\frac{1}{36}$ oz. of pepper; ½ lb. of fresh vegetables when procurable, or 1 oz. of compressed vegetables; also ½ oz. of lime juice, with ¼ lb. of sugar: and 2½ oz. of rum, when ordered by the general officer commanding, on the recommendation of the principal medical officer.

A war ration should contain at least 400 grains of nitrogen and 5000 grains of carbon.

The following ration, which under ordinary circumstances could be easily supplied, is recommended:—Bread, 1½ lb.; fresh meat (without bone), 1 lb.; peas or beans, 3 oz.; potatoes or green vegetables, 1 lb.; cheese, 2 oz.; bacon, 2 oz.; sugar, 2 oz.; salt, ½ oz.; pepper, $\frac{1}{20}$ oz.; ground coffee, 1 oz.; tea, ½ oz.; red wine, 10 oz. The nutritive value of this diet is: proteids, 5·6 oz.; fats, 3·45 oz.; carbohydrates, 16·6 oz.; salts, 1·37 oz. In this diet there are 410 grains of nitrogen and 5000 grains of carbon.

Bread should always be issued as long as possible; salt meat should never be used for many days in succession, and with the present excellent supply of preserved meat in tins this need not be done; if no vegetables are obtainable, lime juice should be issued.

In the tropics it is sometimes advisable to increase the vegetables, and diminish the quantity of meat issued, if the troops are not upon active service. When fresh vegetables are not procurable, preserved vegetables may be substituted, but not compressed vegetables, which contain but a small proportion of their salts, and therefore possess little antiscorbutic properties. Vegetables are often found among the natural products of a country; melons and potatoes were obtainable during the Afghan War.

The cooking of food supplies is often a difficulty in war, and men have been frequently kept for many hours without food; this tends to exhaust their energies when these are most needed. It can only

be avoided by a carefully organised regimental system, which includes a good supply of condensed and ready-cooked food that can be made available on emergencies. Warren's compressed steam boilers are admirably adapted for this purpose; by means of these soup with vegetables is always ready, and when the troops are ordered to march the pot can be placed on the waggon, and the soup can be issued at once at the end of the march. These are used in addition to the ordinary means of cooking practised in camp life, and the contents given in addition to the rations supplied by the Army Service Corps. The cooking utensils for an army should preferably be made of block tin, and round in shape, so that they can be "nested"; this shape is also easier to keep clean.

Water supplies must be carefully guarded from contamination, and the source ascertained with as much precision as possible. All water should be boiled before being used for drinking, unless it is proved to be pure and fit for use. Norton's tube well and Bertier's pump are generally used to raise water, but the former is liable to choke if the boring is made through sand. At the base and along the lines of communication the water, if necessary, may be subjected to some process of chemical treatment or filtration. Water from stagnant pools, or from sluggish rivers flowing through jungles, or from marshes, should never be used without being previously boiled. Water is sometimes turbid owing to fine silt being suspended in it; the addition of a few grains of alum will render this water clean after the deposit has subsided. If much organic matter is present, it is well to add a little Condy's fluid until a pink colour is discernible, after which the water may be used for cooking purposes, making tea, etc. Filters, as a rule, are of little use when an army is on the move; they are not adapted to the rough life, bustle, and excitement incidental to campaigning; and it is doubtful if, under the conditions of modern warfare, transport will be available for the old-fashioned filter. The one that seems best adapted is the Chamberland-Pasteur filter, which is very portable, tolerably rapid in its action, and is said to sterilise water. For the lines of communication, and at the base, a pure source of supply is generally available, either from wells or rivers. The "Morris" filter may be used at these places if it is thought desirable to filter the water.

When the base of operations is on the sea coast, it may be necessary to distil sea water. Care should be taken that the water is clean, and not drawn from a port or harbour to which sewage gains access, as ammonia and other matters, volatilised at the temperature of boiling water, pass over into the distillate, and may produce diarrhœa. Distilled water is insipid, and requires aeration

before use; it is best taken with a little lime juice. Alcohol in the form of spirits is not only not necessary, but destructive to the health and discipline of the soldier in war. The issue of a daily spirit ration has ceased, and the advantages gained by its discontinuance are so great that it can never be reintroduced as part of the soldier's ration in war; it neither gives strength to the body nor sustains it against disease; it is not protective against cold or wet, and rather increases than mitigates the effects of heat. There may be exceptional cases in military service when a ration of spirit may prove of advantage, and for this reason the Regulations sanction its issue under special authority.

The use of red wines is sometimes indicated; they were issued in Egypt with good effect, and have a slight purifying effect on water, due to the tannin they contain. The kind of clothing that is best adapted for war has already been indicated. Woollen underclothing should always be worn; the abdomen should be carefully guarded against cold by wearing a flannel belt; the coat should fit loosely—the Norfolk jacket is perhaps the best shape, and it should be well provided with pockets. "Putties" afford the greatest support and protection to the legs. Woollen socks should always be worn, and at the end of the march the soldier should be instructed to bathe his feet and put on dry socks.

Reference has already been made to the foot gear, and to the disadvantages of the present boot. Softer leather should be used, and more care exercised in the finish of the boots. The fastening by means of laces is also objectionable—particularly on service—as taking up time, and the difficulty of replacing broken laces; a leather strap and buckle might with great advantage take their place.

Each soldier on service is provided with a waterproof sheet; it is used to sleep on, and affords protection against damp. Two blankets are also provided; these should be frequently exposed to the sun and air.

Marching in the tropics is undertaken under greater difficulties than in temperate climates. The temperature of the air is relatively higher, and therefore in a given quantity of air less oxygen is inspired; respirations are also more shallow and after a time less frequent, so that there is a less capacity to oxidise effete products in hot climates. In addition to the fatigue of marching, there are also the heat, dust, and often insufficient food; these can be mitigated by marching in the most open order possible, by separating, when possible, cavalry and artillery from the infantry, and by securing an ample and properly cooked ration; but even then the mechanical work done is greater in the tropics. In war the length of marches

varies; as few as 6 and 8 miles in a day only have been done, and as many as 20 miles. A forced march has been made in which 30 and 40 miles have been covered within twenty-four hours. As a rule, the larger the force the greater is the fatigue entailed. A single regiment will march 20 miles in eight hours, while a division would probably take twelve or fourteen hours, including halts. It is on the heart that the effects of long continued marches in the tropics is first seen; the condition known as "irritable heart" being the result, and the chief factor in its causation is over exertion due to long continued marches. Vicissitudes of climate and fever may also be included as exciting causes.

The best time for marching is in the early morning; night marches have always been followed by increased sickness. Night should be devoted to the purposes of rest, so as to refresh exhausted nature. In the tropics there is no rest in tents during the day; the temperature, the glare, and flies prevent sleep, and men soon break down when deprived of it.

Before starting on the march, coffee or cocoa and biscuit should be provided; and, if possible, breakfast should be arranged for, farther on. Open order and free movement should be maintained, so as to reduce temperature and mechanical labour. During the march, water should be freely supplied to the men. The skin is a great eliminator of effete matters, which, if retained, tend to produce such diseases as heat stroke; anything, therefore, which interferes with its action, such as an insufficient quantity of fluids that aid in transpiration, induces suppression of the function of the skin, and favours the production of heat stroke. Cold tea is one of the best drinks when marching; it lessens nervous activity, and increases the action of the skin.

The use of quinine as a prophylactic has been recommended. The experience of the writer of this paper is that when given in small doses daily, it has no effect in either preventing an attack of malarial fever or mitigating its severity, and its use should not be continued over any lengthened period. If a belt of malarious country has to be passed through, a large dose of quinine may be of some advantage; but the march should be made through such a place as speedily as possible, and protection sought by quitting the part with the least possible delay, or by such means as selection of site and drainage, if it has to be occupied, rather than seeking to combat it by means which have hitherto failed. This has also been the experience of medical officers in the Ashantee War, and in the War of the Rebellion in America.

On the march the surgeon must be constantly on the look out

for heat stroke and the effects of fatigue, as evidenced by increased action or failure of the heart, and timely aid afforded to those who require it. The danger is greatest on those days, so frequent in the tropics, when there is little movement in the air, and the air itself is hot and sultry.

Marches undertaken under these climatic conditions favour the production of heat stroke by a stoppage of the cutaneous perspiration which should be encouraged, especially when increased mechanical work is done; frequent bathing of the hands and feet, and drenching the head with cold water, are among the best preventive measures in these cases. Ample provision should also be made to carry men who fall out. Nothing interferes so much with the onward progress of an army on the march than men constantly falling out; it has also a depressing effect on the men themselves. To reduce mechanical labour to the lowest limits possible, and thus to eliminate, as far as can be, fatigue, which is one factor in the production of heat stroke, men's kits should be carried for them; long and rapid marches have never been successfully made with men who are heavily weighted, and the large number of articles and weight of the soldier's kit has always been a fault in the British army. Robert Jackson truly states: "It is a mistake to multiply the equipment of the soldier with a view to adding to his comfort." To summarise: when marching in the tropics, the most open order must be maintained, not only between the men, but between bodies of troops as well; the clothes and equipment should be adapted to the work the soldier is called upon to do; proper food and pure water must be supplied, and in abundance; no spirit ration should be permitted; the weights carried should not be greater than is absolutely necessary; there should be regular halts at short intervals for short periods; and, if possible, one day at least in the week a regular halt made, when men should take the opportunity of washing, etc., but there should be no drill.

If marches are undertaken during the rainy season, a change of dry clothing should be provided at night. Men will bear being wet during the day, and no harm will result from it; but in war, when they are frequently wet by night as well as by day, the hardiest men will break down. If cholera appears during the march, the site of the camp should be immediately changed. It has been recommended to march at right angles to the wind; this is not always possible, but it is possible to move away from the infected area. The sick should be separated and placed to leeward, and all discharges disinfected; strict watch should be kept over the latrines, and separate ones used for any

body of infected men. All water used for drinking purposes should be carefully examined, and precautions taken against its being contaminated.

In India the bheesties should be paraded to see that before leaving the infected camp their mussucks are empty; and no water should be taken by them from an infected site. A regiment attacked by cholera should be completely separated from other troops, and no regiment should follow in its wake. Towns and villages in which cholera prevails should not be entered by troops.

In war, men frequently have had to bivouac, and probably will do so in future wars much more than in the past. Bivouacking has this advantage, that there is no overcrowding, but the exposure to the vicissitudes of climate and variations of temperature are much greater than in camps. In the German army, bivouac tents are provided which have been favourably reported on. The component parts of the tent poles are distributed among as many men (two at least) as are meant to be sheltered by it; the canvas part, which has the appearance of tanned waterproof canvas, is rolled round the soldier's overcoat, which is strapped down on the top and sides of the knapsack; and in bad weather this tent section may be unrolled and worn as a watertight poncho by the bearer. In the British army no provision is made for a shelter tent.

In all cases when men have to bivouac, two blankets and a waterproof sheet should be issued to each man; two poles and a ridge-pole can be improvised on which blankets can be stretched, and this will afford covering for two men; the greatcoat should always be carried on such occasions.

The camp followers will always demand attention. As a rule, they are badly clothed and fed, and unless precautions are taken, epidemic diseases are likely to occur among them, and to spread to the fighting line; proper clothing and proper food should be provided, and their general sanitary condition attended to. Separate latrine trenches should be dug for their convenience, and they should not be permitted to use other places.

The disposal of the dead in war has always been a difficulty, and with the present modern weapons of precision is one likely to be largely increased. Burial has hitherto been the universal custom, but it is not always possible to carry this out so as to render the place where the bodies are disposed of safe and free from objectionable odours. These objects were not attained at Metz, where, months after the battle had taken place, attempts to destroy the decomposed bodies had to be resorted to, which were only partially successful.

An army which has to follow on any advantage gained after a battle, or is obliged to retreat, is unable to dispose of the dead by means of burial.

In the tropics decomposition rapidly sets in, and the effect on the *morale* of the men who are ordered to perform the duty of burial is bad. At Sedan, burning, by means of pouring tar on the bodies and setting fire to the pile, was practised; this system tended to disorganise the troops.

If burial is the system adopted, it should be as deep as possible, and charcoal, if procurable, should be thrown over the bodies; if it cannot be obtained, sawdust and sulphate of zinc or carbolic acid may be employed. Quicklime has also been commonly employed, but it is less useful.

Speedy disposal of the dead is absolutely essential on the battle-field, not only in the interests of those living near, but of the wounded and the troops in the neighbourhood. One system alone appears to solve the present difficulty, and commends itself; that is cremation. It is the only system which insures speedy removal of the dead, and fulfils all the requirements from a sanitary point of view. It abolishes the practice of interring numbers of bodies in one large pit, and does away with the necessity of large quantities of disinfectants; there is no repulsive feeling connected with its use, and therefore it can only have a salutary influence on the troops, while thorough disinfection of the battlefield is gained; it renders the scene of action at once clean, the air pure and whole-some, and the end is attained with the least labour and at the minimum expense.

Change of Climate.

The effects of climate on health is a subject of more than ordinary interest, affecting, as it does, all classes, but more particularly those who are forced by circumstances to spend a large part of their life within the tropics.

Climate (from *κλίμα*, a slope, from *κλινείν*, to incline) originally signified that obliquity of the sphere with respect to the horizon from which results the inequality of day and night. Now, however, it is generally understood to mean the sum of all those meteoro-logical conditions of a place or region, including not only those of temperature, but also the meteorological environment of a region generally, so far as these exercise an influence on the animal or vegetable kingdoms.

Tropical climates have undoubtedly a deteriorating effect, not only

on the European race and on those from northern latitudes, but also on the indigenous populations. This is known, in the case of the European, from the recognised impossibility that exists of his permanently occupying and colonising tropical regions.

The following are the chief influences which determine the climate of a place :—

1. The distance from the equator.
2. Elevation above the sea level and distance from the sea.
3. Prevailing winds.
4. Local conditions, such as configuration of the surface of the land, nature of soil, vegetation, cultivation of the soil, etc.

The nearer we approach to the equator, the greater is the amount of heat received from the solar rays; this is due to the sun's rays falling almost vertically in intertropical countries, while the rays from the sun strike the earth in an oblique direction at the poles, and have to pass through a greater volume of air.

Temperature is the most important factor in the determination of climate, and the temperature of a place is largely dependent on its distance from the equator. Maps have been constructed showing the places having the same annual temperature, and these are connected by *isothermal* lines. An examination of such maps shows that these lines—or isotherms—exhibit the greatest deflections especially in the northern hemisphere, and that the greatest divergencies are seen over large areas of land.

The temperature of the air is also influenced by elevation above the sea level. The effect that height has is to lower the temperature roughly about 1° F. for every 300 feet of ascent on land. Direct observations made by ascending in a balloon show that the temperature falls 1° F. for every 100 feet ascended, but the fall is not so great on the surface of the ground. This fall is due to the lessened amount of moisture in the air, to the greater movement of the air itself, and to the greater rarefaction due to elevation. It is, however, a fact that valleys are colder than mountain ridges and slopes; this is caused by the down flowing current of cold air along the hillsides, on account of its greater density, as well as by mists, which generally arise from valleys and retain their own cold produced by radiation. It is on this account that valleys at the foot of hills suffer from frosts while higher altitudes escape.

The sea coast is never so hot as the interior of a country at the same level. Cold currents of air pass from the sea and lower the temperature on land. The sea modifies the temperature by moderating the extremes of climate, and the result is to make the climate of the coasts an equable one. There is little variation in the

temperature between day and night; the air, however, is laden with moisture, and the atmosphere is usually free from those organic and inorganic impurities which are generally present in the air of inland places.

The effects of such climates on the body are to lessen evaporation and transpiration of effete products, and there is generally an increase in the body temperature. At the same time, man adapts himself better to a warm and moist climate than to a cold and moist one.

Winds depend on differences of temperature in different places; air flows from an area of high pressure into an area of low pressure; heat renders air lighter, it then ascends, and cold air, being of greater density, takes its place. Air in motion is cooling, causing evaporation from the surface of the body, and thus reducing the temperature. It follows, therefore, that the more rapid the movement of the air, the greater is its effect in reducing temperature. A still atmosphere has the opposite effect. Winds also distribute moisture and temperature; they depend on the direction they come from whether they are moist or dry,—land winds being hotter and drier than those which blow over a wide expanse of water. Hot winds tend to cool the body by promoting evaporation, while moist winds raise the body heat by preventing evaporation from the surface of the skin. In the tropics winds carry dust and organic particles in large quantities very long distances.

The local conditions which influence climate depend also on the aspect of the place, whether it is protected from cold winds by higher ground or by a belt of trees, etc. In the tropics the chief local condition lies in the nature of the soil. Schubler has ascertained by experiment the relative power of absorption of heat of different kinds of soil; that of sandy soil being taken as 100, it follows:—

Sand, . . .	100·0	Clayey earth, .	68·4
Gypsum, . .	72·2	Chalk, . . .	61·8
Pure clay, . .	66·7	Humus, . .	49·0

The hottest ground, therefore, is a sandy soil. The heat in the tropics is often so intense that the natives are unable to remain in these places during the hot weather. On the other hand, clays and humus soil are among the coldest. Herbage lessens the power of soil for absorbing heat, and radiation is more rapid. In India the sands are objectionable from their heat; they radiate heat slowly, and therefore the air is hot over them day and night.

Experiments made by Lewis and Cunningham in Calcutta show that the temperature of the soil varies with the season. During

the hot season the temperature was highest in the air, next highest in the upper stratum of the soil, and lowest in the lower stratum. In the cold weather these conditions were reversed. The colour of the soil influences temperature; white glaring soils reflect the rays of heat as well as those of light.

Trees modify climate and tend to make it equable; they attain their maximum temperature about 10 o'clock P.M., and therefore become reservoirs of heat which they give up at night to the surrounding air, while by day they cool the air by evaporation from the surface of their leaves, as well as by preventing the ground from absorbing the rays of heat. Thus they tend to make the days cooler and the nights warmer.

The rains differ from those which occur in northern latitudes, in so far as that they are periodical in character and fall in the hot weather; in the subtropics winter is the rainy season, while in temperate climates rains fall throughout the year, and may be said to have no season. As a rule, in the tropics the rainfall increases with the elevation. More rain falls in the hills in India than in the plains up to a certain height (10,000 feet), then the fall is less, snow taking its place.

The effects of heat on the functions of the body are largely dependent on the length of residence within the tropics. How soon the body, when it has become once accustomed by residence for many generations in one climate, can accommodate itself to the conditions of another of a widely different character, is a question which is yet undetermined.

At first both respiration and the circulation are quickened. This condition is very transient, lasting only a few months, when the respiratory movements and pulse become lessened, but the vital capacity is somewhat increased. The result, on the whole, is to greatly reduce the total respiratory action; there is, therefore, a lessened quantity of carbon dioxide expired. Rattray has also shown that the average pulse in the tropics is lower by 2½ beats per minute than in the temperate zone.

Europeans suffer in the tropics from anæmia; this is generally the result of long continued residence from which few escape. It is due to a diminution in the red corpuscles in the blood, the result of deficiency of oxygen in the air of the tropics. The digestive powers are impaired, and there is a lessened desire for animal food. There is also congestion of the liver, due in the majority of instances to excess in food and alcohol.

The urine is lessened in quantity, and so also are the urea and salts; this is partially due to the lessened quantity of food ingested.

The result of long residence on the nervous system is to reduce the vigour of mind and body. The climate has a depressing influence, especially if moisture is present, so that there is lessening of evaporation from the surface, and increased body heat is the result. Dry and hot climates do not produce the same result.

There can be no doubt but that the human body is capable of adapting itself to life in the tropics; the skin acts more, the lungs act less, and the circulation lessens as men pass from a temperate climate into the tropical one.

Man becomes seasoned to the new conditions. The process is one of adaptation and seasoning, with elimination of those who are unfitted to withstand the effects of climate. This is the case in military life, where the process of selection is carried on by invaliding, leaving only those behind who can adapt themselves to the climate and environment in which they are placed. Acclimatisation has not yet taken place, it probably will never do so, and is not possible until conditions hitherto little understood have been successfully grappled with. So much for the tropics as a whole.

Let us now consider whether altitude within the tropics will compensate for all or any of the disadvantages which are to be found therein generally, and whether a climate exists at a certain height adapted for northern races. Elevation, as has been pointed out, influences temperature, this diminishing with the height. There is also a much larger proportion of sunshine, a more rarefied air, less moisture in the air, and therefore more evaporation from the skin. The rainfall is, however, increased up to a certain height, after that it diminishes.

Although such a climate would at first sight appear to offer an environment within the tropics suitable to acclimatisation of the northern races, nevertheless there are elements present which detract from the advantages of hill climates; these are, the rarefied air, affording a lessened supply of oxygen, the heat and direct rays of an intertropical sun, and the absence of well marked seasons, which perhaps afford the greatest vitality to the European races. To many, also, the climate is too invigorating, and length of residence does not in their case shape man to his environment. In speaking of altitude and hill climates generally, it is well to state precisely what is meant, as very vague ideas exist on this point.

The height at which troops are generally located in the Himalayas is between 3000 and 10,000 feet above sea level, and having a mean annual temperature of 38° to 67° F. This corresponds closely to the climates of the temperate zone. An example of such a climate is found at Landour, N.-W. Provinces.

The diseases which are most prevalent in high altitudes in the tropics, are inflammations of the lungs and pleura, diseases of the heart and circulatory system, and rheumatism. Stone is frequent; so are goitre and cretinism. Malaria is also present, and in some places hepatitis and diarrhœa.

Persons coming direct from cold climates are more liable to be attacked by tropical diseases than those who have been partially seasoned by residence for a time in a warm climate. For this reason regiments are frequently sent from the Mediterranean stations to India and the West Indies, and experience proves that by doing so a "seasoning" of the troops has taken place, and they are better fitted to withstand outbreaks of epidemic diseases. The influence of tropical climates on soldiers affords the best standard by which such climates are to be judged, as the conditions of service insure that all statistics of sickness and mortality are carefully recorded, while the large numbers under observation afford a surer basis for inference than is likely to be obtained from observations on small and isolated bodies of civilians.

In India, where by far the largest number of European troops are quartered, the climate varies greatly according to the part occupied. The temperature of the southern part of the peninsula, and extending to the centre, is moderated by the elevated tracts of land in these parts; while the north and north-west suffer from hot winds in summer, which blow over the deserts that lie to the north; during the winter the mountain ranges form a barrier to the cold winds from the north, and moderate the cold. Roughly, the climate of India may be said to be of two kinds, that which is found to exist in the plains and in the hill stations. This division is very marked in Bengal, which is the largest and most important Presidency, not only in the character of the climate, but also on its influence on health; the cold and hot seasons are well divided, and the extremes between these well defined, especially in the North-West Provinces and the Punjab.

In the interior of the country the temperature is always greater than on the sea coast, where the climate is equable and uniform, especially towards the south. Winds influence the temperature to a great extent, land winds causing it to rise, while those which blow over the sea lower it and increase the humidity. Humidity is the most important factor in determining the climate in different parts of India; the more moisture that is present in the air, the smaller is the difference of temperature between the hot and cold seasons. Thus Bengal proper is very damp, and the climate is equable though hot; while parts of Scinde and the Punjab are so dry that large tracts of the country remain uncultivated, and much more would be

devoid of vegetation if it was not at times watered by the immense canals which flow through it. In the Punjab the thermometer has frequently risen to 120° F. in the shade during the hot season, while frosts are of ordinary occurrence in the winter. The west coast of the peninsula, and Bengal proper and Assam, may be said to have very damp climates, with no hot season as seen in other parts of India, while the north-western parts are dry, and exposed to dry hot winds. Northerly winds and winds from the north-west are dry, having passed over large tracts of land; those from the south and south-west are charged with moisture, and, blowing over the west coasts and Bengal, make these provinces the dampest and most rainy. In Bengal the north-west winds blow during the months of April, May, and June over the North-West Provinces and the Punjab, and at that time the season is dry and hot.

The following table shows the mean temperature and height above sea level of a few of the principal stations:—

MEAN TEMPERATURE AND HEIGHT ABOVE SEA LEVEL OF SOME OF THE PRINCIPAL STATIONS.

Months.	Calcutta, 21 feet above sea level.	Simla, 7048 feet above sea level.	Lahore, 732 feet above sea level.	Peshawur, 1110 feet above sea level.	Jacobabad, 186 feet above sea level.	Quetta, 5501 feet above sea level.	Madras, 22 feet above sea level.	Bangalore, 2981 feet above sea level.	Rangoon, 41 feet above sea level.	Bombay, 37 feet above sea level.	Poona, 1849 feet above sea level.
January,	65°	41°	54°	50°	57°	40°	76°	67°	75°	74°	72°
February,	70	41	59	52	62	40	77	72	77	75	76
March,	79	50	69	62	74	50	81	77	81	79	83
April,	85	58	81	70	83	58	85	80	84	82	86
May,	85	64	88	80	91	66	87	79	83	85	85
June,	84	67	93	89	96	74	88	74	79	83	79
July,	83	64	89	89	94	77	86	72	78	81	75
August,	82	63	88	87	91	75	85	72	78	80	75
September,	82	61	85	81	88	67	84	72	78	80	75
October,	80	56	77	71	78	56	81	72	80	81	78
November,	72	49	64	58	65	46	78	70	78	80	75
December,	65	45	55	51	58	41	76	67	76	76	72
Mean of year,	78	55	75	70	78	58	82	73	79	80	78
Amplitude of yearly fluctuation (difference between hottest and coldest months),	20	26	35	39	39	37	12	13	9	11	13

The rainfall depends on the monsoon, which is a wind that sets in from the south-west heavily loaded with moisture,—reaching the highlands of the west coast and Upper India, it meets a body of cold

air whereby its own temperature is rapidly lowered, causing rapid condensation and rainfall. As the rainfall depends almost altogether on the monsoon, it follows that one part of the year is very rainy and the remainder dry, or with only a few occasional showers.

In the cold weather in the north-west of India, the wind blows from the land towards the sea, and this season of the year is dry and rainless; while from the middle of June until the end of September, the south-west winds blow and bring rain.

The yearly amount of rain in some of the principal stations is as follows:—

	Average.		Average.
Bengal Presidency—		Bombay Presidency—	
Calcutta,	65·5	Bombay,	50·0
Allahabad,	37·6	Poona,	28·3
Meerut,	28·5	Belgaum,	48·8
Lucknow,	37·6	Mahabaleshwar,	261·4
Agra,	26·2	Madras Presidency—	
Delhi,	27·6	Madras,	49·1
Simla,	70·1	Bellary,	17·6
Lahore,	21·9	Bangalore,	35·6
Sialkote,	36·8	Secunderabad,	28·3
Rawal Pindi,	32·4	Burmah—	
Peshawur,	13·5	Rangoon,	99·0
Quetta,	9·9	Thyet Myo,	45·5

During the hot weather the heat is most intense, just before the setting in of the rains, although this is, as a rule, the healthy season. The diseases most prevalent during the hot months are heat stroke, diseases of the nervous system, and acute liver disease, associated with ardent fever. During the onset of the rains, dysentery and diarrhœa are frequent; and towards the end of the rains, and in the cold weather, malarial fevers, dysentery, hepatic congestion, liver abscess, and renal diseases occur, those who have been long resident in the country being more liable to be attacked.

Age has an important bearing with regard to sickness and mortality in India. As a rule, young soldiers suffer from those diseases incidental to change of climate and youth; these are enteric fever, dysentery, and diarrhœa, also cholera, when that is prevalent, —diseases, in fact, under the control of sanitary measures, and which are largely preventable. For old soldiers newly arrived in the country, the diseases which are to be feared are hepatitis, heat apoplexy, dysentery, and heart diseases, all diseases of deterioration, and frequently favoured and aggravated by intemperance.

As regards fevers, the most serious mortality during recent years has been from enteric fever, which attacks the young soldier in his earlier years of service,—the mortality is between 5 and 6 per 1000 under 25 years; it is less than half this from 25 to 29 years, and less than one-eighth from 30 to 34. The mortality from all

other fevers decreases after 30 years of age. Heat stroke attacks most frequently the old soldier; phthisis causes the largest mortality in old soldiers also; hepatitis is essentially a disease of deterioration; and heart diseases and those of the circulatory system increase with age and length of service in the country.

European troops have been, during recent years, largely located in the hills in the hot season, and the advantage of this practice cannot be over estimated; the men are removed from the debilitating influence of excessive heat, and malarial poison is comparatively slight at the elevation at which troops are quartered.

With reference to age and recent arrival in the country, as being the chief factors contributing to the sickness and mortality of the soldier on his arrival in India, it should be noted that cholera attacks these first as a rule, and that residence seems to afford some immunity, similar to that which exists in parts of the West Indies which are visited by outbreaks of yellow fever. This is seen from the following table compiled by Dr. Bryden:—

	Lucknow, 1864.	Lucknow, 1869.	Benares, 1865.	Meerut, 1869.
Mortality of new troops from cholera,	27	42	9	105
Mortality of old troops, . .	11	11	Nil.	6

The hill stations in Bengal vary slightly in the character of their climate, but possess many features in common. Mussouri, in the North-West Provinces, may be taken as a type of the average of hill stations occupied by Europeans, and the description given applies in its main features to Simla, Kasauli, Naini Tal, Rhaniket, Chakrata, and Dalhousie. In March the frost and snow of winter disappear, and spring sets in with a few showers, after which fine weather prevails for some time. Southerly winds blow as a rule from noon until sunset, when they are succeeded by northerly breezes, which are very cool; but these die away towards early morning. The rains commence between the middle and end of June, and are usually ushered in by thunderstorms. They last until the end of September, and are heavier at some stations than at others; they are generally accompanied by mists and fogs, which prevail during the whole time. It practically means that for those who live in the hills at this time, life is passed in the clouds, these disappearing only in the evening, when they may be seen resting on the plains and in the valleys beneath. After the rains, which last about three months, the

weather is perfect, bright clear days, while the air is bracing and invigorating; the sun is bright and warm, and the atmosphere beautifully clear. Frosts occur during the latter part of October and November, and snow generally falls during December, January, and February. In many hill stations the rhododendrons form a special feature, and add greatly to the appearance of the place; this is the case at Dalhousie, Chakrata, and Landour. The hills are covered with forests of oak and fir, which supply fuel.

Darjeeling differs somewhat from the other hill stations in Bengal by reason of its large rainfall, which lasts for several days at a time, and is very depressing during this period. It is nevertheless a healthy station, and as the immediate neighbourhood is brought more under cultivation, the rainfall probably will be lessened. There are no very heavy winds, the prevailing one being from the south. Mists and fogs are common, the sky is often overcast; the rainfall averages 121 inches annually.

The following table shows the mean temperature in shade, the greatest elevation, and the average fall of rain in inches at the principal hill stations:—

	Name of Hill Stations.	Mean Temperature outside in the Shade.												Ascertained greatest Elevation.	Average Fall of Rain in Inches.
		January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.		
Bengal Presidency.	Darjeeling, .	40°	39°	47°	53°	54°	56°	61°	61°	59°	54°	48°	43°	7421	127·1
	Naini Tal, .	42	46	55	59	64	69	65	66	63	58	55	48	6400	86·6
	Landour, . .	38	38	46	56	63	67	63	61	60	54	45	40	7200	78·4
	Chakrata, .	42	43	51	60	64	67	64	64	63	58	51	46	7052	60·8
	Simla, . .	41	41	50	58	64	67	64	63	61	56	49	45	7048	70·1
	Kasauli, . .	39	39	54	54	64	69	67	65	66	61	53	45	6400	72·0
	Sabāthū, . .	52	54	61	72	75	82	75	75	74	67	60	48	4200	70·0
	Dagshai, . .	39	54	58	63	69	74	69	68	67	64	57	47	6100	71·0
	Dalhousie, .	57	54	69	77	78	85	76	73	72	66	55	50	6000	65·0
	Murree, . .	39	39	49	57	65	71	68	67	65	58	49	43	6344	56·6
	Cherat, . .	...	...	...	...	79	83	79	79	74	61	63	...	4300	13 inches in 7 months.
	Pachmarhi, .	58	62	72	79	83	78	71	70	70	67	60	56	3528	78·0
Madras Presidency.	Ootacamund,	48	51	55	58	59	56	55	55	55	54	53	51	7252	45·0
	Kotagherry, .	59	60	61	63	63	64	65	65	64	62	60	59	6100	55·0
	Wellington, .	55	58	63	65	66	63	63	62	62	61	58	56	6200	48·0
	Coonoor, . .	60	62	68	68	68	65	70	70	70	65	62	62	5161	50·0
	Shevaroys, .	65	65	68	71	71	68	68	68	67	66	66	65	5260	40·0
	Ramandroog,	70	76	80	80	75	73	71	70	70	71	71	67	3400	46·0
Bombay Presidency.	Mount Abū, .	58	60	69	75	79	75	70	68	69	70	64	59	3945	63·1
	Mahabaleshwar	63	65	72	74	72	67	63	64	64	66	64	63	4700	261·4

The mortality from all causes is immensely lessened in the hills in India. Malaria is not uncommon, and has been known to have attacked those who had not previously suffered; still the poison is comparatively feeble.

At the foot of the hills the worst forms of malarial fever are found, especially during and immediately after the rains; remaining one night in those places has been followed by a most severe attack. Phthisis is less prevalent in the hills, and unless well-pronounced, cases do well there. Acute cases benefit during the dry season, if no large amount of lung tissue is destroyed, but fall off during the rains—no improvement taking place, but rather the reverse. Enteric fever is certainly less prevalent; and where this is not so, the cause can generally be traced to an impure water supply.

Diarrhœa and dysentery are common at all hill stations, due to the sudden alternations of temperature, and partly to chill during the rainy season; drinking water has been the cause in many instances; it is generally very hard, and contains lime and magnesia salts in solution. Goitre is common among the native population, and Europeans also have suffered from it, but to a much less extent. Heat stroke is almost unknown, and hepatic diseases are less frequent than in the plains. Cholera is comparatively rare, and where it occurs can almost always be traced to an imported case.

Convalescents from acute diseases, provided there is no organic lesion, do well in the hills; but chronic dysentery, chronic hepatitis, severe cardiac disease, and cases of advanced phthisis are decidedly rendered worse by being transferred for change to a hill climate, as well as those patients who are suffering from rheumatism and secondary syphilis.

The hills are especially suitable for women suffering from anæmia, leucorrhœa, etc., and for children, who appear to gain more benefit from residence there than any other class.

CHAPTER III.

TROPICAL NAVAL HYGIENE.

BY FLEET-SURGEON R. W. COPPINGER, M.D.

General Considerations.—The chief obstacles to the maintenance of a high standard of health among those who live for long periods at sea are—(1) the small amount of air-space available between decks; (2) the use of a dietary consisting largely of preserved foods; and (3) the limited extent of deck-space available for exercise.

When placed amid tropical surroundings the evil effects of the two first mentioned conditions become accentuated; in the former case, on account of the obstacle offered by the heated condition of the general atmosphere to providing for adequate ventilation by *natural* agencies; and in the latter case, because it is more difficult in hot weather than in cold to maintain sufficient supplies of those articles of food which in a hot climate should enter largely into the constitution of the dietary.

A ship, viewed as a habitation, contrasts with an ordinary dwelling house in the following respects:—(1) It is built altogether of wood, or of iron, or of a combination of both; (2) it is immersed in water; (3) it is much exposed to the weather from above; (4) it (if a steamship) contains a large quantity of coal.

1. If made of *wood*, its shell is more porous, and therefore more capable of absorbing moisture, than are the containing walls of a house, whether the latter be constructed of brickwork or of stone. Moreover, wood, by virtue of its vegetable nature, has the disadvantages of being prone to undergo putrefactive decomposition, and also of affording a resting place, and possibly a multiplying ground, for facultative pathogenic microbes. The warmth of a tropical climate, acting in conjunction with moisture and the presence of such organic material, is a condition tending greatly to favour the vitality and growth of micro-organisms.

If the ship be constructed of *iron*, it compares unfavourably with

a house in regard to iron being a better conductor of heat than either brick or stone. From this consideration it follows that in warm climates a large amount of solar radiant heat is absorbed by the hull of the vessel, and this heat, being rapidly taken up by the moist air between decks, renders the restricted air-space available for the crew much less tolerable than in the case of a house on shore in the same region.

2. A large part of the hull of a ship being immersed in water, we may compare this floating dwelling with a house built on a damp foundation. The drawbacks in the case of the ship are that sea water leaks in from below, and that this, as well as slop water and waste oil, which finds its way down from above, cannot be removed by gravitation drainage, but only by pumping contrivances, which are unavoidably less thorough in their action than well-constructed house drains. We have therefore in a ship to cope with a certain amount (small though it may be) of sea water and slop water containing putrescible matter, in the bilge, if it be a wooden ship, or on the floor, if it be an iron vessel. In the case of a wooden ship, the influence of tropical heat upon the sulphates in the sea water associated with the organic matter of the hull itself, or with organic matter otherwise introduced, will promote a decomposition resulting in the production of bilge gases, such as hydrogen and ammonium sulphides. In the case of an iron ship, a somewhat similar decomposition will take place, but to a less extent, owing to the absence of timber; although it is questionable whether the existence of iron sulphide, occurring as an impurity in the metal framing and plating, is not, under the conditions above mentioned, largely responsible for the evolution of gaseous sulphides.

3. A ship is much exposed to the weather from above. We may fitly compare a ship to a two- or three-storied house from which the roof has been removed, thus exposing the upper floor to the influence of rain and sun. The upper deck of a ship has also the disadvantage of being in stormy weather flooded with sea water.

If the hatchways be covered up in order to prevent the ingress of rain or sea water, we abolish for the time one of our most trustworthy means of ventilation.

At sea and in stormy weather awnings have only a limited application. When they are used, and more especially when they are fitted with side curtains, they greatly interfere with the action of all tubular ventilators which do not pass through and above them.

The upper deck is of necessity frequently subjected to scrubbing operations, and thus becomes rough and porous. Sea water is

mainly used for cleansing this deck, while sea water also reaches the deck in the shape of spray and green seas dashing over the ship's bulwarks. From these causes the upper deck becomes in time saturated with sea salt, which, owing to its deliquescent nature, takes up moisture from the atmosphere, especially at night time. When, therefore, in the tropics the rays of the morning sun fall upon the upper deck, evaporation rapidly takes place, suddenly lowering the temperature of the deck, and causing a condensation of aqueous vapour from the air between decks, where the men have been sleeping, on to the under surface of the upper deck. A similar chilling effect is produced when, after a shower of rain, the fierce rays of the tropical sun are allowed to play upon the wet planking of the deck. An unprotected upper deck in hot climates is therefore liable to be associated with rapid changes of temperature between decks.

4. Steamships of large coal capacity and vessels trading in coal may to some extent be regarded as floating coal mines, in which the crew are necessarily living in close proximity to an unstable material which, under certain conditions, may evolve noxious gases in considerable quantities. Coal is sometimes found to contain iron pyrites; and in tropical weather this compound, when exposed to air and moisture, is liable to undergo oxidation changes, resulting in the production of heat and the evolution of marsh gas and hydrogen sulphide. Spontaneous combustion may possibly ensue; but, apart from this, the liberation of marsh gas in the interior of a ship constitutes an insanitary condition for which due provision should be made. There is also some reason to believe that when coal which has been for a long time stacked on shore in malarial localities is taken on board ship, the "materies morbi" of malarial fever may thereby be introduced into the ship's bunkers with disastrous consequences to the crew.

Accommodation for Crew and Passengers.—The berthing and messing accommodation for crew and passengers varies somewhat in different classes of ships. Thus, in men-of-war the crew are berthed mainly "between decks," and the same space is made use of both for sleeping and for messing. Immediately after "turning out" the hammocks are lashed up and stowed in covered receptacles on the bulwarks or on the upper deck, so that the mess-decks are thus entirely cleared of all bedding appliances. In this respect the use of the hammock as practised in men-of-war is much to be preferred to the fixed bunk so commonly seen in merchant ships. Then, the cleansing of the mess-deck is facilitated by the portable nature of the mess-tables and lockers, which are so

constructed that they can be readily slung up so as to leave the surface of the deck clear for sweeping and scrubbing.

Several men-of-war of recent construction have been fitted with lofty and roomy top-gallant forecastles, in which a portion of the crew are accommodated, thus providing better light as well as greater facilities for ventilation by natural means.

The net cubic air space available per man varies a good deal in different classes of ships, and in different parts of the same ship. At a rough estimate it may be set down as 200 cubic feet per man; this estimate assuming that the ship is in harbour, and almost everyone below. However, at sea, when one watch (equal to about a third of the ship's company) is stationed on deck, the total available air-space is augmented by the body-space of the men who are away on deck, while the gross respiratory vitiation is also proportionately diminished. The smaller air-space per man available in harbour is to some extent compensated for by the greater facilities then existing for effecting natural ventilation, as the fact of the ship being on an even keel and in smooth water provides for ports and scuttles being safely left open.

On board troopships the crew of the vessel are housed in a large top-gallant forecastle; the ship's officers live under the poop, and the passengers are berthed between decks. As regards air-space, the crew and passengers are in much the same condition as the crew of a man-of-war.

In the mercantile marine the crew are berthed mainly in deck-houses, while in passenger ships the conditions are much the same as obtain in troopers. The Board of Trade Regulations require a minimum air-space of 72 cubic feet per man, and a deck-space of 12 square feet; limits, needless to say, which are to be regarded rather as a basis for the enforcement of legal penalties than as a sanitary guide. In this connection it is to be noted that a liberal air-space is more important in men-of-war and vessels similarly circumstanced, in which the bulk of the crew live on board continuously for three or four years, than in the case of passenger steamers and troopships, in which the voyages are rapid, and the passengers are rarely for more than a few weeks on board.

On tropical service the large amount of water vapour given off by the skin into a heated atmosphere renders it desirable that a more ample air supply should be provided than in cold or temperate climates. Therefore we find that when vessels are specially constructed for permanent service in hot climates, accommodation is provided for the crew in large deck-houses, as in the case of vessels

serving in the Persian Gulf and in the rivers of tropical Africa.

Ventilation.—The unavoidably small amount of air-space available for the inhabitants of a ship renders the provision of adequate ventilation one of the most important considerations in naval hygiene. This subject is of relatively greater moment in hot climates than elsewhere, because the influence of excessive heat in confined spaces tends to aggravate the ill effects ordinarily produced by an excessive accumulation of respiratory products. Therefore one of the conditions for efficient ventilation in tropical regions is, that the means adopted must not have the effect of raising the temperature of the space to be ventilated. In cold climates it is desirable to warm the fresh air which is to be introduced, but in warm climates the desideratum is rather to lower the temperature of the purifying air. It is, as a rule, easier to raise the temperature of air than to lower it. Again, in cold climates the heat radiated off from the human body tends to produce an upward current of the vitiated air, and therefore favours the escape of such air through hatchways and skylights; while in hot climates, when the normal temperature of the body does not differ widely from that of the general atmosphere, the difference in specific gravity between the air of inhabited spaces and that of the outside air is so slight that the general condition is almost one of equilibrium.

As regards ventilation, our ships may conveniently be divided into two broad groups, viz.—(1) vessels with broadside ports, and (2) vessels without broadside ports. The first group includes (*a*) men-of-war carrying their principal armament in broadside ports; (*b*) troopships; and (*c*) passenger steamers. To the second group belong (*a*) turret and barbette ships; (*b*) vessels of low freeboard (yachts, torpedo vessels, etc.); and (*c*) cargo vessels.

The old grouping into "vessels of high freeboard" and "vessels of low freeboard" is no longer applicable; because vessels of the Monitor type, which the latter group was especially intended to embrace, have in recent times developed into huge turret and barbette ships with considerably higher freeboard than many vessels of the broadside group.

Before considering the means by which ventilation is provided for in the various kinds of ships above mentioned, it may be well to refer very briefly to ventilating agencies in general:—

Systems of ventilation are divisible into two broad groups, viz.—(1) "natural ventilation," and (2) "artificial ventilation." Natural ventilation may be again subdivided into (1) "natural ventilation without special contrivances," and (2) "natural ventilation by special

contrivances." The heat of the sun causing expansion of a certain stratum of air, and thus producing an upward current by the different specific gravities of contiguous bodies of air, is a natural ventilating agent. Similarly, the upward current produced by the heat radiated off from the human body into a cold atmosphere is an example of natural ventilation. But, on the other hand, the up-draught produced by a fire in a chimney grate; the draught produced in a locomotive engine when steam is allowed to escape from within the funnel with a view to urging on the fires; the draught produced by a rotary bellows; and the draught caused by an air-pump, are examples of artificial ventilation. Natural ventilation is, in general, effected in either of three ways, viz.—(1) by diffusion; (2) by perflation; (3) by inequalities of temperature.

The principal means employed for effecting *natural ventilation* on board ship may be enumerated as follows:—Ports and scuttles, hatchways and skylights, windsails and trysails, tubular ventilators of various forms, funnel casing (fires not alight), and hollow iron masts.

Ports and Scuttles.—These apertures ventilate by diffusion and by perflation. They act most efficiently when the ship is broadside on to the wind. When a ship is lying at anchor, head to wind, the scuttles on the broadside may be made to act as inlets or outlets by being fitted with half cylinders or scoops of sheet iron, which should project outwards for about a foot from the ship's side, and should be made to face forwards or backwards according as they are intended to introduce or extract air. Whenever it is found desirable to use these contrivances (as may often happen in tropical harbours), it is a good plan to make the scuttles on one side of the ship act as inlets, while those on the other side act as outlets, thus establishing a complete lateral movement of air through the vessel.

Ships lying in tropical harbours, more especially in trade-wind regions, may sometimes be ventilated by having a kedge anchor laid out from the stern, by means of which the stern of the ship can be hauled up sufficiently far to allow the wind to enter the ports and scuttles.

The same object may be attained in rivers or tideways by putting the helm "hard over" one way or the other, so that the pressure of the water may have the effect of moving the axis of the ship out of the line of the current. This method is especially applicable to ships anchored in tropical rivers, when it sometimes happens that although there is no general movement of the atmosphere, yet the friction of the river current produces a wind motion of the low-lying stratum of air in the same direction as the flow of the river.

In some vessels of the mercantile marine, the scuttle-glasses are hinged by their lower margins, and are so adjusted that under normal conditions they lean outwards a few degrees from the vertical position, so as to leave a somewhat crescentic opening for the passage of air. On being struck by a sea, the scuttle shuts inwards automatically against an annular rubber seating, the poising of the scuttle being so arranged that it closes instantly on the least pressure from without. The amount of ventilation thus afforded is necessarily small, but it is better than none at all, and these contrivances are therefore well suited to vessels which have to encounter much stormy weather, or which are subject to excessive rolling.

Hatchways and Skylights.—These deck openings permit of interchanges of air by diffusion, and also furnish a means for the exit of foul heated air from the interior of the vessel. In ships of low freeboard they are obviously not available in stormy weather.

Windsails and Trysails.—These act as "supply" ventilators, and cannot be used to "exhaust" air. Windsails are collapsible canvas tubes, usually of about 2 feet in diameter. The upper extremity is fashioned in various ways, the form most usually adopted being a throat opening, sometimes known as the "shark's mouth" aperture, which, of course, requires to be trimmed to the wind by suitable "guys." Sometimes the head portion is provided with four mouths, so that the windsail may act automatically, no matter in what direction the wind may be blowing; but this arrangement has the great drawback of materially diminishing the amount of air which can be admitted.

Trysails and other lower sails act as supply ventilators by deflecting the wind downwards towards the deck openings. The fore and main "courses" (lower square sails) form very efficient ventilators by driving air down fore and main hatchways respectively. Vessels at anchor may be ventilated on the same principle by having a triangular sail (apex upwards) set up over the fore part of the deck, and trimmed so as to face forwards or otherwise according to the wind, thus deflecting air down to the hatchways.

Tubular Ventilators.—The class of ventilators hereby referred to are made either of wood or of metal, and may be adapted either for supply or for exhaust, or to perform both functions simultaneously.

Wooden ventilators are largely used in the Indian troopships. These are square in horizontal section, and are divided by diagonal partitions into four compartments. The head of the ventilator rises well above the level of the bulwarks, and has four large throat openings controlled by doors, so that while the opening facing the

wind is receiving and sending down fresh air, the opening turned away from the wind is exhausting foul air from below. Each of these compound ventilators communicates, as a rule, with only one compartment of the ship, and beneath its lower termination is suspended a large metal tray for the reception of rain or spray which may find its way down from above. These ventilators are unsightly objects, but they act most efficiently.

A great variety of metal ventilators are used on board ship, some having fixed heads, others movable heads, and others again having revolving heads which trim themselves automatically.

Those with *fixed* heads are fitted so as to act either as uptakes or downcasts; under either circumstances the volume of air which they deliver is comparatively small, but they have the advantage of requiring no care.

One of the best types with *movable* head is the cowl-headed ventilator, which is the pattern used in the Royal Navy. According as it is turned to or from the wind, it acts either as a downcast or as an uptake. It delivers a large volume of air in proportion to its size, and is a simple and most reliable form of ventilator.

Those with *automatically revolving* heads are usually constructed so as to act as uptakes. They have the disadvantage of being cumbrous and easily disarranged, while as regards efficiency they are no better than a plain straight tube fitted with a hood to keep out the rain.

The Funnel Casing.—This is a large plate-iron structure rising from the top of the boiler and enclosing funnels, steam-pipes, etc. It is usually fitted with louvred openings affording communication with the decks through which it passes, so that even when the stokehold fires are not alight the heated air between decks finds a ready exit by way of the funnel casing.

Hollow Iron Masts.—The hollow iron lower masts, now so largely used in men-of-war, merchant steamers, and sailing ships, are available as exhaust ventilators if fitted with suitable openings above and below. The natural upward current of heated air from below is here reinforced by the aspirating action of the wind blowing horizontally over the cap of the mast. It is a common mistake to have so many openings in the mast between decks that the exhaust of foul air takes place only through the opening nearest to the upper deck. The mast should be fitted so as to extract only from the lowest inhabited space through which it passes, and the inlet opening should be placed near to the roof of that compartment.

In vessels such as men-of-war, where the crew sleep in hammocks, and therefore usually in close proximity to the deck overhead, it is

advisable to have a large number of perforations in the deck, so as to provide for the escape of respiratory products. These openings should be connected with siphon-shaped tubes, having the short arm of the siphon facing the upper deck and terminating in a "rose" head. By this arrangement the admission of wash-deck water from above is prevented, while the heated air has a means of escape.

Contrivances for effecting *artificial ventilation* will now be referred to under the following heads:—Funnels and funnel casing (fires alight), rotary fans, steam-jet ventilators, Perkin's automatic ventilator, punkahs.

Funnels and Funnel Casing.—In vessels with open stokeholds and without watertight bulkheads, the draught through the funnel produced by the engine fires acts as an exhaust ventilator on the general interior of the ship, while in vessels with closed stokeholds its ventilating action only applies to the stokers. Similarly, the heat radiated off from funnels, steam pipes, and boilers, produces a powerful up-draught in the funnel casing, which may then be made to act as a very efficient exhaust ventilator on the spaces between decks. Indeed, in all vessels without broadside ports the funnel casing may be regarded as the main exhaust ventilator of the ship, except in those cases where the stokehold is worked under forced draught, and the funnel casing is made to admit fresh air to the supply fans.

Rotary Fans.—Centrifugal fans, driven by hand power, have for many years been used in vessels of the Royal Navy for the occasional supply of fresh air to magazines, shell-rooms, and double-bottom compartments. In turret and barbette ships steam-driven fans acting on the same principle are used for general ventilation, especially as regards those parts of the ship which lie below the steel protective deck. In some vessels of this class separate fans are fitted for "exhaust" as well as for "supply," thereby effecting a complete circulation of air; but in most of our men-of-war, "supply" fans only are fitted, the funnel casing being then relied on as the artificial means of "exhaust." In tropical climates the heat radiated off from the fan engine (usually a direct acting engine of the Brotherhood type) becomes an objection to its use. This difficulty may be got over by using compressed air as the medium for conveying the power to the fan engine, or, as in the case of the s.s. *Scot* of the Union Line, by using the electric current generated by a dynamo to drive electromotors geared to the axles of the rotary fans. In the vessel just mentioned, a large number of small fans are used instead of a few large ones. Quite recently, one of our battleships has been fitted with an electrically-driven Blackman fan,

in which the fan itself acts as the armature of the motor while the field-magnets are placed in the annular frame.

The linear velocity of air passing through the tangential opening of a properly constructed centrifugal fan may be roughly estimated at three-fourths the velocity of the periphery of the fan. The actual delivery in cubic feet will, of course, depend mainly upon the sectional area of the aperture.

Steam-Jet Ventilator.—This class of ventilator, which is largely used in the Indian troopers, is suitable only for exhaust work. It consists essentially of a vertical metal tube, into which is introduced a steam pipe from the main boilers. This steam pipe terminates in a ring-jet from which steam is discharged through a number of small apertures placed some yards below the upper extremity of the ventilator. The upward current of air is produced in two ways; firstly, by the friction exercised upon the air by the numerous jets of steam; and, secondly, by the air expansion caused by the heat radiated off from the steam pipe. The disadvantages of this ventilator are—(1) the noise made by the escaping steam; (2) the condensation of steam in the interior of the tube causing rapid oxidation of the metal; and (3) the heat evolved. The last-mentioned feature is especially objectionable in hot climates. It is important that the steam should be liberated at a rather low pressure.

It has been proposed that compressed air should be used instead of steam for producing this "induced draught" in hot climates, as the expansion of the compressed air would rather tend to produce a lowering of temperature.

Perkin's Automatic Ventilator.—This contrivance is used in the Indian troopers, where it is fitted so as to extract foul air from the lower parts of the vessel. It may, however, be fitted so as to act either for "supply" or for "exhaust." It consists essentially of two metal cylindrical vessels, placed one on either side of the ship, connected at their bases by a horizontal tube, and about half filled with water. The rolling of the ship causes the water to gravitate from one vessel to the other, and by the action of suitable inlet and outlet valves fitted to each cylinder, air is displaced from one and drawn into the other, and *vice versâ*. The inlet apertures are connected by tubes with the spaces to be ventilated, while the outlets are made to discharge to the upper deck. The drawbacks to this ventilator are—(1) that under the most favourable circumstances the volume of air set in motion is comparatively small; and (2) that when the vessel is on an even keel the contrivance is absolutely useless.

Punkahs.—These arrangements constitute materially useful ventilators, inasmuch as they tend to produce an equal distribution of the components of the otherwise stagnant air of apartments. Thus it is that punkahs oscillating over the mess-table in a ship's saloon will cause the air immediately surrounding the sitters, and therefore heavily charged with respiratory products, to mingle uniformly with the air of the entire apartment, and will thus reduce the proportion of impurities in the particular area where these impurities (aqueous vapour, carbonic acid, and organic matter) arise.

Punkahs are extensively used in the Indian troopers, and in passenger ships plying in tropical seas. In the former they are commonly driven by cranks connected with shafting from the main engines.

Finally, it is to be noted that in all systems of ventilation in which tubes are used for extraction or supply, the final output or delivery of air, considered independently of the source of motion, will be influenced by certain conditions which have reference to the nature of the tubes. These conditions are—(1) the length of the tube; (2) its shape; (3) the nature of its internal surface; (4) the number of turns; (5) the number of branches. Referring to these *seriatim*—(1) the delivery of air varies inversely with the length of the tube; (2) a tube of circular section is the most efficient, the skin friction being less than in tubes of other forms; (3) a smooth internal surface offers less resistance to the passage of air than a rough one; (4) each right-angled bend, when the air pressure is low, diminishes the current by about one-half, but a turn of large radius offers less resistance than an angular turn; (5) subdivision of tubes causes a considerable loss unless the sum total of the sectional areas of the branches be considerably greater than the sectional area of the main tube.

Dietary.—In tropical regions the average temperature of the air is frequently not much below the normal body temperature. Therefore the maintenance of the body temperature does not call for the ingestion of so large an amount of food, especially fatty foods, as in cold climates. On the other hand, unless the relative humidity be very great, hot weather largely increases the discharge of water vapour from the skin, thereby demanding a large consumption of fluid.

The diet naturally adapted for the inhabitants of warm climates is for the most part of a vegetable nature. There is less desire for animal foods than in cold climates, and also a less capacity for digesting animal foods, especially when of a fatty nature. The inhabitants of warm climates can also perform satisfactorily a large

amount of severe bodily labour on a purely vegetable dietary. But the same rule does not hold good for the British sailor when removed to a warm climate and called upon to undertake laborious duties. Therefore experience has shown that in devising a scheme of naval dietary suitable for warm climates, it is not wise to make a radical change from the diet scheme of temperate climates, but rather to introduce modifications in matters of detail. Much has been said as to the advisability of having at least two separate schemes of dietary in the Royal Navy and mercantile marine, one adapted to temperate climates, the other to tropical climates; but the fact appears to have been overlooked that in neither of these services are vessels, as a rule (excepting coasters and harbour ships, whose crews are to a great extent under the same dietetic conditions as landsmen), employed continuously in one climate or the other. To provide distinct systems of dietary for large bodies of men living on shipboard, employed one week in a cold and perhaps the next week in a warm latitude, as happens in the case of many ocean-going vessels, and to have shifts of diet schemes effected like shifts of clothing, would involve such great commissariat difficulties as to be wellnigh impracticable.

Experience has proved that it is best to adopt as a basis a well constructed temperate region dietary, and to make alterations in details so as to adapt it to tropical conditions of life. For this purpose the diet scheme of the Royal Navy, under the use of which almost from boyhood the British sailor has developed a splendid physique and has maintained a high standard of health, may well be put forward as a standard. This scheme is so constructed as to supply animal food in excess of what is ordinarily required for the wants of the system, so that the men, being allowed a money equivalent for those portions of the ration which they intimate a desire to leave behind, are enabled to form a mess fund with which they purchase from the ship's canteen or from the shore various articles of food to supplement the portion of the ration actually used. In this manner provision is made for the gratification of individual tastes, a very important element in practical dietetics, while articles of doubtful digestibility, although perhaps of great nutritive value, are not forced upon the sailor.

It will be seen from the following table that on harbour service fresh meat and vegetables are, as a rule, issued daily; but that at sea, and occasionally at other times when fresh provisions are not procurable, for the fresh meat ration is substituted either salt meat 1 lb., or preserved meat $\frac{3}{4}$ lb. The rotation of meats is arranged in such a manner that salt pork is issued every second day, and is

followed alternately by salt beef or preserved meat, the latter two thus alternating with each other. It follows from this arrangement that salt pork is eaten twice as often as either salt beef or preserved meat. By the term preserved meat is to be understood boiled beef or mutton preserved by canning:—

ROYAL NAVY SEA DIETARY FOR SEVEN CONSECUTIVE DAYS, IN OUNCES.

		First Day.	Second Day.	Third Day.	Fourth Day.	Fifth Day.	Sixth Day.	Seventh Day.
		oz.	oz.	oz.	oz.	oz.	oz.	oz.
Equivalents.	Biscuit,	20	20	20	20	20	20	20
	Bread,	24	24	24	24	24	24	24
	Preserved Meat,	12	...	...	...	12	...	...
	,, Potatoes,	2	...	...	...	2	...	...
	Rice,	2	...	...	...	2	...	...
	Salt Pork,	...	16	...	16	...	16	...
	Peas,	...	5·2	...	5·2	...	5·2	...
	Celery Seed,	0·5 oz. to every 8 lbs. of Peas.						
	Salt Beef,	...	...	16	...	...	...	16
	Flour,	...	...	9	...	...	...	9
	Suet,	...	...	7·5	...	...	...	7·5
	Raisins,	...	...	1·5	...	...	...	1·5
Issued Daily.	Rum,	2·5	2·5	2·5	2·5	2·5	2·5	2·5
	Sugar,	2	2	2	2	2	2	2
	Cocoa,	2·2	2·2	2·2	2·2	2·2	2·2	2·2
	Tea,	0·25	0·25	0·25	0·25	0·25	0·25	0·25
Issued Weekly.	Oatmeal,	3 oz. weekly.						
	Mustard,	0·5 oz. weekly.						
	Pepper,	0·25 oz. weekly.						
	Vinegar,	5 oz. weekly.						
	Lime juice,	0·5 oz. daily after ten days at sea.						
	Sugar,	0·5 oz. daily after ten days at sea.						

Bread and Biscuit not issued simultaneously.
Rum not issued to any one below 20.

In the Harbour Dietary, fresh meat 16 oz. and fresh vegetables 8 oz. are issued daily, instead of salted or preserved meats and their accompaniments. Biscuit not issued in harbour.

The salt pork ration is accompanied by an issue of split peas; the salt beef is supplemented by an issue of flour, suet, and raisins; and the preserved meat carries with it, either an issue of flour, suet, and raisins, or an equivalent of preserved potatoes and rice.

It has often been suggested that a liberal issue of preserved vegetables would entirely obviate any necessity for the use of lime juice; but, on the other hand, it is found that lime juice is very much appreciated by the men (especially on tropical service), as it forms an agreeable adjunct to the midday meal, when the only other drinks available are either plain water or grog.

There is a general consensus of opinion that, in adapting a temperate climate dietary to the conditions of life on board ship in tropical regions—(1) animal food should be reduced; (2) fatty food should be reduced; (3) vegetable food should be increased; (4) the allowance of fluids should be increased; and (5) the use of alcoholics should be altogether omitted.

But when a large amount of muscular exertion has to be put forth, as in the case of blue-jackets performing arduous drill exercises, then experience has shown that the regular service ration of animal and fatty food cannot be widely departed from.

Surgeon-Major Duncan, speaking of the requirements of the soldier, puts the matter tersely thus: "In the *resting stages* of a tropical campaign, let animal food be in part substituted by vegetable. Let the fats be diminished, the carbonaceous elements being furnished rather by the starches. In the *marching and fighting stages*, on the contrary, let animal food assume its wonted proportions."

With regard to the use of alcoholics as an item in tropical naval dietaries, it may safely be said that they are undesirable and probably hurtful, either in the form of spirituous or fermented liquors; although light red wines are useful, inasmuch as they reinforce the vegetable portion of the dietary in an agreeable form, and are also valuable as antiscorbutics. The rum ration, although still forming part of the naval diet scheme, is not issued to men below the age of 20 years; while its use by the others is very wisely discouraged by allowing the men to take up in its stead somewhat more than its equivalent value in tea and cocoa. Large numbers of blue-jackets now use these latter articles in preference to rum.

Water Supply.—It is clearly essential that men living on board ship in tropical regions should have an ample supply of pure drinking water. In the Royal Navy the supply of water for drinking purposes is practically unlimited, whether the ship be at sea or in harbour; while as regards ablutionary and other purposes there is no definite restriction on the allowance of fresh water. It is, however, found that the consumption per head for all purposes, while varying in different ships, does not usually exceed 5 gallons per man per day. This small consumption (small as compared with shore-going communities) is owing to salt water being readily available for sanitary flushing and for deck washing, and also in some degree to the circumstance that the water supply is largely procured by distillation, whereby, as regards washing purposes, the water, owing to its perfect lathering, goes further than the same amount of shore water which usually contains lime salts.

The unfavourable conditions under which water is stored on board ship, especially as regards restricted ventilation, are accentuated in hot climates by the influence of heat in favouring the multiplication of microbes. It is therefore all the more important to take special precautions in tropical regions to guard against the admission to a ship's tanks of water which is not of a high degree of purity.

The Admiralty Instructions direct that all shore waters, before being accepted for consumption on shipboard, are to be examined analytically by the medical officer; and for this purpose a set of reagents for qualitative analysis is supplied. By this means a water which is decidedly bad may readily be excluded. The tests which may thus be made refer to the presence of free ammonia, chlorides, sulphates, nitrites, nitrates, and organic matter. The albuminoid ammonia process is not usually attempted owing to the difficulty of carrying out such an analysis on board ship. However, the detection of organic matter by means of the gold chloride reaction supplies information of the greatest value, provided the test be carefully conducted, checked by blank experiments with distilled water and by comparison with samples of distilled water adulterated with known quantities of organic matter.

It is important that shore waters should be tested at two different stages—(1) before being drawn from the shore; (2) after admission to the tanks.

Impurities may reach the water during its transit from the shore in different ways, according to the means employed. If brought off in casks, the water may be contaminated by previous foulness of the casks. If conveyed "in bulk" in open boats, sea water may reach it through leakage or by water washing over the boat's gunwale. Men-of-war are now supplied with collapsable canvas bags, which are so shaped that when distended with water they adapt themselves to the inner surface of the boat and protect the contents from sea-water contamination. The most satisfactory method of watering ship is by means of iron tank-vessels specially constructed for the purpose, and nowadays available in most seaports of any consequence.

In whatever way water may be conveyed to the ship's side, it is again liable to get contaminated by being pumped through greasy leather hose, or by being received inadvertently into tanks already containing brackish water, or into tanks coated with whitewash made up with "size." Moreover, rats or cats (or their excreta) may fall into the tanks through ventilating apertures, and thus cause pollution of the water.

When the shore water is open to suspicion, men-of-war now

invariably use distilled water, obtained from sea water by means of Normandy's condenser. The distinguishing feature of this apparatus is, that the distilled water is aerated during the process of condensation by the air which the heat of the condensing steam expels from the circulating sea water of the condenser. It therefore follows that if distillation be carried on while the ship is anchored close to the outlet of a sewer, or while the ship is lying in a close harbour whose waters are highly polluted with sewage material, the aeration of the distillate will include the introduction of sewage gases, such as ammonia and hydrogen sulphide. Under the same conditions there is also a danger of the distillate being directly contaminated with sewage matter from the circulating water, should the condenser tubes happen to be leaky.

Again, if the steam for distillation be taken from the main boilers, the distillate may be rendered brackish by the "priming" of a boiler; and in those ships where the water derived from the working steam of the engines is collected in "surface condensers" and so returned to the main boilers, the distillate may be contaminated with volatile oily matters derived from lubricating materials used in valves and cylinders, and also with gases evolved by anti-incrustation compounds used in the main boilers.

In men-of-war of recent construction, the Normandy condensers take their steam from "evaporators," which are quite distinct from the main boilers.

Filtration.—Water properly distilled and aerated, as by an efficient Normandy condenser, does not need filtration, unless in the event of the steam having been taken from the main boilers, when filtration becomes necessary for the removal of oily matter. The original pattern of Normandy condenser is fitted with a very simple filtering apparatus which intercepts such oily impurities. This filter is arranged for downward and upward filtration, and is intended to be charged with animal charcoal or any similar filtering medium.

Filtration is also necessary in the case of shore waters containing a large amount of suspended matter, and also in the very exceptional cases when one is compelled to take on board ship waters of doubtful potability.

A filter suitable for the wants of the seaman should have the following characters:—(1) It should be simple in construction, so that it may be readily taken asunder, cleaned, and readjusted; (2) it should be strong and compact; (3) it should admit of rapid filtration; (4) it should exert a large amount of chemical and mechanical purifying action; (5) the filtering medium should be of

one kind, and should be of stable composition as regards storage; (6) the filtered water should not remain in contact with the filtering medium; (7) the filtering medium should not require frequent changing; and (8) neither filter vessel nor medium should add anything injurious to the water. A filter which aims at meeting all these requirements can obviously only be a compromise between efficiency and simplicity.

Crease's filter is the pattern at present mainly used in the Royal Navy, all the large men-of-war having filters of 200 or 400 gallons gross capacity. The medium used is "carbalite,"—a black or dark slate-coloured granular material, manufactured by a patent process which is understood to consist in roasting and impregnating with carbon a natural calcareous earth. In regard to both chemical and mechanical action, "carbalite" enjoys the reputation of being one of the best filtering media.

Storage of Water.—The storage tanks in the Royal Navy are of two kinds, movable and fixed. Both are constructed of iron plates, joined by angle irons and rivets. The movable tanks are somewhat cubical in shape; they have a capacity of about 600 gallons; they are fitted with manhole openings on top for the admission of suction and delivery pipes, as well as for ventilation; and they have a tap at bottom in order to provide for thorough drainage in connection with cleansing operations.

If the internal surface be left bare and the tanks be not constantly full, a copious deposit of iron oxide takes place, rendering the water somewhat turbid. To prevent this state of things various coating compounds have been suggested, but nothing altogether satisfactory has as yet been introduced. Calcareous and siliceous coatings (the so-called enamels), which do not expand and contract concurrently with the iron plate under the influence of heat and cold, readily separate from this cause as well as from the vibration to which the tanks are subjected by the motion of the vessel. The great desideratum in this direction is a smooth and durable enamel of harmless composition, which can be made to form a uniform coating over the whole interior of the tank, and which shall have the same coefficient of expansion as iron. On the other hand, flexible tenacious coatings of the asphaltic class are liable to contaminate the distilled water on account of the latter being delivered from the condensers at a temperature of 100° F. or more. On the whole, it seems best, pending the discovery of some satisfactory coating, to leave the internal iron surface quite bare, and when the amount of iron oxide in the water becomes excessive, to resort to mechanical filtration for its removal.

In men-of-war of recent construction, certain cellular spaces, between the inner and outer sheathing of the vessel, are set apart for the storage of water. These are the *fixed* storage tanks. They are usually more difficult of access for cleansing purposes than the old-fashioned movable tanks, and they do not seem to have much to recommend them beyond the economy of space which is effected by their use.

It has been found that both shore water and distilled water when stored on board ship have a tendency to deteriorate in regard to the multiplication of microbes, although contact with the iron surface of the tanks exercises a purifying action in other respects. It is therefore advisable that water should not be stored for long periods, the adoption of this rule being more important in tropical regions than elsewhere.

Clothing and Personal Cleanliness.—The clothing of the sailor on tropical service, as compared with that of the landsman, is mainly governed by two conditions. These are—(1) limitation of space for storage of clothing; and (2) the duties aloft, which necessitate a somewhat closely-fitting attire.

The functions to be performed by the sailor's clothing in hot climates are—(1) reflection and non-absorption of solar radiant heat as regards all parts of the body, more especially the head and spine; (2) permeability to vapours exhaled from the skin; (3) a large capacity for absorbing perspiration, attended by a slow rate of evaporation from the outer surface of the clothing; and (4) adequate protection of the skin from abrasions.

These conditions are fairly fulfilled by the flannel vest and drawers covered by duck jumper and trousers, and by the straw hat when on deck, or by the cloth cap covered by duck or drill when aloft.

The chief difficulty in providing a suitable tropical attire for the sailor is in regard to the head covering. The pith or cork helmet of the landsman is quite inadmissible on account of the peculiar nature of the sailor's duties, while the straw hat, in order that it may be sufficiently durable to resist blows from ropes and flapping sails, is of necessity rather unpleasantly hard and heavy. The white-covered cloth cap must also be stoutly made, so as to afford sufficient protection to the head against injuries; and although the white duck or drill covering is an efficient reflector of the solar heat, yet it has the disadvantage of checking the radiation of body heat, and of impeding the evaporation of moisture derived from the scalp. The cap would be more efficient in these respects if it were practicable to have ventilating apertures made in both the cloth and the white cover.

The duties of the sailor not admitting of the use of braces for his nether clothing, the short flannel vest frequently gets hitched above the waistband of the trousers, thus leaving the loins and abdomen unprotected. Such an event is very apt to occur on tropical service when the men of the night-watches lie down on the bare deck and perhaps fall asleep in this imperfectly covered state. Rheumatism and diarrhœa have often been attributed to this cause. This deficiency in the blue-jacket's attire is in a great measure covered by the use of cholera belts, the habit of wearing which is now becoming general.

The working clothes of the day should always be changed before the cool of the evening. The custom of the naval service is that at five o'clock in the evening, the regular work of the day being then over, the men change their clothing, putting on serge jumpers and trousers instead of the white clothing.

Washing.—Owing to the large amount of cutaneous excretion in tropical climates, frequent ablution becomes more imperative than in cold or temperate regions. For this purpose there is now a liberal supply of fresh water on board all vessels of the Royal Navy. Those who need it most are the stokers, who now form a large proportion of the crews of men-of-war, and for whom a special wash-place is provided, with unlimited supplies of fresh water. This wash-place is usually partially partitioned off from the general deck-space; is provided with a flooring of sheet lead or concrete; and is so arranged that the stokers can have access to it at any reasonable time. The seamen perform their regular personal ablutions every morning, and for this purpose each mess is supplied with two tubs of fresh water. In some ships there is also a specially fitted wash-place, where the seamen can wash themselves all over after evening quarters. Blue-jackets readily avail themselves of these facilities for personal washing, and, indeed, are almost invariably most cleanly in their persons and attire.

On two days in the week arrangements are made after evening quarters for the washing of white clothing and underclothing.

With regard to the bedding, the Admiralty Regulations require that it shall be triced up in the rigging once a week for thorough airing, according as the weather permits; while the blankets are to be washed in soap and warm water twice a year.

The quantity of fresh water thus supplied for personal and clothes washing amounts to about 2½ gallons per man per diem.

Ship Cleanliness.—It may be laid down as an axiom with regard to ship cleanliness that "a ship cannot be considered clean unless she is dry." While, therefore, water must be used in some form for

the removal of filth from the decks, its use should be so regulated that the decks may be dry for the greater part of the day. Routine is, as a rule, essential for the maintenance of discipline on board ship, but in respect to deck washing, routine should be made to give way to meteorological conditions. Deck washing should, therefore, not be practised on a rainy day, or when the humidity of the air is close to the saturation point. A rough and ready rule as regards the latter condition is, that there should be no flooding of the decks when the difference between the wet and dry bulb thermometers is less than 3° F.

It is above all things essential that the lower deck, where the men are mainly berthed, should be kept as dry as possible. The three following methods have been tried with a view to attain this end :—(1) Dry holy-stoning ; (2) washing with hot fresh water ; (3) having the deck varnished or painted, and cleansing by means of swabs soaked in warm water.

" Dry holy-stoning " consists in rubbing the deck with sand, which rubs out and collects the filth, with which it is afterwards swept up. The objections to this procedure are—firstly, that the sand is apt to find its way among the engines, to their great injury ; and, secondly, that the decks get rapidly worn away.

Washing with hot fresh water effects the removal of greasy filth more readily than sea-water washing. It, moreover, insures that the decks will not be excessively wetted, owing to the scarcity of fresh water as compared with salt water ; while it also provides for more rapid evaporation and consequent drying of the deck.

The best method, so far as health is concerned, is to have the decks varnished or painted so as to be impermeable to moisture. The necessary cleansing is then readily effected by rubbing with swabs or mops soaked in fresh water, while the subsequent drying may be accelerated by the use of dry deck cloths spread smoothly over the damp deck for some hours. This method has been adopted in some men-of-war fitted with the steel protective deck upon which rests a thin layer of wood planking ; the main object in this case being to prevent the corrosion of the metal by the soakage of washing water through the porous planking.

Attempts have been made to render the deck impervious to moisture by having it covered with linoleum, or some similar waterproof material, which is permanently secured to the deck by cement and tacks ; but this method has proved unsatisfactory owing to the large number of apertures which have to be left for getting at valves, sluices, flooding cocks, etc., and the impossibility of making the linoleum quite watertight at these places.

On the other hand, if cleanliness is to be gauged by the production of a white surface which shall be pleasing to the eye (which is not the view of the sanitarian), no doubt there is nothing so effectual as the old-fashioned scrubbing.

If the cleansing method adopted be one in which common soap is used, it is important that no soapy residue be left on the deck, as the organic materials which enter into the composition of common soap are apt to decompose and thereby constitute filth of an objectionable nature. This consideration is obviously of especial importance in hot climates.

With regard to the upper decks, *i.e.* the uncovered decks, it is the custom in the Royal Navy to wash them down every morning with salt water, and once a week to scrub them with salt water and sand.

For all decks below, fresh water is used. The mess-decks are usually washed down every morning directly after the men have completed their personal ablutions.

Latrines and Bilges.—*Latrines.*—Latrines both for officers and men should, as far as practicable, be placed outside the main structure of the ship. They should, at all events, be on the upper deck, and should be isolated from the living parts of the ship by means of lead flooring and metal combing, so that there may be no danger either of the excreta or of the water used for washing down the latrines gaining access to the wood planking.

Nowhere does the water-carriage system for the removal of sewage act so efficiently as on board ship. Water for flushing purposes is here always present in unlimited quantities, and only needs the aid of a hand-pump and a cistern to bring it into operation.

In the Royal Navy the style of W.-C. supplied for officers is the "valve closet"; water-seal closets being inadmissible on account of the pressure of air in the soil-pipe caused by the plunging of the ship in a seaway, and the consequent rise of sea water through the lower extremity of the soil-pipe. It is advisable that a small relief-pipe should be led from the soil-pipe below the trap of the closet outwards through the ship's side and for a few feet upwards, so as to protect the soil-pipe against bursting from excessive air pressure during regurgitation of sea water.

Officers' W.-C.'s should, when practicable, be placed side by side in an almost horizontal line, so that the pans with their separate valves may connect with a slighly inclined common soil-pipe. The latter should be thoroughly flushed daily (or more frequently) by attaching a flexible hose from the "fire main" to a screw-capped

opening at the upper end of the horizontal soil-pipe. This, of course, in addition to the regular flushing of each pan from the cistern overhead.

For the men's latrines there is nothing better than the trough closet, which consists of a long capacious trough with slightly-inclined bottom, surmounted by a row of seats, and which is flushed periodically by lifting a large plug-tap at the most depending part of the trough.

All soil-pipes should reach externally to just below the water-line, and should be fitted with metal or leather non-return valves, so as to limit the tendency to sea-water reflux.

In the Royal Navy W.-C.'s are provided for the officers and petty officers; while the accommodation in the trough latrines is calculated at the rate of three seats and upwards for every one hundred men of the rank and file.

Bilges.—The management of the bilges requires the utmost care in hot climates. There are important differences between wooden and iron men-of-war in regard to—(1) the structure of the bilge; (2) the gases which it evolves; and (3) the method of cleansing it. But in iron merchant ships constructed with a single shell, the position and shape of the bilge somewhat resembles that of a wooden ship.

In the wooden ship the bilges take the shape of longitudinal gutters extending from end to end of the ship, and lying at either side of the keelson. Sea water from without leaking through any part of the hull readily finds its way down to the bilges through the spaces between the timbers or ribs. Waste water from the decks may also pass through badly-caulked seams in the "water-ways," and so reach the bilges. Again, waste oil from the engines collects in the bilges; while particles of coal, pieces of cotton waste, the bodies of dead rats, and the excreta of cats, may also enter into the composition of bilge material. In addition to these, the bilges may receive various fluids and dissolved solids (such as sugar, salt, etc.), according to the nature of the cargo.

Material of this nature rapidly undergoes decomposition under the influence of heat, moisture, and vegetable micro-organisms, for whose existence and multiplication the sodden and perhaps decayed wood of the bilge itself affords a suitable organic nidus. The gases which may thus be evolved are hydrogen and ammonium sulphides, ammonia, carbonic acid, and hydrogen phosphide. The most important of these gases, as regards the frequency of its occurrence, is hydrogen sulphide. Its presence, as well as that of ammonium sulphide, is commonly attributed to a decomposition of the sulphates

in sea-water, and to a dissociation of the nitrogenous compounds in woody and oily matters, effected by the agency of micro-organisms. The evolution of hydrogen sulphide may be readily detected by the fetid odour of the gas, and by its blackening a strip of bibulous paper soaked in solution of lead acetate; while the presence of ammonium sulphide may be similarly detected by its reaction with sodium nitro-prusside.

In iron men-of-war the bilge material collects upon the more or less flat surface of the inner shell of the vessel, that is, upon the upper surface of the double-bottom compartments. From here it is removed by small openings fitted with non-return valves, through which it flows into a large pipe (known as the "main-drain"), which passes in a fore-and-aft direction between the two skins of the vessel, and is connected with suctions from the pumps. Should such a vessel become leaky, the sea water is arrested in the double-bottom compartments, and does not therefore gain access to the bilge.

From this it follows that sea-water is not so material a constituent of the bilge fluid in iron men-of-war as in wooden vessels, and that, in the former, very little wood, if any, is present to form a nidus for micro-organisms. Moreover, the ironwork of the inner skin exercises a certain amount of antiseptic and purifying action upon the bilge fluid. On the other hand, iron pyrites may be present as an impurity in the iron plates, and by its decomposition hydrogen sulphide may be evolved. But, on the whole, it may be laid down as a general rule that the bilges of iron ships give less trouble in regard to noxious effluvia than those of wooden vessels.

As regards the treatment of the bilges in both classes of vessels, the first consideration is to keep them as dry as possible. It was formerly the practice in some ships to run clean sea-water into the bilges directly after cleansing. This was known as the "wet method." The "dry method" is preferable.

With respect to disinfectants, both carbolic acid and Condy's fluid are useful. An objection to carbolic acid is that when introduced into the bilges, its odour, which to many people is highly disagreeable, pervades the entire ship for a considerable time.

Finally, it is to be remembered that during rough weather the rolling of the ship may cause the bilge material to gravitate to a considerable distance from the middle line, and that a flotsam and jetsam deposit of decomposable matter may thus take place far away from the bilge proper, and beyond the reach of ordinary cleansing operations.

Exercise.—The general rule that in hot climates laborious work should not be performed during the middle hours of the day,

when the sun is nearly vertical in the heavens, holds good on board ship as well as on shore. Therefore, as far as possible, all heavy work, especially in the open air, should be avoided between the hours of 10 A.M. and 3 P.M. It is not always practicable to adhere to this rule on board ships at sea, where the trimming of the sails and so forth must be performed as occasion requires; but in harbour, drill exercises may very well be relegated to the early forenoon and late afternoon.

It fortunately happens that the special maladies to be apprehended from undue exposure and over-exertion in hot climates, viz. sunstroke and heat-stroke, are not nearly so prevalent on the high seas as in harbour or in such confined seas as the Persian Gulf and Red Sea. Indeed, in the Royal Navy, where the bulk of our vessels are employed in hot climates, and drill exercises on deck and aloft are performed almost to the same extent as on the home station, these maladies are rare, excepting in a few specially ill-favoured localities.

Isolation of Infectious Diseases.—This subject is here considered only with reference to ships on the high seas. The measures to be adopted must vary very widely according to—(1) the proportion of the crew affected at the outset; (2) the structure of the vessel; (3) the state of the weather as regards wind and rain; and (4) the direction of the ship's course with respect to the prevailing wind.

If the outbreak be not recognised until a large proportion of the crew has been attacked, then it is of the first importance to take steps for the isolation of the healthy members. The latter may be grouped together under a poop or forecastle which had previously been disinfected, or they may be housed in canvas structures erected on the upper deck. The general ship accommodation will then, for the time, be left to the sick.

Infectious diseases on board ship tend to spread rapidly in horizontal lines and from below upwards. Therefore, if the ship be not divided by transverse bulkheads, it is, as a rule, best to isolate the infected cases on the upper deck. Here the infected area should be surrounded by a canvas screen, the latter being so arranged as to include one or more scuppers for the removal of slop water; while a "shoot" for the disposal of excreta should be within easy reach. Under such circumstances it is a good plan to have the canvas screen continually moistened with some disinfecting solution.

It has sometimes been found practicable to isolate the sick, if few in number, by berthing them in the boats hung at the davits, and housing them in with light awnings fitted to each boat.

If the ship be fitted with a poop and forecastle which can be set apart for the sick, such positions are most suitable for the isolation and treatment of infected cases.

In ships with watertight compartments, an entire compartment from bulkhead to bulkhead, extending vertically throughout the whole depth of the ship, may be devoted to the sick. This latter arrangement is one which may well be adopted in turret and barbette ships, when deck accommodation is usually altogether impracticable.

In wet and stormy weather, deck accommodation for the sick, other than in poop and forecastle, is obviously impracticable.

The position of the infectious area must also be regulated with due regard to the direction in which the wind is blowing over the ship, in order that the sick may be placed to leeward of the healthy members of the crew. It therefore follows that with a head wind, the poop is more suitable than the forecastle; while with a wind from aft, the opposite condition will obtain. If the wind be on the beam, and a canvas enclosure be used, the sick area should, of course, be placed on the lee side of the deck.

Disinfection.—Disinfectants may have to be used on board ship under four different conditions—(1) for the disinfection of the whole interior of empty vessels; (2) for the disinfection of portions of inhabited vessels; (3) for the disinfection of clothing and bedside appliances; and (4) for the disinfection (as well as deodorisation) of bilges and latrines.

1. For the disinfection of vessels which have been entirely vacated for the purpose, either sulphurous acid or chlorine should be used. The former is, as a rule, the more readily available of the two, but has the disadvantage of damaging some articles owing to its blackening the surface of those metals with which it forms sulphides. It may be easily and safely evolved by setting fire to brimstone or flower sulphur placed in a metal dish standing in a large vessel containing a couple of inches of water. The ignition of the sulphur may be facilitated by the addition of a little spirit. The sulphur should be used in the proportion of 1 lb. to every 1000 cubic feet of air-space, in order that the sulphurous acid evolved may be in the proportion of 1 per cent. to the volume of air contained in the ship.

Chlorine may most readily be evolved by adding hydrochloric acid to chloride of lime; the proportions being 22 lbs. of the former and 15 lbs. of the latter for every 1000 cubic feet of air to be disinfected. This is Koch's estimate, but in the case of ordinary disease-germs a much less amount is found to be sufficient.

Another convenient method of producing chlorine is by heating a mixture of common salt, manganese binoxide, and sulphuric acid.

The action of either gas may be rendered more effective by previously moistening the air of the ship by means of steam.

The process of disinfection should extend over six hours at the very least; and during that time all skylights, ports, scuttles, and other openings should be kept closed. Then, before the ship is again inhabited, every portion of it should be subjected to thorough ventilation so as to effect the removal of every trace of the disinfecting gases.

2. For the disinfection of enclosed portions of inhabited vessels—such as cabins, sick-berths, and such places—gaseous disinfectants should, as far as possible, be used. The difficulty which attends their use is that, owing to the very poisonous nature of the gases in question, provision has to be made against any escape of these gases into the inhabited portions of the ship. Great care must, therefore, be taken to close up effectually all apertures through which the gases might escape. It is also advisable to have the disinfection of such spaces carried out during the daytime, in order that the gases used may be dissipated by free ventilation before the crew turn in for the night.

This is one of the cases in which "euchlorine" may well be substituted for either chlorine or sulphurous acid, as it possesses the advantage of being less irritating to the lungs. "Euchlorine" may be produced by gently heating a mixture of potassium chlorate and hydrochloric acid.

As, under the circumstances above referred to, gaseous disinfection cannot usually be carried out in a sufficiently thorough manner, it is advisable to supplement this procedure by having the walls, ceiling, floor, and furniture sponged over with sublimate solution (1 per 1000), or with carbolic solution (5 per cent.).

3. One of the simplest and best methods for the disinfection of clothing and small bedside appliances is by soaking them for twenty-four hours in a 1 per 1000 solution of corrosive sublimate. It is to be noted that for this and similar purposes there are certain drawbacks attending the use of a plain solution of corrosive sublimate in water. These are—(1) that it is colourless, and may therefore be mistaken for a harmless liquid; (2) that it corrodes metals; and (3) that it forms an insoluble compound with albumen, and is therefore soon rendered inert on coming in contact with clothes saturated with blood or other albuminous material. These objections may be partly got over by using a solution made up as follows:—Corrosive sublimate, ½ oz.; methyl blue, 5 grains; hydro-

chloric acid, 1 oz.; and water, 3 gallons; and by steeping the clothes in a stoneware or enamelled vessel.

4. With respect to bilges (which occasionally require special treatment in hot weather), it is the practice in the Royal Navy to use for their purification a 2½ per cent. (1 in 40) solution of carbolic acid. A 2½ per cent. solution may be made by dissolving the contents of a 1 lb. bottle of carbolic acid in 5 gallons of water. Condy's fluid is sometimes employed for the same purpose. It should not be used in conjunction with carbolic acid.

Latrines and W.-C.'s on board ship ought not, under ordinary circumstances, to require the use of any disinfectant or deodorant. But should there be cases of enteric fever or cholera on board, it is well to place some disinfecting agent in the latrine troughs and in the flushing tanks of the W.-C.'s. Under these circumstances it is advisable to use a disinfecting agent which mixes rapidly with water, so that it may be introduced in the crude state into the water lying in the troughs and tanks in such quantities as to make a solution sufficiently strong for disinfecting purposes. For this reason permanganate of potash is recommended, although not so powerful a disinfectant as either carbolic acid or corrosive sublimate. The crystals should be cast into the water in handfuls, according to the bulk of water in question, several times a day.

Tropical Ship Diseases.

The ailments and injuries which will here be referred to are those which may, perhaps, be regarded as more liable to occur among the crews of ships in hot climates than elsewhere. They are as follows:—Prickly heat, febricula, sunstroke and heat-stroke, malarial diseases, mosquito bites, boils and ulcers, fish poisoning, poisoned wounds caused by fish spines, moon-blindness.

Prickly Heat (*Lichen tropicus*).—In persons of a plethoric habit of body, this ailment is apt to occasion very serious inconvenience. It appears to be due, primarily, to first exposure to a permanently high atmospheric temperature; while among contributing causes may be mentioned—(1) want of adaptation of diet to climate; (2) the use of alcoholic stimulants; (3) bathing in sea-water, followed by exposure of the naked body to the sun; and (4) the irritation produced by coarse and uncleanly underclothing. That a *continuous* high temperature is a principal factor in the causation of this disease, is shown by its infrequent occurrence among stokers in cool climates, where the high temperature to which they are exposed by reason of their work at the fires is *intermittent*.

The measures to be adopted for the prevention of "prickly heat" are—(1) to reduce the amount of animal food; (2) to abstain altogether from alcoholics; (3) to wash down with fresh water immediately after sea-bathing; (4) to wear soft non-irritating underclothing; and (5) to practise thorough personal cleanliness.

Febricula.—A simple continued fever of about one week's duration is sometimes experienced, especially by the younger members of the crews of men-of-war, on first entering the tropics. It seems to indicate a temporary failure in the struggle made by the human body to adapt itself to a new environment, and may be also, to some extent, attributable to the sudden evolution of bilge gas which is liable to take place on a ship entering the tropics. The same form of fever has been found to appear concurrently with heat-stroke and sunstroke among the crews of ships stationed in the Red Sea and Persian Gulf during exceptionally hot weather. The evidence afforded by the naval records points very strongly to this being a true thermic fever.

Sunstroke and Heat-Stroke.—The crews of ships on the high seas are, as a rule, less liable to these affections than landsmen in the same region. Cases of heat-stroke, however, occur among stokers working in badly-ventilated stokeholds in hot climates. Such cases occur in the Red Sea and similar places. But in comparison to the large amount of tropical service performed by vessels of the Royal Navy, the occurrence of sunstroke and heat-stroke among blue-jackets may be regarded as rare; while the mortality from these causes is exceedingly small.

The measures which should be taken for the prevention of these ailments include—(1) adjustment of exercise on deck to the cooler hours of the day; (2) the use of an ample, light, and well-ventilated head covering; (3) double awnings over the decks; and (4) efficient ventilation of lower deck and stokeholds.

Malarial Diseases.—It is to be noted that malarial diseases may occur among seamen who have not *landed* in malarial localities. In such cases the disease is commonly traceable to air-borne miasmata, introduced by anchoring close to leeward of a malarial focus or in a land-locked harbour in a malarial region. Although it has been alleged that malarial diseases may be introduced on board ship by means of drinking water, the records of the Royal Navy do not give much support to this view. Indeed, the statistical returns for the last thirty years do not show a diminution in the proportion of cases of malarial fever, although very great improvements have been made during that period in regard to supplies of drinking water and the more extended use of distilled water. In this respect, viewed from a naval medical standpoint, the causations of dysentery and malarial fever appear to run on widely different lines. There is some reason to believe that ships anchored for long periods in malarial harbours may become independent centres of disease; but such a contingency is so rare that it may almost be left out of consideration.

When ships are obliged to anchor off a malarial shore, the preventive measures to be taken are—(1) to anchor, if possible, to windward of the malarial focus; (2) to anchor a long distance (at least a mile) from the shore; (3) to avoid land-locked harbours; (4) to anchor so that a belt of trees or a hill may be interposed between the ship and the infective area; (5) to avoid the use of shore water unless it has been boiled; and (6) not to allow the crew to remain on shore after sunset.

Mosquito Bites.—These are sometimes a source of great trouble to the younger members of the crew, giving rise to considerable œdematous inflammation of the eyelids, neck, and forearm. They may usually be prevented by the local application of a weak oily solution of carbolic acid.

Boils and Ulcers.—Eruptions of boils are apt to occur as a sequela

to prickly heat; and if the boils be irritated by exposure to sun and sea-water, they frequently degenerate into troublesome ulcers. The prevention of these affections may be inferred from what has been said with regard to "prickly heat."

Fish Poisoning.—Serious and even fatal illness arising from the ingestion of poisonous fish is not very uncommon among sailors in tropical seas. Unfortunately there is no hard and fast rule by which poisonous may be distinguished from edible fishes. An endeavour will therefore be made to indicate those fishes which are known to be poisonous, and also the families and genera which are credited with containing poisonous species.

Clupea thryssa ("yellow-billed sprat," "sardine doré") is a highly poisonous fish, belonging to the same genus as the common herring. It may be distinguished from other fishes of the same genus by its having the last ray of the dorsal fin prolonged into a filament.

Clupea venenosa is a tropical species of the same genus, occurring in West Indian waters, and decidedly poisonous.

Scarus (sp. ?) ("parrot fishes").—Certain species of this genus, inhabiting tropical waters in the vicinity of coral reefs, are found to be poisonous.

Tetrodon and Diodon ("toad fishes," "bladder fishes," "globe fishes," "sea porcupines").—Fishes of these genera are easily recognised by their general shape, their repulsive appearance, and their peculiar dentition. None of these should be used for food. There is one notoriously poisonous species, *Tetrodon punctatus* which is abundant at the Cape of Good Hope.

Silurus ("cat fishes").—Fishes of this genus met with in tropical seas are commonly regarded as poisonous; although there is a species inhabiting the Nile which is considered to be an excellent food-fish. The "cat fishes" are easily recognised by their general appearance.

Sphyræna.—To this genus belongs the well-known *Barracuda.* The *Sphyrænas* are for the most part large voracious fishes, inhabiting tropical and subtropical seas. Certain tropical species, especially in West Indian waters, have been found to possess poisonous properties, which are attributed to their having fed on smaller poisonous fish.

Balistes ("file fishes," "trigger fishes").—The fishes of this genus possess a tough, hard integument, and are distinguished by having three strong spinous dorsal rays, the first of which cannot be forced down unless the second has been previously depressed.

Ostracion ("trunk fishes," "coffer fishes").—This genus belongs to the same family as *Balistes,* viz. the *Sclerodermi.* The trunk fishes are easily recognised by their general appearance. Fishes of these two genera (*Balistes* and *Ostracion*) should be regarded as poisonous when caught in the vicinity of coral reefs. Their poisonous properties are supposed to be derived from feeding on medusæ and corals.

Lachnolæmus.—This is a genus occurring in the West Indies, and belonging to the well-known family of the *Labridæ* ("wrasses," "lip fishes"). The species met with in the West Indies are regarded as poisonous when taken on coral reefs.

Tetragonurus.—This is a genus of the *Atherinidæ,* a family closely allied to the *Barracudas. Tetragonurus* may be met with on the Atlantic coast of Northern Africa, as well as in the Mediterranean. Some of the species are poisonous.

Caranx and Thynnus.—Certain fishes of these genera, occurring in tropical

seas, have been found to exhibit poisonous properties; although, as a rule, most of the species are wholesome. Therefore, it can only be said that caution should be exercised regarding the use of unfamiliar species.

Tinned Fish.—It is to be noted that tinned fish, especially when preserved without the aid of salt or oil, decomposes more rapidly after exposure in hot climates than elsewhere, and that alarming and even fatal results may arise from the ingestion of the ptomaines produced during the process of decomposition. Hence it follows that tinned fish should be consumed directly after the tin has been opened.

Poisoned Wounds caused by Fish Spines.—Injuries of this kind are of more frequent occurrence on tropical service than elsewhere. The poisonous element in such injuries is due either to a special poison-sac fitted at the bases of certain spines, or to the general cutaneous secretion with which the spines as well as other external parts are bathed. The weapon causing the puncture or laceration may be the spinous ray of a fin, or a projecting spine borne on the tail, or a spine projecting from a gill-cover. The fishes concerned in these injuries will now be briefly referred to.

Trygonidæ ("stingrays," or "stingarees") and *Myliobatidæ* ("eagle rays," "sea devils," "devil fishes").—In certain representatives of these families the dorsal surface of the tail is armed with one or more powerful spines which are sometimes barbed, and may attain a length of 9 inches. When at rest, the spine lies folded backwards, but it is capable of being instantly elevated into the erect position. It is the habit of these fishes to lie in shallow pits on muddy or sandy bottoms, and the wound is generally inflicted when, in wading through shallow water, one treads accidentally on the broad and flat upper surface of the fish. The spine is then at once erected, the powerful tail lashes upwards, and the formidable weapon is usually buried in the calf of the leg. These creatures are not provided with any special poison-bag; yet, nevertheless, the wounds inflicted even by small spines cause the most intense pain and profound mental and bodily prostration.

Scorpæna.—Fishes of this genus present a formidable array of strong sharp spines on all the fins, and also on the gill-covers. The poison is derived from the general cutaneous secretion, and causes great pain. The wounds are commonly the result of incautious handling of the fish during capture.

Synanceia.—In four species of this genus which are met with in the Indo-Pacific seas, the dorsal spines are grooved, and communicate each with a special poison-sac. Seining parties when wading in shallow waters are apt to tread upon these fishes as they lie half-buried in the sand. Wounds thus inflicted have been known to prove fatal.

Trachinus.—No special poison-sac has been found among the members of this genus, but the dorsal and opercular spines are provided with deep double grooves in which the cutaneous secretion lodges; being thus readily inoculated into punctured wounds.

Thalassophryne.—Two species of this genus occur on the Atlantic and Pacific coasts of Central America. The poison apparatus is more perfectly developed than in any other known species of fish. The dorsal and opercular spines are tubular, and they communicate with special poison-sacs situated at the bases of the spines.

Siluridæ ("cat fishes").—The sea-inhabiting fishes of this family, already referred to as being undesirable food fishes, are provided with powerful

spines on the pectoral fins, with which they inflict exceedingly painful wounds when incautiously handled. The poison is derived from glandular organs situated at the axils of these spines.

Moon-Blindness (*Nyctalopia*).—Young seamen and boys on first entering the tropics are liable to suffer from this affection. It is usually painless, but causes disability for days or weeks, inasmuch as the patients are unable to find their way about the ship during the gloom of the evening or by ordinary lamplight. In the Royal Navy this condition is commonly attributed to sleeping on the upper deck with the eyes, protected only by the eyelids, exposed to the fierce glare of the tropical sun or to the moonlight. The sailors' name for the affection has reference to the latter view as to its causation.

Other Ship Diseases.

It has been thought advisable to give, under the above heading, some further brief notes on diseases which, although not especially incidental to tropical service, are yet liable to occur in hot as well as in cold or temperate climates, and are, moreover, diseases whose causation and prevention are intimately associated with sanitation afloat.

Scurvy.—This disease, although happily banished from the regular naval service, still exists in the merchant service. It may be produced by a dietary in which there is an absence of fresh vegetables or of the lime-juice substitute; it may also arise from a dietary restricted to a very few articles of food; and, again, it may occur where there has been a sudden change of diet, and consequent non-assimilation. In relation to these three specific sources of the disease, exposure to wet and cold, excessive bodily labour, and other defective hygienic surroundings should be regarded as important predisposing causes.

In respect to prevention—(1) there should be a wide and varied food scale, which should not only contain the requisite quantities of albuminoids, fats, and carbohydrates, but which should also be varied from time to time in regard to the media through which these proximate food principles are conveyed; (2) the albuminoids and fats should be frequently ingested in the form of freshly-killed meat; (3) either fresh vegetables or well preserved vegetables, or lime-juice, should be included in the dietary; and (4) all contributing causes in the shape of defective hygienic surroundings should be carefully avoided.

Enteric Fever.—Of late years, owing to the watchful supervision exercised over water supplies obtained from the shore, and the largely extended use of distilled water, this disease, when occurring in the naval service, is almost invariably found to be derived directly from the shore. Naval experience also tends to show that the usual precautions with regard to disinfection and careful removal of excreta are sufficient to prevent the spread of this disease on board ship.

Dysentery.—The above remarks apply also to dysentery, a disease whose causation in the naval service has been mainly ascribed to the use of polluted food and water, and which has diminished in frequency as sanitation in these respects has improved. The proportion of cases occurring in the years 1860, 1870, and 1880 respectively, were 12·7, 3·5, and 1·2 per 1000 of all the men employed; and when we remember that the use of distilled water on board ship was coming into general use about the year

1870, the above figures are strongly suggestive of an intimate causative relationship between polluted water and dysentery.

The China station has been mainly responsible for the dysentery which occurs in the navy.

Diarrhœa.—Outbreaks of diarrhœa occurring on board ship may sometimes be traced to the water used in cooking having been contaminated with sea water; the contamination in the case of tea, soup, etc., being sometimes not noticeable to the taste until the occurrence of diarrhœa has called attention to the quality of the water supply.

CHAPTER IV.

MALARIAL DISEASES.

BY ANDREW DAVIDSON, M.D., F.R.C.P.Ed.

Nomenclature and Synonyms.—The term malaria,—from the Italian *mal' aria*, bad air,—however inappropriate etymologically, has been so generally accepted as signifying the infective agent of a well-marked group of febrile, cachectic, and nervous affections, that we shall continue to employ it in this sense, and without prejudice to the question whether water or air is the medium by which the infective principle effects its entrance into the system. The French designate the malarious process *paludisme* (*palus*, a marsh), and the malarial class of affections are frequently spoken of as *maladies palustres*, or marsh diseases,—terms which unfortunately suggest an exclusive marsh origin to these diseases, and are specially inappropriate to malaria in its epidemic manifestations. The following synonyms, though strictly applicable only to the febrile manifestations of the infection, are nevertheless used comprehensively to designate malarious affections generally:—Gr. *Διαλείπων πυρετός*; L. *febris intermittens*; E. *ague, intermittent fever, remittent fever*; Fr. *fièvre intermittente, fièvre tellurique, fièvre périodique*; It. *febbre intermittente*; G. *Wechselfieber, aussetzendes Fieber, kaltes Fieber.*

Nature of the Malarial Infection.

Malarial diseases, notwithstanding their diversity of manifestation, have long been recognised as constituting a natural group, the members of which are related to one another in their etiology, symptomatology, and pathology. They are observed to prevail together in the same localities, seasons, and years, to exhibit in most instances a marked periodicity, and to give rise to alterations in the same organs—notably in the spleen and liver. As the precise nature of the lesions, which should be regarded as pathognomonic of the infection, was, up to quite recent years, imper-

fectly understood, the limits of the group could not be very accurately defined. Considerations derived from the circumstances in which malarial diseases occur, and from the symptoms by which they are characterised, proved insufficient to prevent the inclusion of enteric and other non-malarial fevers into the malarial group. Indeed, there is good reason for believing that the process of differentiation is not yet complete, and that fevers are still classed as malarial that have no claim to be regarded as such. It is of importance, therefore, to seek for some character by which the group may be defined.

The existence of melanæmia is a distinctive character of the malarial infection. It may be accepted as established, that black pigment is never met with in the blood during the course of any febrile disease other than those of malarial origin, and that it is never absent from the blood of malarial fever, even in mild cases, although it may be so scanty as to escape detection in the blood drawn from the periphery. The presence of black pigment, whether free, or included in leucocytes or other bodies, in the blood of a fever patient during life, or in the spleen, liver, or other organ after death, is thus conclusive evidence of the malarious nature of the malady. Conversely, no form of fever which is not characterised by melanæmia, and which leaves no trace of melanin after death, is to be admitted into the malarial group.

For purposes of classification, then, the presence or absence of melanæmia may be taken as pathognomonic of malarial fever.

If we desire, however, to form an opinion respecting the nature of the malady, we must proceed a step further, and ask, What is the origin of the pigment which we recognise to be distinctive of malaria?

In 1880 Laveran discovered in the blood of fever patients certain pigmented and non-pigmented bodies, either free in the plasma, or attached, as he believed, to the red corpuscles, which he regarded as parasitic—as, in fact, the *miasma vivum* of the malarial infection. The presence of these bodies in the blood of patients suffering from malaria has been confirmed by observers in all parts of the world. Their existence is now entirely beyond dispute. Further observations must determine if they are met with in every individual case occurring in every malarious country, but the evidence, so far as it goes, points in this direction. Nor can the parasitic nature of most of the elements described by Laveran be any longer seriously contested; although views differ as to their origin and evolution, and their relation to the different forms and stages of fever. All observations go to prove that the

pigment, which we have come to regard as characteristic of the malarial infection, is elaborated by the agency of these bodies from the hæmoglobin. Melanæmia is thus the consequence of the presence in the blood of these parasites. Inoculation experiments, to which we shall have to refer in the sequel, have further proved that the blood of patients suffering from intermittent fever, and containing these bodies, is capable of giving rise to intermittent fever in healthy subjects.

In order, however, conclusively to demonstrate that in these parasites we have the *miasma vivum* of malaria, it has still to be shown that they are constantly present in the blood of malarial patients in all parts of the world, and that in pure cultures they are capable of giving rise to the infection.

The evidence of their association with malarial fever in the principal endemic seats of the infection is already very weighty, although not complete and exhaustive. But as yet all attempts at culture have failed, and they have not been detected in the soil, air, or water of malarious localities. It is for these reasons, as well as from the difficulty of bringing the multiform phenomena of the disease into relation with the, as yet, imperfectly understood action of the parasites, that we have preferred to make melanæmia the distinguishing character of the group, — a character the existence or non-existence of which is a simple matter of observation, independent of all theories as to its origin.

Although many points respecting the origin and evolution of these parasitic bodies still remain obscure, the evidence is conclusive that they are intimately related to some of the essential phenomena of the febrile paroxysm, especially to the destruction of the red cells and to the formation of pigment, and, none the less, to the pernicious accidents that sometimes occur in the course of the infection. The nature of these bodies, therefore, claim our attention in connection with the etiology of the malarial infection.

The Etiology of Malaria.

The Parasites of Malaria.—If we examine the blood of a patient suffering from malarial fever, and who has not been treated with quinine, we will generally be able to detect, during some period of the fever, parasitic bodies which, according to Laveran, may be referred to one or other of the four following types, viz. :— (1) Spherical bodies; (2) flagellated bodies; (3) crescentic bodies; (4) segmented bodies or rosettes.[1]

[1] Laveran, *Du Paludisme et de son hématozoaire.* Paris, 1891.

1. *The spherical bodies* are formed of a colourless and very transparent substance, their contours being indicated only by a very faint line. They are endowed with amœboid movements, which are generally effected with a certain slowness; but in the form met with in tertian fever these movements are often so lively that they can only with difficulty be followed by the eye. These elements vary greatly in size, the smallest scarcely exceeding one-sixth, while the largest attain a diameter equal to or larger than that of a red corpuscle. The smallest of these bodies are entirely destitute of pigment; those of a somewhat larger size generally contain a few granules, and the pigment, as a rule, increases with the growth of the parasite. The amount and arrangement of the pigment within the parasites vary greatly. In the smaller bodies one or two centrally-placed granules may be observed; in the larger ones, the pigment is more abundant, and is either gathered toward the periphery, where it may be arranged in the form of a crown, or it may be irregularly disseminated throughout the whole of the protoplasm. In the final stages of their development, the pigment becomes amassed in the centre.

The pigment granules are often observed to be in lively motion, but the motion is neither constant nor steady; it may stop for a short time, and then recommence; it may diminish in activity, and then become more lively. The motion appears as if it were communicated to the granules by the protoplasmic mass forming the substance of the spherical bodies. The movement of the pigment ceases when the bodies assume their cadaveric forms. (See Fig. 4, *g*.) These bodies, although spoken of as spherical, are often very irregularly so; discoid and annular forms are frequently met with in some stages and forms of the disease.

The largest spherical bodies are, of course, always, and those of smaller size are occasionally, found free in the plasma, although the latter are more generally found within the substance of the red corpuscles. They are, in fact, *Cytozoa*. In some instances they appear to be attached to the exterior of the corpuscle, but their normal development is accomplished within the blood cell. They live and grow at the expense of the corpuscle, which becomes paler and paler in proportion as the parasite grows in size, and as the pigment it contains increases in amount. The parasite-bearing corpuscles are frequently altered in appearance. They often lose their discoid form and become pale and swollen; in other instances, they become dark and shrunken. Two or more of these hyaline bodies may be found within a single corpuscle.

The larger, spherical, pigmented bodies may be distinguished from melaniferous leucocytes by the fact that the latter are visibly nucleated, and by their nuclei becoming coloured with carmine; while the nuclei of the spherical bodies are not to be detected by ordinary examination, and their substance is not at all, or only slightly, coloured with carmine. (See Fig. 4, *b*, *c*, *d*, *e*, *f*, and *l*.)

2. *Flagellated bodies.*—These elements are spherical in form, and furnished with from one to four long, delicate, transparent, mobile filaments, which often terminate in a slight bulb. In some cases a spindle-shaped swelling may be observed in some part of the length of the flagellum. The spherical bodies to which the filaments are attached are about the size of a red corpuscle, or a little larger, and are pigmented, the pigment being often found in lively motion. The flagella measure in length three or four times the diameter of the body to which they are attached. Laveran aptly compares their movements to those of an eel fixed by the tail and trying to free itself. The filaments sometimes become detached, and rapidly disappear from the field. This variety is less frequently met with in malarious blood than the other forms. Laveran found flagellated bodies in the blood of 92 only out of 432 patients examined, but their frequency probably varies in different countries. Carter found them in six out of seven cases examined in Bombay. Their development has not, as yet, been satisfactorily made out. Laveran states that the crescentic bodies become oval and finally spherical in shape, and that from these crescent-derived spheres the mobile filaments escape. That this is one way in which they arise is probable, for Celli and Guarnieri, as well as Sakarhoff,[1] have witnessed the transformation of crescents, first into oval bodies, then into spherules, finally into flagellated bodies. Canalis found that after the appearance of crescentic bodies in the blood of a patient suffering from fever, flagellated bodies could generally be discovered if one could follow the patient long enough, but always in company with round bodies of a somewhat larger size, as if the mass of the round bodies was diminished by the formation of the flagella.[2] On the other hand, flagellated bodies are met with in blood in which no crescentic bodies can be observed.[3] (See Fig. 4, *h*, *i*.)

3. *Crescentic bodies.* — These are cylindrical elements, tapering more or less towards their extremities, and curved upon themselves. To the same class belong the oval and rod-shaped bodies. They

[1] *Annales de l'Instit. Pasteur*, July 1891.
[2] *Archiv. italiennes de biol.* tome xiii. p. 275.
[3] *Archiv. per le scienze mediche*, vol. xiv. Torino, 1890.

are colourless and transparent, with more or less of immobile pigment in the centre, and do not possess amœboid movements. When fully developed they are larger than a red corpuscle. In fresh blood their contours are indicated by a single faint line. The larger crescents are always free in the plasma, but smaller ones are found within the red corpuscles. This variety is most commonly associated with irregular forms of fever of long intervals, with grave types of disease, with relapses, and with the malarial cachexia. Canalis believes that he has traced their development from small hyaline protoplasmic masses belonging to the spherical type which we have described, and situated within the red corpuscles. These hyaline bodies enlarge and become pigmented; the pigment collects in the centre, the bodies lose their amœboid motion, assume a long oval form, then become curved into a crescent. The blood corpuscle in which the crescent develops loses colour, and is in great part destroyed; a faint curved line within the concavity of the crescent is all that remains of the corpuscle. The fate, as well as the origin, of this body is still obscure. Canalis holds that it is gradually transformed into a spherical body, which becomes segmented, and the small hyaline segments, entering anew the red corpuscles, give rise to a new generation of crescents. Bastianelli and Bignami have not succeeded in detecting the sporulation of these bodies, and incline to regard them as sterile forms of the parasite. The more recent observations of Mannaberg [1] and those of Babes and Gheorghiu [2] go to show that

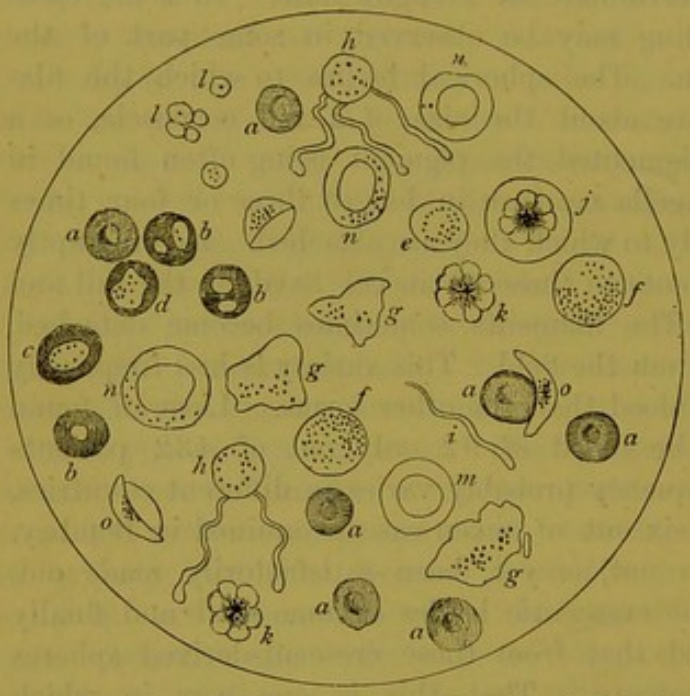


FIG. 4.—Schematic Representation of Parasites of Malaria.

a, Red corpuscles without parasites.
b, *c*, *d*, Red corpuscles containing one or more spherical pigmented or non-pigmented bodies.
e, *f*, Free spherical bodies.
g, Cadaveric forms.
h, Flagellated bodies.
i, Flagellum detached.
j, *k*, Segmented bodies.
l, Small spherical bodies singly or in groups.

[1] *Berlin. klin. Woch.* June 1892.

[2] *Archiv. de méd. expér.* March 1893.

these crescentic bodies are not simply degenerative forms, but they do not offer any satisfactory solution of the problems connected with their life-history. (See Fig. 4, *o.*)

4. *Segmented bodies or rosettes* (*Corps en rosace* of Laveran) are regularly segmented bodies, having a mass of pigment in their centre. Like all the other elements, they were first recognised and described by Laveran, but it was left to Golgi to discover their true nature. These bodies are now looked upon as sporulation forms, derived from the development within the corpuscles of the small hyaline bodies belonging to the first type. Each segment or division forms a new spherical or amœboid body, which attacks a fresh corpuscle, and runs through various phases until it forms a segmented body or rosette. The central mass of pigment is set free in the blood when the segmentation is complete. The development of these various bodies will be better understood in connection with our study of the successive phases in the evolution of the parasites of the tertian and quartan types of fever. (See Fig. 4, *j*, *k*.)

Along with these, and sometimes even when none of these are to be detected, there are found in the blood of fever patients leucocytes and larger white cells (macrophages), containing pigment. (See Fig. 4, *n*.) It appears highly probable, as we have already said, that the pigment contained within these phagocytes has been formed by the action of the parasites upon the hæmoglobin of the corpuscles, and that on being set free in the blood during the process of segmentation, it is taken up by the leucocytes in order to be removed from the circulation. Recent researches of Nepveu appear to demonstrate that algæ and schizomycetes are occasionally to be met with in the blood of malarial patients in Algeria, and some of these micro-organisms are not unfrequently to be found in considerable numbers. It has not been proved that any of these are constantly present in the blood of fever patients, or that any of them are characteristic of a particular form of the disease. It is not, however, to be inferred that, when present in the blood in large numbers, they are entirely innocuous, and for this, among other reasons, the presence or absence of micro-organisms other than those we have described should be determined.[1]

Examination of the Blood.—The object in examining the blood may be—(1) to ascertain the presence or absence of parasites and pigmented leucocytes; (2) if parasites be present, to discover the type and stage of the febrile process; (3) to trace the forms and phases of the parasitic bodies; (4) to study their minute structure. When the examination is made with any of the first

[1] *Comptes rendus Soc. de biol.* 1891.

three objects in view, it is better to examine the fresh blood without any preparation. In order to study the minute structure of the parasites, dried and stained preparations are required. For a simple diagnostic examination of the blood, a magnifying power of 500 diameters will usually suffice; and a good natural light should be made use of. The layer of blood to be examined should be thin, so as to display the individual elements. When the examination is protracted, the edges of the preparation may be sealed; but, as a rule, the coagulation of the blood around the border of the cover glass is sufficient to prevent evaporation, or the entrance of air germs. It must not be expected that in every case in which these elements are present they will be discovered at the first glance; repeated examinations of different preparations may be necessary before they are detected. The blood may be preserved by drying, effected by passing the glass, with a thin layer of blood, two or three times over the flame of a spirit lamp; the side on which the blood is placed being turned away from the flame. Preparations made in this way may be preserved for a long time.

For colouring the parasitic elements, methylene blue is frequently resorted to. It colours the spherical bodies, crescents, and segmented bodies a pale blue,—much paler than the nuclei of the leucocytes,—the red corpuscles, in most cases, retaining their normal colour. Laveran recommends that we should pour a few drops of a concentrated aqueous solution of methylene blue upon the glass on which there is dried blood, wash it with distilled water at the end of thirty seconds, and mount the preparation dry, as Canada balsam renders the elements too transparent.

Then there is the double coloration by eosine and methylene blue. The preparation is treated for thirty seconds with a concentrated aqueous solution of eosine, washed in distilled water and dried; then treated with a concentrated aqueous solution of methylene blue for thirty seconds, washed and dried. The blood corpuscles become coloured of a rose tint, those invaded by parasites colour less distinctly. The round bodies, whether within or without the corpuscles, the crescentic, and segmented bodies become pale blue; the protoplasm of the leucocytes is coloured pale blue, and the nuclei dark blue.

Other methods of preparation and coloration will be found described in the monographs of Celli and Guarnieri,[1] of Mannaberg,[2] of Malachowsky,[3] and of Feletti and Grassi.[4]

The frequency of the Parasites in Malarial Blood.—

[1] *Annali di Agricoltura*, 1889.
[2] *Centralbl. f. klin. Med.* 1891, No. 27.
[3] *Centralbl. f. klin. Med.* 1891, No. 31.
[4] *Riforma med.* 1891, No. 232.

Laveran, whose field of observation was in Algeria, found one or other, and in most cases several, of the bodies we have described in 432 out of 480 cases examined. Osler, Councilman, and James, in the United States, found them in from 90 to 100 per cent. of their cases. Paltauf, Bamberger, Kahler, and Quincke, in Germany and Austria, succeeded in finding them in all the cases they investigated; but the numbers examined were comparatively few. Canalis examined the blood of sixty-three persons suffering from malarial fever, and he met with these bodies in every instance; and the Italian observers rarely, if ever, fail to discover the parasites in the blood of malarious patients. Vandyke Carter, in Bombay, found pigmented parasites, either free, or both free and sessile, in about 13 per cent. only of the cases examined by him; but the cases were chronic ones, and he did not include the instances in which there were minute changes within the blood discs, or a slight pigmentation of leucocytes; if these had been included the proportion would have been increased. While admitting that in Bombay the microscope frequently failed to supplement the ordinary means of diagnosis, yet upon review he regarded such failures as incidental, and thinks that an infection "not abrogated will always be detectable through its attendant blood changes."[1]

There are few malarious countries in which these bodies have not been detected, but some observers record negative results. Fischer, of Kiel, examined the blood of eighty malarious patients in the Cameroons, the West Indies, and other regions; and Schellong,[2] that of a large number of patients in New Guinea, without meeting with these bodies. Soulié has found them in the blood of malarious patients in Alger; but his examinations have also often proved negative. What do these failures indicate? Are these bodies present in some, and absent in other cases of malarial fever? Are they met with in certain countries and not in others? We think it more probable that the negative results of some examinations are to be ascribed to the want of skill, habitude, and patience in searching for the parasites. This conclusion is strengthened by the fact that other observers have succeeded in detecting these bodies in the blood of fever patients coming from the same localities. Kohlstock, for example, demonstrated the parasites in the blood of a patient who had contracted fever at Finschhafen in New Guinea during a severe epidemy that occurred there in 1892, when thirteen out of the small European population were carried off within two months. He has also found them in the blood of patients from the East and

[1] *Scientific Memoirs. Indian Med. Officers*, part iii. 1887.
[2] *Internat. Journ. of Med. Science*, January 1891.

West Coasts of Africa.[1] Prout has observed them on the West Coast.[2] When we remember that they have now been found in almost every case by many of the best observers, and in many parts of the world, we hesitate to attach great importance to the negative results to which we have alluded.

It should be added that these parasitic bodies have never been found in the blood of patients suffering from any other disease than malarial fever. They have, however, been observed in the blood of malarious patients free from fever, but suffering from cachexia.

The Evolution of the Parasite in relation to the Types of Fever.—As the observations of Golgi[3] respecting the evolution of the parasite in the two simple types of fever, namely, quartan and tertian, have been in the main confirmed by other observers, and furnish an explanation of the phenomena of intermittence and periodicity—phenomena that had up till then baffled all attempts at interpretation, it will be necessary here to give a brief account of them. In quartan fever the *interval* between the beginning of one paroxysm and the commencement of the next is seventy-two hours. The *intermission*, that is, the period between the termination of one paroxysm and the beginning of the next, usually varies from sixty-four to sixty-eight hours. The long interval of the quartan type of fever permits the evolution of the parasite to be traced with greater certainty than has hitherto been possible in respect to the tertian and quotidian types. We shall, therefore, follow the appearances of the parasite during the progress of quartan fever, which, we may remark, usually begins at or after noon.

If we commence our examination of the blood of a patient suffering from quartan fever on the morning of the first day of the apyrexia, hyaline bodies containing pigment granules towards their periphery are to be observed within certain of the blood corpuscles, occupying from one-fourth to one-sixth of their diameter. If closely watched they are seen to move slowly in the protoplasm of the containing cell; this motion is indicated less by a change of position than of outline. The blood corpuscle containing the parasite is of normal size, and, apart from the presence of the parasite, of normal appearance.

If we repeat the examination in the afternoon of the same day, these bodies will be seen to have increased in size; and this increase continues during the apyrexia. Some of these elements may also be observed free in the serum, but these are not numerous. These

[1] *Berlin. klin. Woch.* January 1893. [2] *Lancet*, August 1, 1891.
[3] *Archiv. italiennes de biol.* 1887.

in the serum, like those within the corpuscles, change their form slowly. A slow movement of the pigment granules is also observable both in the intra- and extra-corpuscular bodies. The microbe gradually increases in size, and *pari passu* with this growth two changes are to be observed: the blood corpuscle grows paler, and the pigment within the microbe augments.

If we again examine the blood on the morning of the day on which the paroxysm is due,—some six to ten hours before the fever fit,—the microbe will be seen to have grown so much that it now almost fills the whole corpuscle, of which only a very fine peripheral portion remains. Soon no trace of this is longer visible, and we have a free spherical, pigmented body, consisting of a whitish substance in which the pigment is irregularly distributed. About this time a concentration of the pigment towards the centre takes place; first in streaks and then in irregular or star-formed masses, which always converge towards the centre. The concentration becomes more and more marked, until the pigment is collected in a sharply defined globular mass in the centre. As the concentration of the pigment proceeds, incipient signs of division appear towards the periphery, and go on until it affects the whole substance of the body. As a result of this, six to twelve pear-shaped bodies are found arranged symmetrically around the central pigment mass, like the leaves of a daisy around its central disc. This is the *forme de marguerite*, or the *rosette* of Golgi. The body is now said to be segmented. These pear-shaped bodies become rounder, and separate a little from each other; the delicate substance which binds them together disappears, and now we have irregular groups of round bodies, numbering from six to twelve, among which is to be seen, centrally or peripherally, the little pigment mass around which the segmentation took place. The segmented bodies are thus no longer to be met with, and the non-pigmented amœboid bodies which become free are with difficulty recognised in the blood of the periphery. They are conjectured to take up their abode temporarily in some internal organ, such as the spleen, to reappear in the blood cells the following day as non-pigmented bodies, representing the first phase of the succeeding cycle. Some of these young amœbæ may, however, be seen within the blood cells soon after the disappearance of the segmented bodies; and, according to Antolisei, even before the commencement of the paroxysm. These, apart from melaniferous leucocytes, are the only forms met with in the blood during the period of defervescence and the first hours of the apyrexia. In short, to sum up in the words of Golgi: "The parasites develop gradually in the red corpuscles, passing from

their initial amœboid to pigmented forms, which grow by appropriating to themselves the substance of the corpuscle, until a certain evolutionary phase having been attained, they undergo a series of characteristic metamorphoses, the final result of which is their segmentation, which coincides with the commencement of the rise in the temperature, or a little before. As a result of this process, new generations of parasites originate, attacking other red blood corpuscles, repeating the cycle, and leading to other paroxysms." It appears from recent researches that all the adult forms do not undergo sporulation, some of them become free in the plasma, and degenerate. That the febrile paroxysm is related in some way to the sporulation of these bodies is sufficiently evident; but it has not been determined in what way the introduction of free spores into the circulation gives rise to fever. Is the febrile paroxysm caused by mechanical irritation of the nervous centres? or is there some pyrogenic substance set free in the blood on the breaking up of the segmented bodies? These are questions to which, as yet, no final answer can be given; but the latter view seems the more probable.

If nature had no means of defence against the attacks of these parasites, we see no reason why, having once effected an entrance into the system, they should not by their multiplication through successive generations speedily destroy the whole of the red corpuscles. But such means of defence do exist. Apart from the assistance of art, nature is able to summon to her aid hosts of supporters capable, not only of waging a defensive war on her behalf against her assailants; but ultimately, in many cases, of completely destroying them. No sooner has the fever been lighted up than a wonderful drama proceeds to develop itself within the circulation. Numerous leucocytes appear on the scene of action, and these are promptly supported by a host of macrophages derived from the spleen and other organs. These phagocytes are seen rapidly to infold or ingulf the smaller parasites. Even the large flagellated bodies are attacked. The macrophage advances towards the parasite, spreads itself out, and in a little is seen to have surrounded it. After a time the parasite is destroyed, and all that remains of it are a few pigment granules. The only form of the parasite which seems to resist or escape the attacks of the phagocytes is the crescent-shaped variety. Carter observed the phagocytes repeatedly to turn from and leave the crescents untouched, which, as he suggests, may account for their longer persistence in the blood.[1] The pigmentary débris in the circulation is also taken

[1] *Loc. cit.* p. 151.

up by the leucocytes, and is more slowly eliminated from the circulation. Numerous pigment-bearing leucocytes are observed in the blood during and after the paroxysm. This process of phagocytosis is as distinctly periodical in its manifestations as is the development of the parasites in the fever itself. It has been conjectured that the increase in the temperature of the body is the cause of the increase of the phagocytes. In favour of this view is the fact that leucocytes become more active under the influence of artificial heat, rapidly seizing and ingulfing grains of pigment that are put in their way. Cold, on the other hand, has been proved to have a noticeable influence in diminishing the activity of these bodies.[1] From observations made by Golgi on the blood of the spleen during the febrile paroxysm, he concludes that what is observed going on in the peripheral circulation is only as it were a by-play; that the real drama is enacted in the spleen, liver, and bone-marrow. He found phagocytes to be much more numerous in the blood of the spleen at the onset of the paroxysm than in that obtained from the finger, and he found also that the phagocytes of the spleen often contained a great number of malarial parasites.

Instead of describing in detail the evolution of the hæmatozoon of tertian fever, it will be sufficient to state the principal differential characters of the two forms of parasite as given by Golgi.[2]

Biological Characters.—(*a*) *Difference in the evolution cycle.*—The parasite of tertian completes its cycle in two, that of quartan in three days. (*b*) *Difference in the character of the amœboid movements.*—The movements of the hæmatozoon of quartan fever are slow, and difficult to observe except in the first stage. In tertian, the movements are so lively that they are difficult to follow; the young parasite rapidly protrudes reticulated processes in all directions, reaching to the periphery of the corpuscle; then, withdrawing them, other processes in succession emerge and furrow up the corpuscle in every direction. (*c*) *Difference in their action on the corpuscles.*—The parasite of tertian decolorises the corpuscle more rapidly and completely than that of the quartan. In the quartan, the corpuscle retains a yellowish colour until the last phase of its destruction, that is, until it is represented only by a thin border.

Morphological Characters.—(*a*) *Difference in the aspect of the*

[1] Maurel found that leucocytes could only bear a cold below 16° C. for a short time, and that when the temperature fell to 14° C. they died immediately. A temperature of between 44° and 47° C. proved fatal to them. They are active at the normal temperature of the body, but a febrile state under 40° C. (104° F.) is still more favourable to their activity. (*Comptes rendus Soc. de biol.* 1890.)

[2] *Archiv. italiennes de biol.* tome xiv.

parasites.—In the tertian, the protoplasm of the parasite has a much more delicate appearance than that of the quartan. The contour of the parasite in quartan fever is more defined and distinct. This point of difference is most marked in the first stage of the development of the two varieties. (*b*) *Difference as regards pigment.*—In the quartan, the pigment presents itself in the form of granules and rodlets, coarser and larger than in the tertian, in which they are extremely delicate. (*c*) *Differences in the mode of segmentation.*—These will be best understood by examining the figures 11–15, A and B, in the accompanying illustration (Plate I.): A represents the development of the parasite of tertian, B that of the parasite of quartan (after Golgi). As in the case of quartan, all the adult forms of the tertian variety do not undergo sporulation; those that do not go on to segmentation pass into the plasma. The number of segments in the sporulation bodies of quartan is from six to twelve, in tertian from fifteen to twenty, and are of a smaller size.

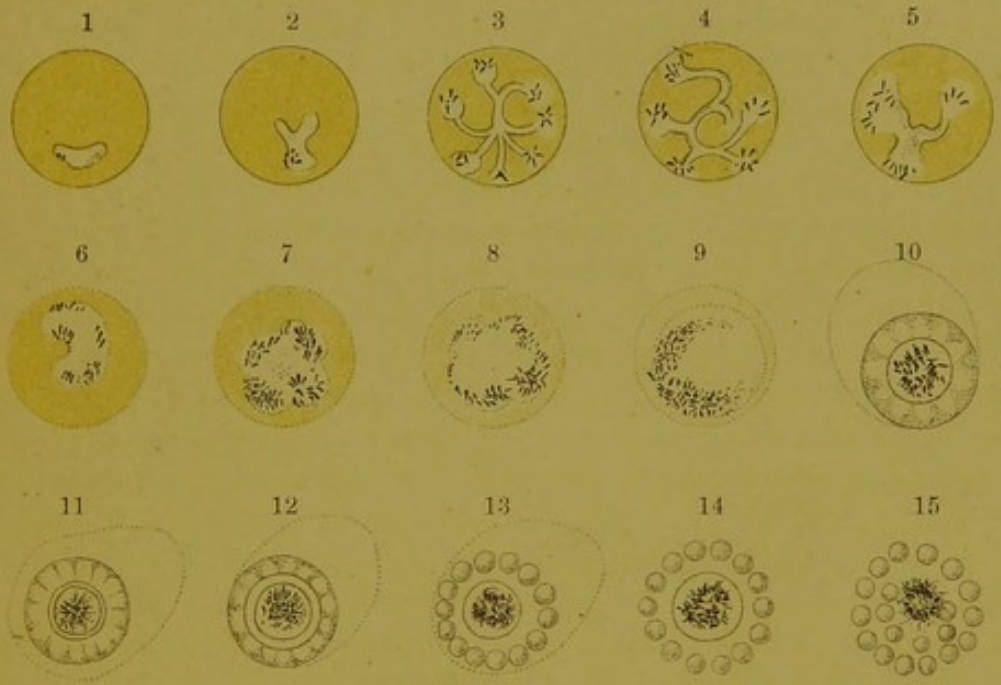
Such are some of the more striking differences between the forms of the parasite in the tertian and quartan types of fever; but one or two points still require to be noticed. The tertian, although its paroxysm recurs every other day, may appear daily, the paroxysms of alternate days corresponding in intensity, duration, and period of onset. This is the variety known as double tertian (*tertiana duplex*), and may readily be, and is frequently, mistaken for quotidian. This variety of tertian is believed to be connected with two crops of parasites running their cycle in the blood, but coming to maturity on alternate days. In the same way a quartan, which has normally two days of apyrexia interpolated between the fever periods, may have febrile paroxysms two days in succession, with one day of intermission (*quartana duplex*), the paroxysms being alike every fourth day; or there may be daily paroxysms, those of every fourth day being alike (*quartana triplex*); the latter form again may be, and often is, mistaken for quotidian. These forms are accounted for by the maturation of different broods of parasites, each running the cycle of quartan.

These, then, are the evolution-cycles and the distinctive characters of the parasites of the two simple clinical forms of malarial fever, such as are met with in temperate climates, in spring, in late autumn, and in winter. From these observed facts the following laws have been deduced, which will probably be found to apply to all the different forms and types of malarial fever both in temperate and in warm climates:—

(1.) Each paroxysm is related to the evolution-cycle of a generation of parasites.

A

SOME PHASES IN THE EVOLUTION CYCLE OF TERTIAN FEVER.



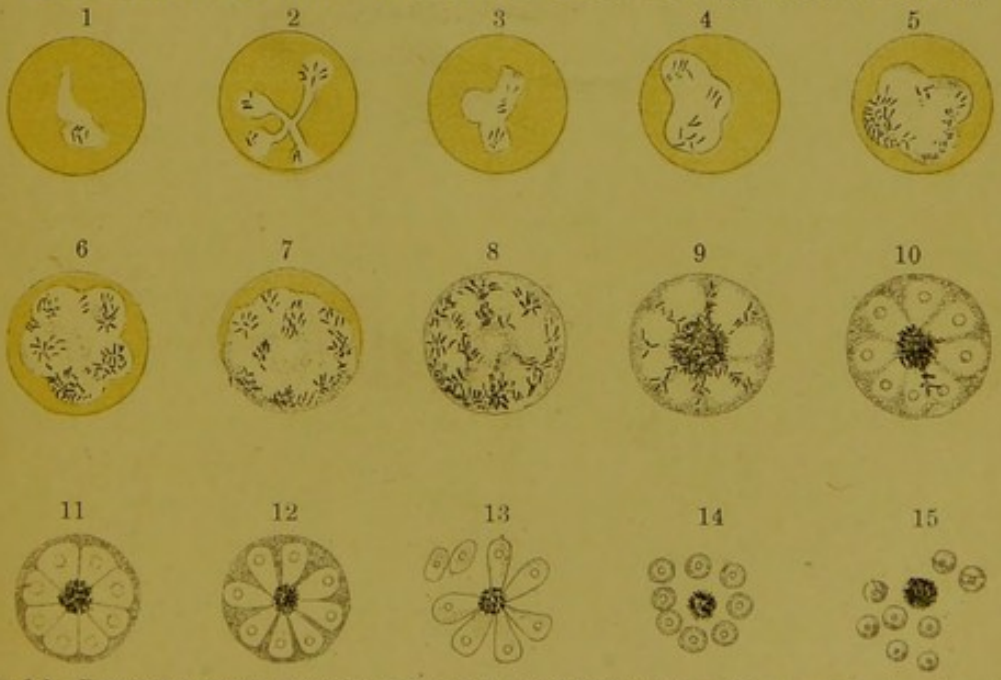
FIGS. 1-5.—1st phase. Progressive growth of the malarial parasite, with progressive decolouration of the corpuscle.

FIGS. 5-10.—2nd phase and commencement of 3rd. Development of parasite to maturity. In figure 10 the differentiation at the periphery is already beginning to show itself.

FIGS. 11-15.—3rd phase. Gradual differentiation of small globules representing a new generation. Fig. 15 represents a second mode of segmentation.

B

SOME PHASES IN THE EVOLUTION CYCLE OF QUARTAN FEVER.



FIGS. 1-3.—Progressive growth of parasite during first day of the apyrexia with progressive transformation of hæmoglobin into melanin.

FIGS. 4-6.—Later growth of parasite during second day of the apyrexia—the parasite invades the greater part of the corpuscle.

FIGS. 7-15.—Third day (day of paroxysm, it being understood that the paroxysm begins about mid-day). Further development, maturation, and segmentation of the parasite. In figure 7 there still remains a trace of the corpuscular substance. In figure 8 this has entirely disappeared.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(2.) The commencement of each paroxysm coincides with the maturation of a generation of parasites.

(3.) The severity of the paroxysm in a given type of fever is in direct relation to the number of parasites in the blood. It does not necessarily follow, however, that the gravity of the case is proportionate to the intensity of the paroxysm, and this point should be borne in mind.

Observations have been made on the summer and autumn fevers of Italy, which, although they still require confirmation in many points, are of special interest, as they represent the forms more frequently met with in warm climates. In this group are included —(1) remittent fever, that is, fever with more or less marked exacerbations daily, or every other day, or every third day, but without any period of complete apyrexia; (2) fevers in which no very distinct exacerbation can be noticed, the pyrexia being often practically continued; (3) fevers with long or indefinite periods of apyrexia, and with paroxysms of irregular duration; (4) intermittent fevers with protracted paroxysms. The fevers belonging to this group present a character of gravity, in some cases even of malignity, foreign to the simple types we have just considered. They are accompanied with great debility, have a tendency to relapse, often rapidly produce cachexia, occasionally develop what are called pernicious symptoms, and, finally, they are frequently obstinate or intractable to treatment.

The microbic characters of the group are, as we have said, still imperfectly known. Marchiafava and Bignami[1] recognise two varieties of parasites as the cause of these fevers—(1) a parasite running its cycle in twenty-four hours, which is the course of a quotidian fever; (2) a special parasite having an evolution-cycle of forty-eight hours, like the simple tertian, but distinguished from it by special characters. The parasite of the quotidian fever of Marchiafava and Bignami is a small non-pigmented intra-corpuscular protoplasmic mass, endowed with active amœboid movements. It may proceed to segmentation[2] without being pigmented; more frequently it contains a few granules of pigment. The segmentation of these bodies occurs within the red corpuscles, and principally in the spleen, where they are to be found at the beginning of a paroxysm. At the end of the period of apyrexia, blood corpuscles containing parasites are met with which are smaller than normal, shrivelled, and of a

[1] *Riforma med.* 1891, No. 217; also *Sulle febbri malariche estivo-autunnali.* Roma, 1892.

[2] It would appear from the researches of Celli and Sanfelice that all of these bodies do not undergo sporulation. They believe that some become converted into crescents. *Fortschritte der Medicin*, 1891.

dark yellow or bronze colour. Although Laveran contends strongly against the recognition of special varieties of parasites, it is to be remarked that in his description of the bodies found in connection with quotidian fever of primary invasion, the words repeatedly occur: "corps sphériques de très petit volume." This seems to confirm the view that quotidian fevers are distinguished by a small amœboid parasite; and this much may, perhaps, be taken as proved. If we are to suppose that quotidian paroxysms may result from daily maturing crops of the quartan and tertian forms of fever, and that, in addition, a distinct form of quotidian with a special parasite exists,—points as yet by no means settled,—we should have a striking explanation of the remarkable description given by Celsus of the "various and manifold" character of the quotidian type.[1] The parasite of the severe or malignant summer tertian of Marchiafava and Bignami is smaller and less pigmented than that of the mild tertian, and often assumes an annular form. In the mild form of tertian fever the invaded blood corpuscles are swollen or normal; in the severe summer fever they are shrunken, and darker than normal. The mild tertian has no crescentic phase in its development; while in the summer tertian such a phase exists.

Although special parasitic forms are thus generally associated with each clinical type of fever, yet this association is not so absolute as to justify us, until further evidence be adduced, in assuming a plurality of specific malarial organisms. It appears, at least, improbable that there should be as many distinct parasites as there are types of fever. The facts, so far as they are at present known, are consistent with the view of Laveran, that we have to do in malaria with a single polymorphous hæmatozoon. It is perfectly conceivable that this organism may undergo modifications of form and variations in its virulence by certain conditions of season and soil.

Space does not permit us to enter even cursorily into the researches of Celli and Guarnieri,[2] of Feletti and Grassi,[3] and of Mannaberg[4] on the intimate structure of the malarial parasite; suffice it to say, that it is to be looked upon as a single protoplasmic cell, containing a vesicular nucleus, within which may be distinguished one or more nucleolar particles. This organism multiplies by forming spores. The nucleus divides directly, and not by way of karyokinesis.

Results of Inoculation.—If the blood of a patient suffering from malarial fever be injected within the veins of a healthy person, it uniformly produces the disease; and, in most cases, it reproduces

[1] *Lib. tertius*, sec. iii.
[2] *Archiv. per le scienze mediche*, vol. xiii.
[3] *Riforma med.* 1890.
[4] *Centralbl. f. klin. Med.* 1891, No. 27.

the same type of fever. Gualdi and Antolisei have succeeded in reproducing the quartan type by making use of the blood of a patient suffering from a primary infection—by primary infection, I mean a fever which began as a quartan, and not a quotidian or a tertian which had changed into a quartan.[1] The induced fever in this case appeared on the thirteenth day, was regular in type, and the blood of the person inoculated contained the quartan parasite, which in each interval ran through its typical evolution cycle in the same way as it did in the blood of the patient from whom the inoculation was made. Di Mattei[2] has also succeeded in reproducing, not only the special form of fever, but has shown that two parasitic forms may be present in the blood at the same time, causing two distinct types of fever, each running its course independently. In a number of the recorded experiments, however, although fever resulted from the inoculation, the exact type was not reproduced.

Analogous Hæmatozoa in Birds.—Danilewsky[3] has given an interesting description of the parasites discovered by him in the blood of some species of birds, principally magpies, jays, ravens, and owls, which, as regards appearance, habitat,—the red blood corpuscles,—mode of development and sporulation, and the pathological changes to which they give rise, bear a very close resemblance to those which we have described as occurring in human blood. He concludes that certain birds are subject to three forms of malaria —(*a*) an acute infection, characterised by hyperthermia and symptoms of grave disease due to the presence of a "cytosporozoaire," entirely analogous to the parasite of malaria as met with in man; (*b*) a chronic infection, without manifest fever, the blood presenting flagellated bodies and crescents; (*c*) a mixed form in which the two sets of parasites are found together.

Grassi and Feletti have found the same parasites in sparrows and pigeons in the malarious districts of Sicily, especially during the unhealthy season.

Question of Classification. — The precise place of the malarial parasite in the zoological system has not yet been fixed, but there is almost complete unanimity among authorities in classing it among the *Protozoa*. Metchnikoff considers that it belongs to the class of *Sporozoa*, and more particularly to the sub-class of *Coccidiidea*, many of which are parasites of the vertebrata. Feletti and Grassi incline to place it amongst the *Rhizopoda*. This question of classification is one, however, which must be left to naturalists to decide.

[1] *Riforma med.* 1889, No. 264.
[2] *Ibid*, 1891, No. 121.
[3] *Annales de l'Instit. Pasteur*, Dec. 1891.

Conditions of Endemic Development.—*A Soil Disease.*—Malaria is intimately connected with the soil. The crew of a vessel, if free from malaria to begin with, may sail in any latitude, and may be exposed to every vicissitude of weather, without being liable to contract the infection, so long as they do not approach to a malarious shore. Some instances, it is true, have been recorded of outbreaks of aguish diseases at sea, which have been ascribed to exhalations from moist earth, from wet or drying wood, from coal, from mouldy store-rooms, or to some similar local cause, comprehended by the French under the term *marais nautique*.[1] Interesting and, from an etiological point of view, important as these exceptional cases are, they do not require us to modify the proposition that malaria arises only in connection with the soil. Here, then, is the primary conclusion respecting the distribution of the infection, that it is restricted to the land surface of the globe. However it may be conveyed or communicated, it is generated only in connection with the soil.

Geographical Distribution.—If we examine the subject somewhat more closely, it will be seen that it is only a certain portion of the land surface of the globe that is subject to malaria. In other words, malaria has its latitudinal and altitudinal limits, although these do not admit of being very precisely defined.

As an endemic disease it does not extend higher than the 62° N. in the Eastern Hemisphere. In the Western Hemisphere its northern limit may be placed at from 50° to 51°, and its southern limit at 35°. In South Africa it cannot be said to extend, in an endemic form, south of the 30th parallel; or in Australia, south of the 20th. Malaria increases in prevalence and intensity, although by no means so uniformly as is generally represented, as we approach the equator. The altitudinal limit of malaria varies according to latitude. In Germany malaria is scarcely met with at a higher elevation than 1300 feet; in Italy it is met with at elevations of from 1000 to 2000 feet; at Karman, in Persia, it is severe at an elevation of 7500 feet; but here the summer heat is intense, the daily range great, and the country is extensively irrigated. In India malaria is met with at elevations of from 6000 to 7000 feet, perhaps even higher, but it is of a mild type, similar to the fevers met with in temperate climates. In Guadeloupe fevers are rare at altitudes of from 500 to 600 feet, and disappear at altitudes of 1600 to 2000 feet. The altitude, in fact, at which malaria appears, depends greatly upon local conditions, the chief of which are a marshy

[1] Marston, *Edin. Med. and Surg. Journ.* 1862; Holden, *Amer. Journ. Med. Scien.* 1866; Simon, *Rev. d'hygiène*, 1888.

state of the soil, and a high elevation of the summer temperature. It is thus pre-eminently a disease of warm climates. A simple enumeration of the tropical and subtropical countries in which malaria prevails, without an account of the local conditions on which that prevalence depends, would not serve any useful purpose, and for the minute detail that would add value to the study of the distribution of the disease we have no space. The reader who desires fuller particulars on this subject is referred to Hirsch's *Handbook of Geographical and Historical Pathology*, or to the work by the writer of this article on *Geographical Pathology*. One or two points respecting the distribution of malaria, which demonstrate its essential dependence on a specific soil miasm, as opposed to its supposed climatic origin, may here be noticed. There are no extensive continental regions within the tropics, at or near the sea level, unless they be the barren deserts of Atacama, Sahara, Arabia, and Central Australia, entirely free from malaria. Some regions enjoy, it is true, a partial immunity from the disease, notwithstanding the existence of conditions which might be thought favourable to its prevalence. The Malayan peninsula in general, and the island of Singapore in particular, although by no means exempt from malaria, enjoy a singular freedom from the more intense forms of the infection. The same remark applies to the northern coasts of Australia, as contrasted with the neighbouring shores of New Guinea.

While continental tracts all suffer in various degrees, there are numerous tropical and subtropical islands where malaria is unknown, and there are two notable examples of the disease having been introduced in quite recent years into islands believed to have been formerly free from the infection. We refer to the islands of Mauritius and Réunion. Near to Mauritius, now an intense focus of malaria, there is the island of Rodrigues, under the same climatic conditions, which remains free from the infection up to the present day. The Seychelles group, again, although within five degrees of the equator, with a high temperature and a heavy rainfall, enjoys a like immunity. Some of the Polynesian islands are exempt from the infection, others suffer from it in a high degree. Malaria does not exist at all, or is exceedingly rare, in New Caledonia, the Sandwich group, and the Marquesas islands, while in the New Hebrides and New Guinea it is excessively prevalent and severe.

When we see neighbouring islands, similar in climate and soil, the one healthy and the other malarious; and when we observe the sudden appearance of the infection in an island where it had previously been unknown, we are led to the conclusion that the

infection does not depend essentially upon climate or soil, but upon the presence or absence from the soil of some specific organism. When this specific entity is present, its prevalence, intensity, and seasonal development will be determined by meteorological and soil conditions, which are in themselves incapable of giving rise to the infection.

Relation to the External Configuration of the Country.—The distribution of malaria will be found to be determined to a considerable extent by the external configuration of different regions, in so far as this influences the humidity of the soil, the temperature of the soil and the air, and the force and direction of the winds. The basins of the great tropical rivers, such as the Congo, the Zambesi, the Niger, the Senegal, the Euphrates, the Indus, and the Ganges, subject to periodical overflow, and the lake regions of tropical and subtropical countries, are notably malarious. Level coast plains, moistened at certain seasons by tropical rains, and desiccated by the sun, are often unhealthy, especially when the subsoil is an impermeable clay retaining moisture. The elevated table-lands of India, Persia, and other tropical countries, present many localities with the same physical conditions as the level coast plains. The summer temperature of these table-lands is often excessive; the levelness of the surface renders drainage difficult, and the moisture derived from rainfall or irrigation tends to stagnate in the soil. It is chiefly to water-logging of the subsoil that we are to ascribe the excessive insalubrity of many of the shallow depressions running along the foot of mountain ranges, such as the Tarai, which skirts the foot of the Himalayas. Shut-in mountain valleys are frequently *foci* of malaria when the surrounding country is healthy. The evil effects of water-logging of the soil are in these cases augmented by a still condition of the atmosphere; and when the valley is enclosed by high rocky walls, by an excessive temperature.

The underground sheets of water met with in the oases of Africa are excessively malarious. Hewlett has attributed the great prevalence of fever at the military cantonments of Nasirabad, Neemuch, and Mhow, to the existence in these places of underground hollows at no great depth below the surface, in which the rainfall collects during the rainy season, and gradually evaporates during the dry season.[1]

Geological Relations.—The importance of the geological formation in reference to the prevalence of malaria is limited to the physical characters of the derived soil. Hirsch points out the

1 *Sanitary Measures in India*, 1882-83. London 1884.

comparative exemption of sandy soils from malaria, both in Europe and in India; but Day, on the other hand, whose observations refer to India, holds that "loose, porous, and sandy plains are most peculiarly favourable to its production."[1] Much depends upon the nature of the subsoil. Sandy soils in which water stands near to the surface are often malarious. Witness the severity with which fever raged among the British troops encamped at Rosendaal and Osterhoot, in the Netherlands, in 1794, on a soil described as a level plain of sand, perfectly dry on the surface, but percolated within a few inches of the surface with water. Malaria prevails upon the Alentego bank of the Tagus, the soil of which is of a similar nature, that is, sandy and dry superficially, but moist underneath. Sandy soils do not always require to be percolated with water to prove malarious. It is sufficient that they be occasionally rendered humid by copious showers. It was on a sandy soil mixed with disintegrated coral, and containing a certain amount of decayed vegetable matter, that two military stations in Mauritius were placed, at which not a single man escaped during the fever epidemic of 1866–68. Still it may be accepted as generally true that a dry sandy soil—dry, that is, for a considerable distance below the surface—is, as a rule, much more healthy than a clayey soil. The black cotton soil of India, which is remarkably retentive of moisture, is justly regarded as favourable to the production of malaria.

Calcareous soils, being little retentive of moisture, are generally healthy. Jourdanet contrasts the marshy province of Tabasco with the neighbouring dry calcareous province of Yucatan.[2] The former abounds with the worst form of malarious disease, while the latter is comparatively healthy.

Relation to Marsh and Subsoil Humidity. — This review of the relation of malaria to the external configuration of different regions, and to the geological characters of the soil, indicates the etiological importance of subsoil humidity in the generation of malaria. Malarial fever is so frequently found in relation to marshy soil, both within and without the tropics, that it has, not without reason, been designated marsh fever. If we take the term marsh in its widest significance as representing all soils in which there is permanent or temporary stagnation of water in the subsoil, what Maurel says of the relation between marsh and malaria is not far from the truth: "Peu de lois, dans le domaine de la pathologie, me paraissent mieux établis que celle qui fixe les rapports entre le paludisme et le marais; il n'y-a-pas de pays marécageux exempt de

[1] *Indian Annals*, 1859. [2] *Le Mexique et l'Amérique Tropicale.* Paris, 1864.

paludisme, et là ou règne le paludisme on peut le plus souvent affirmer le marais." [1]

The influence of humidity of the soil on the prevalence of malarial fever is most conclusively brought home to us when we observe the effects of a particular inundation in a malarious country, or when we mark the influence of a rise in the subsoil water of a locality caused by irrigation works on the health of a particular community. Examples of severe outbreaks of fever caused in both of these ways are extremely numerous. Verdan relates that at Ouargla, a ksour, formerly renowned for salubrity, was smitten with fever after the boring of an artesian well, which caused humidity of the neighbouring lands.[2]

Relation of Malaria to Soil Disturbance.—Next to the influence of humidity of the soil as favouring the development of malaria, must be placed the denudation and disturbance of the soil by the cutting down of forests, excavations, and tillage. We might almost be justified in speaking of a *marsh* miasm and a *telluric* miasm. The effect of the breaking up of virgin soil, whether prairie or forest land, in giving rise to malaria, has been so abundantly illustrated during the progress of colonisation in America, that it is almost needless to enter into details. No more striking instances of the febrific effects of excavation, conducted in malarious soils, can be found than those furnished by Hong-Kong and the neighbouring peninsula of Kouloon. The epidemic fever which ravaged the colony of Hong-Kong from 1841–46 was solely due to the excavations incident to the founding of the town of Victoria, and to the erection of Government establishments in other parts of the island. The same experience was repeated on a smaller scale in 1864, on the cession of Kouloon. No sooner were the diggings begun for the erection of barracks than fever broke out among the men of the 99th Regiment, who were encamped near to the excavations; and those companies suffered most who were quartered nearest to the cuttings. The mortality from malaria among the labourers engaged in digging the Panama Canal may be mentioned as another remarkable proof of the danger of excavations in malarious regions. A severe outbreak of malarial fever accompanied the construction of the Pedro railway in Brazil. Bourel-Roncière informs us that the workmen engaged on this line filled the hospital of Rio, and that the proportion of deaths among them was very high. After the railway was finished the adjacent localities again became more healthy. The greater liability of the Royal Engineers, than of other branches of the service, to suffer from fever is doubtless to be ascribed to their

[1] *Arch. de méd. nav.* 1887. [2] *Arch. de méd. mil.* 1885.

employment in public works involving soil disturbance. In Cyprus, in 1881, the ratio of admissions for paroxysmal fevers among the troops generally was 142·0 per 1000, whilst among the Royal Engineers it was as high as 842·4 per 1000. The instances are, indeed, extremely numerous in which the cuttings in connection with the opening of new railway lines have given rise to epidemic outbreaks in malarious localities even in temperate climates. Malaria we are told was very common in the arrondissement of Lapalisse (Allier) in France; but the fever increased to an extraordinary extent, and became general in the summer and autumn of 1855, during the execution of railway works effected in a soil distempered by the rains. More than one-third of the workmen were affected during these four months (Kelsch and Kiener). If we insist so strongly upon this point, it is because we believe that the great importance of soil disturbance as a cause of severe malarial outbreaks is often overlooked or underestimated. Unnecessary levelling and clearing of the soil in connection with the construction of camps, as well as of missionary and trading stations in malarial countries, give rise to untold sickness and mortality, which are often ascribed to the effects of climate.

Meteorological Conditions.—*Temperature.*—We are justified in ascribing the absence of malaria from the higher latitudes and altitudes where the temperature of the summer months falls below 58° F., and its greater prevalence in warm regions largely to the influence of temperature. It is difficult, however, to obtain exact evidence of the relation between increase of fever mortality and increase in temperature. The most satisfactory statistics bearing upon this point with which we are acquainted, are those furnished by the census reports of the United States of America.[1] In the Northern Atlantic States, the ratio which the deaths from malarial fever bears to the deaths from all diseases ranges from 2 to 6 per 1000; in the middle Atlantic States it ranges from 16 to 24 per 1000; while in the Southern States of South Carolina, Georgia, and Florida, the ratio ranges between 46·4 and 95·2 per 1000. We cannot be wrong, in this instance, in ascribing the latitudinal increase in the fatality of malaria in great part, at least, to the increase in temperature. It has been accepted as a law that, other things being equal, malaria increases in proportion to the rise in the mean temperature of the year, and more particularly to a rise in the mean temperature of the summer months. But while a certain elevation of temperature is undoubtedly necessary for the generation of malaria, it has not been proved that the prevalence of fever is at all in *direct* proportion to the increase in the mean tempera-

[1] *Tenth Census of the United States: Mortality and Vital Statistics*, 1880.

ture. As a matter of fact, when we come to inquire into the comparative prevalence of malarial disease in different countries and in different districts of the same tropical country, this relation is by no means so evident as is generally supposed. We find malaria, for example, as an endemic malady to be more intense in certain districts of Tuscany and in the Pontine Marshes, than in the south of India within ten degrees of the equator. The reason, we suspect, is that here other things are not equal, and that although a high temperature favours the evolution of malaria, the infection does not increase in intensity directly with a rise of so many degrees in the temperature. The following table, which gives the mean number of admissions and deaths from intermittent and from remittent and continued fevers per 1000 of strength among the European troops at different stations in India for a series of years, along with the mean temperature of the year, and of the months of May, June, July, and August, with the difference between the mean temperature of the coldest and warmest months, will illustrate how subordinate a position after all must be conceded to these meteorological elements in comparison to soil conditions in the development of malarial fever :—

	Fort William.	Lucknow.	Meerut.	Agra.	Saugor.	Mooltan.	Lahore.	Peshâwar.	Bombay.	Madras.
Admissions, intermittent fever,	117·2	122·1	574·5	278·0	703·1	528·9	1406·0	1119·3	917·3	39·7
Admissions, remittent and continued fevers,	166·5	168·2	126·7	181·0	169·3	200·3	48·7	717·8	94·5	116·6
Deaths per 1000 from intermittent, remittent, and continued fevers,	0·0	1·30	0·98	1·82	1·96	2·96	1·77	2·94	1·68	0·29
Mean temperature of year,	76°·7	78°·1	75°·8	78°·7	75°·7	76°·0	75°·3	69°·9	78°·9	82°·1
Mean temperature of May, June, July, August,	82°·2	89°·0	88°·1	89°·9	81°·9	91°·1	89°·7	86°·0	81°·9	86°·3
Difference between mean temperature of coldest and warmest months,	18°·4	32°·0	34°·3	34°·3	25°·2	40°·6	39°·6	39°·5	11°·5	11°·8

The high proportion of admissions for intermittent fever in Lahore and Peshâwar is no doubt largely the result of the high range of temperature producing relapses; but that the range of temperature is not the dominating influence at most of the stations, may be inferred from the greater prevalence of fever in Bombay with its equable climate, than in Mooltan with a range of 40°·6 F. Neither the mean annual temperature nor that of the summer months will explain the difference between Bombay and Madras as regards the prevalence and fatality of malaria. Facts such as these compel us to concede to other conditions than those of temperature a prepon-

derating influence in determining the distribution of fever in the different stations.

That a high temperature, however, does tend to increase the intensity of the infection when other circumstances favour its generation, is indicated by the greater prevalence in warm countries of those types of fever that are associated with the more severe forms of the infection. The types of longer interval, such as the quartan and tertian, indicate a milder form of infection than those of shorter intervals, such as the quotidian and double tertian. Now, as we advance towards the tropics, we observe that the quartan type tends gradually to disappear, and the tertian, which is the common form in Europe, is seen to take a secondary place to the quotidian, which is rarely met with in colder climates; while the severer forms of the remittent and continued types, only met with in the more intensely malarious localities in temperate climates, come into prominence in tropical countries. The relation of the different types of fever to climate will be illustrated by the following table:—

TABLE SHOWING THE PREVALENCE OF THE TYPES OF INTERMITTENT FEVER, ACCORDING TO LATITUDE.

	Number of Cases.	Quartan.	Tertian.	Double Tertian.	Quotidian.	Irregular.
Sweden, .	53,009	16·4	57·1	...	26·4	...
Vienna, .	3,126	7·8	47·8	...	41·3	3·0
Algeria, .	4,211	0·5	28·6	...	70·9	...
India, .	2,574	1·1	23·1	4·5	70·7	0·4

In the colder climates, malarial remittents are rare, and are only met with in the most unhealthy localities and seasons; in warm climates they are very common. The following is the proportion which the intermittent bears to the remittent and continued forms of fever in Algeria and India:—

Countries.	Intermittent.	Remittent.	Continued.
Algeria, ...	83·6	16·4	...
India (European Troops),	33·4	22·7	43·7[1]

[1] It should be noticed that in India continued fever is far from being entirely malarial. The figures are from Day.

The relation of the severe remittent and continued forms of fever to high temperature is further shown by the fact that in the Southern States of the Union they are only met with in the warmest summer months. Colin observes that in Rome these forms of fever are almost entirely restricted to the period when the temperature is highest, that is, in July and the first fortnight of August.[1] In India, in the same way, the remittent and continued forms generally attain their maximum in the warmest months of summer, while the intermittent type attains its maximum in the later months of the year, after the temperature has somewhat fallen. Taking malarial fevers as a class, including the intermittent as well as the remittent forms, they do not, as a rule, attain their maximum coincidently with the maximum temperature. In India, for example, the highest temperature is reached in June,—in some districts in May; while the maximum of fever prevalence is only attained in October, except in those regions which are under the influence of the winter monsoon, where the maximum is reached in January and February.

Nor does the minimum fever prevalence necessarily correspond with the minimum temperature. In Rome, for example, the admissions from fever are fewest in June, when the temperature is only 2·5° C. below the maximum. Borius, who studied the relation of fever to temperature in Senegal, lays it down as an axiom that the insalubrity is in proportion to the mean monthly temperature. A wider survey of the subject will show that no such simple rule regulates the seasonal evolution of malaria. The period of the year at which malaria attains its maximum is evidently determined by several factors of which temperature is only one.

The amplitude of the annual fever curve bears a pretty close relation to that of the temperature curve. Where the temperature of the different seasons is pretty uniform, there the number of fever cases in the different seasons is also nearly uniform. Where the difference between winter and summer is extreme, the seasonal variations in the prevalence of malaria will also be excessive. In countries, however, absolutely destitute of moisture, as in the Sahara, the temperature may be very high and the fluctuations sudden and great, and malaria may be quite unknown. In the Sahara, according to the Arab proverb, "He who is not reaped by the sword sees days without end"; or according to the saying of the Frenchman, "In the Sahara we have health, but must perish of thirst; in the oases we have water to repletion, but we must rot of fever."

Vicissitudes of Temperature.—The table which we have already given shows that in some of the most malarious stations in India,—

[1] *Traité des fièvres intermittentes*, Paris, 1870, p. 138.

Bombay, for example,—the annual range of temperature is by no means high, and at few of the stations does the period of greatest malarial prevalence correspond to the month when the daily range is highest. Further, there are countries, both in temperate and tropical regions, where the vicissitudes of temperature are very great, but where fever is unknown. This is sufficient to prove that the infection itself is not dependent upon changes of weather, as some have maintained. But it has been proved beyond all doubt that sudden falls of temperature cause relapses of fever in those who have already suffered from the infection. It was observed, for example, during the passage home of the troops who had suffered so severely from fever during the epidemic in Mauritius, that every sudden fall in the temperature was the signal for a large number of relapses; but if, after the temperature fell, it remained steadily low, the relapses did not increase.

Relation to Rainfall.—In some marshy countries a heavy rainfall diminishes the prevalence of fever, by covering the marshy ground with a protective sheet of water. Thus, in the Netherlands, it has often been remarked that rainy years are healthy years, while hot and dry seasons are feverish. In India and, indeed, in most tropical countries, fever is most prevalent in wet years; and this is especially the case in level coast plains, table-lands, and other localities in which the drainage is slow or obstructed.

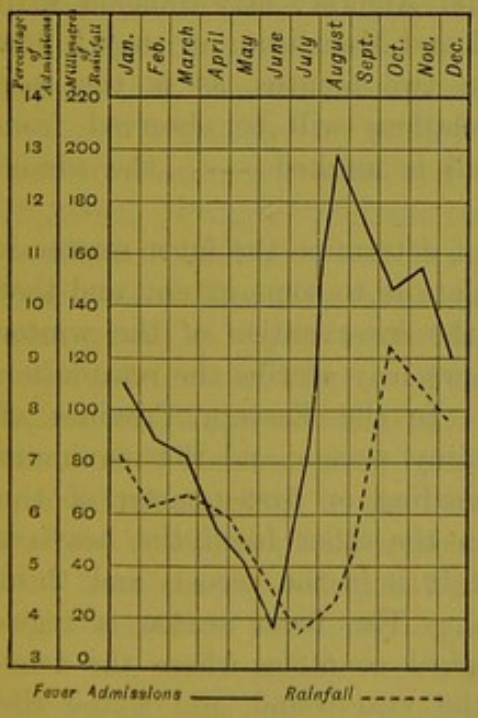

FIG. 5.—ROME.
Monthly percentage of fever admissions into St. Esprit Hospital in relation to rainfall.

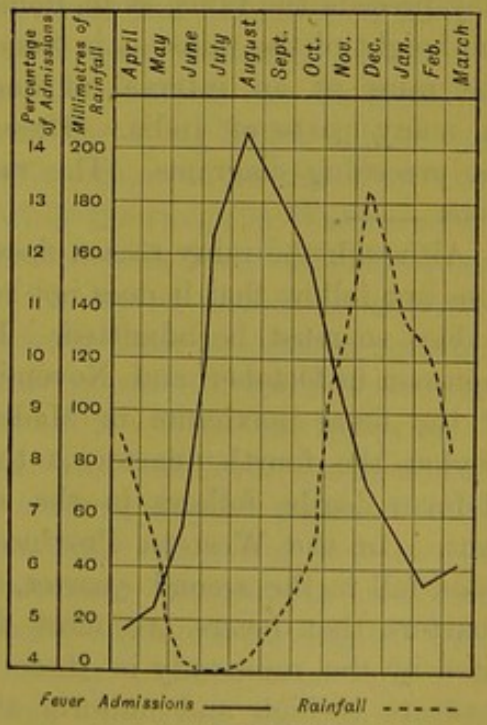

FIG. 6.—ALGERIA.
Monthly percentage of fever admissions into Military Hospitals in relation to rainfall.

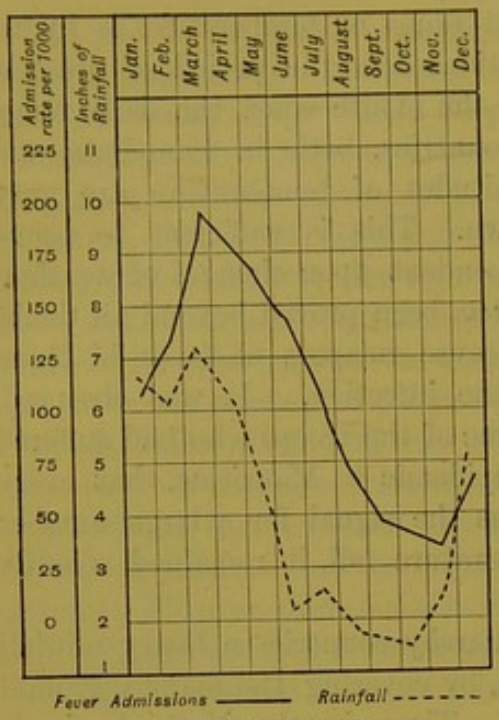

FIG. 7.—MAURITIUS.

Monthly fever admission rate per 1000 of strength into Military Hospitals in relation to rainfall.

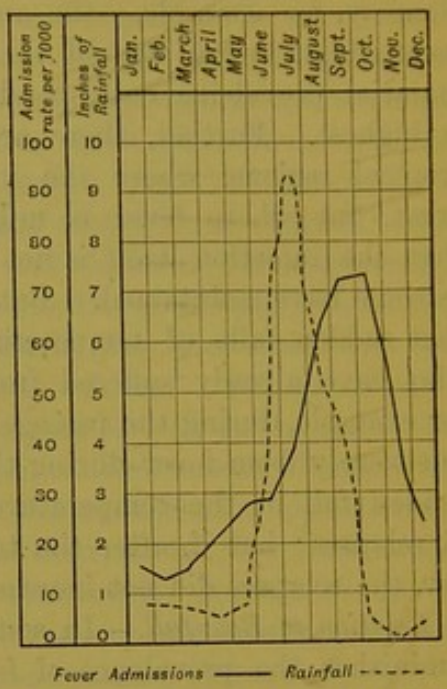

FIG. 8.—MEERUT AND ROHILKHAND.

Admission rate for intermittent fever per 1000 of strength in relation to rainfall.

The seasonal prevalence of fever is not determined, as many suppose, by the drying up of the rains. The maximum of fever prevalence, as judged by the admissions into the military hospitals, may precede the rains, as in Rome and Algeria; may coincide with the height of the rains, as in Senegal and Mauritius; or may follow the rains at an interval of one or two months, as is the case in many parts of India. These relations will be observed from the preceding diagrams. The rainfall is marked ------, the admissions ——.

Although the rainy season does not determine the fever season, it does not follow that it does not regulate it to some extent, and that it does so must be admitted. To the intervention of the winter monsoon in October and November we may ascribe the retardation of the fever maximum in Madras. In the Eastern Province of Ceylon the fourth quarter is the rainy season, and the maximum of fever deaths follows in the succeeding or first quarter of the year. In the Western Province, on the other hand, the heaviest rains fall in the second quarter, and it is in the second and third quarters that fevers are most fatal. The fever season is regulated by the rains only in those tropical countries where the temperature is high and uniform all the year round, and where the time of the rains may be said to determine the summer and winter. Where there is a distinct winter season, the period of greatest

fever prevalence is regulated more by the temperature than by the rainfall. The following diagrams show the relation of the fever season in Ceylon to the season of rainfall:—

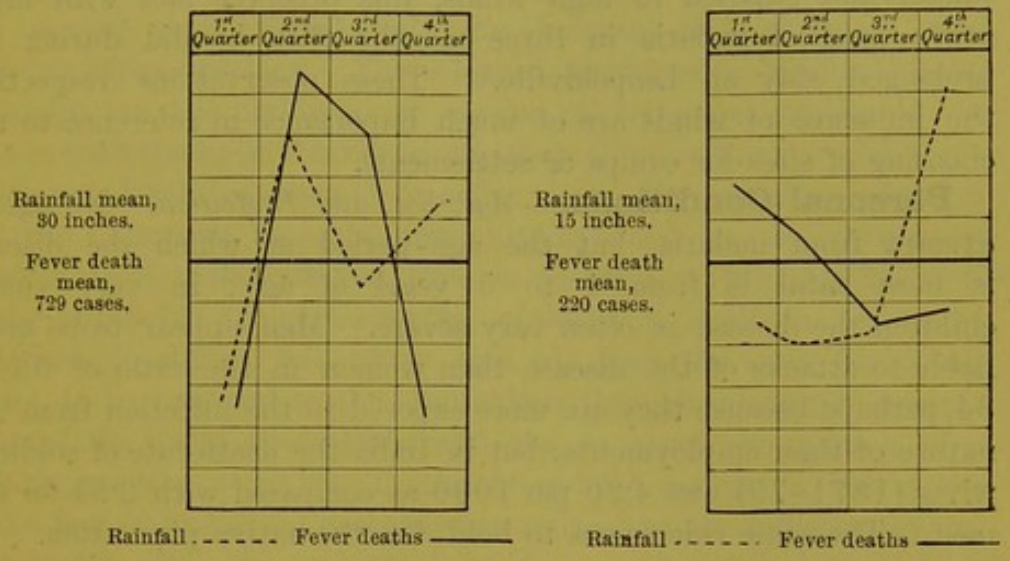

FIG. 9.—WESTERN PROVINCE. FIG. 10.—EASTERN PROVINCE.

Each horizontal thin line represents 5 inches of rainfall and fifty fever deaths.

Relation of Malaria to Winds.—It is believed by many observers that winds are capable of carrying the infection for considerable distances. Parkes suggests that malaria may drift up valleys to a great height; and he thus accounts for the existence of fever at high elevations in India.[1] We have good evidence, however, to believe that the distance to which the infection can be carried in this way is usually very limited. A vessel lying half a mile, or even less, off a malarious coast, is generally safe, even on the West Coast of Africa. Pringle[2] remarks that while the troops in Walcheren and South Beveland in 1747 were affected with fever and dysentery, Commodore Mitchell's squadron, which lay at anchor in a narrow channel about 6000 feet wide, between these two places, remained free from fever and flux; and the same experience was repeated later during the Walcheren Expedition of 1809. While the troops on the island of Walcheren were being decimated with fever, the men of the guardships stationed in the narrow channel enjoyed perfect health.

Cold winds, as we have seen, often cause relapses of fever in those who have already contracted the infection. Stanley, in his account of his travels in the Congo region, says: "While ascending the Congo with the wind astern we were usually exempt from ague; but descending the Upper Congo, facing the wind, we were smitten with the most severe form of it." Mense observed that at Leopold-

[1] Parkes, *Hygiene*, 7th ed. p. 626.

[2] Pringle, *Diseases of the Army*, p. 57.

ville, on the shores of Stanley Pool, the worst cases occurred in a house on the exposed summit of Mount Leopold (230 feet). At Vivi, which is described as situated in the neck of a mountain funnel and exposed to high winds, this observer met with more grave cases of malaria in three months than he did during his prolonged stay at Leopoldville.[1] These observations respecting the influence of winds are of much importance in reference to the choosing of sites for camps or settlements.

Personal Conditions.—*Age, Sex, and Profession.*—No age is exempt from malaria, but the age-period at which the disease is most fatal is from 1 to 5 years of age; in very young children the disease is often very severe. Men appear to be more liable to attacks of the disease than women in the ratio of 53 to 34, perhaps because they are more exposed to the infection from the nature of their employments; but in India the death-rate of soldiers' wives (1871–75) was 4·20 per 1000 as compared with 2·81 for the men. The same rule seems to hold for the native population. In Bombay (1885–86) the female fever death-rate was 10·14; that of males, 7·56. The cultivators of the soil, navvies, soldiers during active service, especially those belonging to the engineer corps, are very liable to contract malarial fever. The inhabitants of detached houses and small hamlets suffer more than those who dwell in large towns; and the more densely peopled and central districts of towns, if the streets are well paved, are less malarious than the suburbs, where the houses are more scattered.

Race.—The constitution of the negro is more tolerant of the infection than that of the Caucasian race. It is found that the West Indian native regiments stand the climate of the West Coast of Africa better than the European soldiers. Yet the negro is not exempt from the infection. Dr. Pieroz says: "Negroes suffer from malarial fevers quite as badly as any race I know. I assert this after an experience of twelve years in the West Indies. They suffer chiefly from intermittent fever."[2] The mortality from malarial fever among the negro population of the United States is greater than that of the white population, in the proportion of 48·3 to 30·7; this, however, is doubtless due to the difference in employment and social condition of the two races. The Niger Expedition of 1841–42 consisted of 145 white men, 133 Kroomen, natives of the coast and accustomed to the climate, and 25 blacks from the West Indies or the United States. Of the whites, 130 were attacked with fever and 40 died, none of the Kroomen suffered, while 11 out of the 25 foreign blacks sickened, but all recovered.

[1] "Rapport sur l'état sanit. de Leopoldville." Bruxelles. [2] *Brit. Med. Journ.* 1892.

Acclimatisation.—It is doubtful whether even a partial immunity from malaria can be attained by prolonged residence in a malarious country. While the liability of the European soldier in India to the remittent and continued forms decreases after the first and second years, attacks of ague become more frequent, and cachexia often develops. The coloured natives of malarious tropical countries are less liable to be carried off by acute attacks of the disease than strangers coming among them; but in the more intensely malarious localities they too suffer from the chronic infection. For the Caucasian race, the immunity attained by the permanent inhabitants of malarious countries is doubtful. This race has certainly succeeded in permanently establishing itself in extremely malarious localities; but, where this has been the case, the result is not to be ascribed to the elimination through successive generations of the more susceptible, and the eventual propagation of a malaria-resisting stock, as Tommasi-Crudeli[1] maintains, but rather to the fact of its having succeeded in mitigating the infection by improving the soil. In proportion as the race has failed in this, it has continued to exhibit its susceptibility in no doubtful way. Nepple informs us that the Dombiste, that is, the inhabitants of the extremely malarious region of La Dombes, between the Soâne and Ain, is recognised by his pale complexion, his drawn out, emaciated, or puffy features, his flabby muscles, by a certain indolence in his movements, his swollen belly, his weak intellect, his indifference to his condition, and his lack of moral energy. It is evident that in this case, at least, acclimatisation, after many generations, has remained imperfect.

Predisposing and exciting Causes.—The most powerful predisposing cause of an attack of fever is a previous attack. A body of men which has never suffered from malaria will remain healthy under conditions which will give rise to fever in those whose constitutions have been more or less broken down by attacks of fever contracted during the previous season. Pringle[2] observed, during the campaign of 1748 in Dutch Brabant, that the troops being encamped on dry ground generally remained healthy; but he adds, "From this good state of health the four battalions that had been in Zealand the last campaign were an exception, as being subject to relapse into irregular intermittents, frequently ending in dropsies." It is difficult to say in such cases whether we have to do with a new infection or simply with relapses of a previous infection. The distinction which the French draw between *rechutes*, or simple relapses of fever, and *récidives*, or the recurrence

[1] *Lancet*, Dec. 10, 1892.

[2] *Diseases of the Army*, p. 61.

of fever as the result of a new infection, is more philosophical than useful, for it is one of the worst features of the malarial infection that it is impossible to say when we have got rid of it. The disease may remain latent for months, even for years after the patient has removed to a healthy locality, and then manifest itself in a series of paroxysms upon the action of some exciting cause.

Every influence, physical or mental, that depresses the system appears to predispose to the disease; while fatigue, exposure to the sun or to cold, act as exciting causes of fever, and are specially effective in producing relapses.

Medium of Infection.—The use of the term malaria testifies to the general acceptance of the doctrine that air is the common medium of infection. That air is one medium of infection scarcely, indeed, admits of dispute. The fact that the men of the vessels of war, compelled to anchor in the malarious creeks of the African rivers and close to shore, contract the infection without making use of the drinking water of the localities, appears to prove beyond all doubt that it can be conveyed into the system by means of the air.[1] Another proof that air is a common medium of infection, is found in the prevalence of malaria in towns such as Rome, which have a pure water supply, derived from what are supposed to be non-marshy localities;[2] and also from its varying incidence on different districts of a town having the same water supply. The greater intensity of the infection in different seasons in the same locality while the water supply remains the same, and its extension in epidemic seasons over large areas of country without reference to the nature of the water supply, seem also to point to the air being a common medium of infection.

At the same time, there are a number of observations which seem to show that air is not the only means by which the infection can be carried. Indeed, the earliest view of the mode of infection was that which ascribed it to the use of marshy drinking water. Hippocrates says: "If there be rivers in the low-lying countries which carry off the stagnant and rain water from it, these may be wholesome and clear; but if there be no rivers, but the inhabitants drink of the water of fountains and such as are stagnant and marshy, they must of necessity have prominent bellies and enlarged spleens."[3]

Aretæus observes that diseases of the spleen "occur chiefly in autumn in marshy localities, and from the use of thick saltish and fœtid waters."

[1] See chap. iii. on *Tropical Naval Hygiene*, p. 108.
[2] Colin, *Traité des fièvres intermittentes*, Paris, p. 113.
[3] Airs, Waters, and Places, par. 24.

Many recent observations seem to show that these fathers of medicine were not mistaken in ascribing malarial disease to the use of marshy water. Parkes, on visiting the Plains of Troy during the Crimean War, found it to be a common belief of the villagers that those who drank pure water contracted fever only during the late summer and autumn months, while those who drank marsh water had fever all the year round. There are numerous instances on record in which the supply of pure water has been followed by the decrease of fever in villages which had previously been unhealthy.[1] In the *Army Medical Report*, vol. xvii., is a paper by Brigade-Surgeon Faught, illustrating the dependence of malaria upon the water supply. The soldiers stationed at Tilbury Fort generally suffer to a considerable extent from ague, their water supply being derived from tanks exposed to soakage from a neighbouring marsh. The people at the railway station and the coastguards, deriving their water supply from a spring, were free from sickness; and during the temporary repair of the tanks, while the troops in the fort made use of the water from the spring, no cases of ague occurred among them. Laveran relates an observation recorded by Pereyra of Bordeaux, and cited by Jacquot, to the effect that the inhabitants of the marshy districts of Landes in the department of the Gironde in France, who use the unfiltered marshy water of the locality, contract fever, while those who use the same water after being passed through carbon filters, escape. If this observation were confirmed it would be very strong proof, indeed, that water is one of the methods by which malaria effects its entrance into the economy.[2] There is thus good reason for believing that malaria may be conveyed into the system both by air and water.

Incubation. — The incubation period generally varies from seven to twenty-one days. Ten to fifteen is the period that has been more commonly observed. The instances on record, such as those recorded by Fonssagrives[3] and Lind,[4] in which the disease has appeared within a few hours of the supposed infection, cannot be entirely relied upon. When induced experimentally by the injection of malarious blood into healthy persons, it has appeared at periods varying from six to sixteen days.

Character and Conditions of Epidemic Development. —Malarial fever is observed from time to time to become so prevalent in its endemic haunts as to be justly regarded as epidemic.

[1] See paper by Mr. Bettington in *Indian Annals*, 1856.

[2] The reader is referred to an interesting article by Professor Notter on this subject in *Brit. Med. Journ.*, November 8, 1884.

[3] *Hygiène navale*, p. 490.

[4] *Essay*, 2nd ed., London, 1771, p. 23.

These local endemic exacerbations are generally to be traced to some appreciable meteorological or physical condition, such as an excessive rainfall, an inundation, or, in marshy countries, to excessive drought or heat. True malarial epidemies differ from these localised outbreaks by the fact that they are not restricted to localities where fever is endemic (although these usually suffer most severely in epidemic seasons), but extend widely over regions where the disease is not endemic, and where the conditions usually associated with malaria are not present. The disease, when epidemic, is also observed to be unusually severe and fatal to natives as well as strangers,[1] rising in many instances to the intensity of the worst forms of pestilence. In Mauritius, out of a population not exceeding 130,000 in the area affected, the deaths in 1867 were 31,920. Another remarkable feature of malaria in its epidemic manifestations is that it influences the prevalence of other diseases.

During the present century malaria has overrun the greater part of Europe on several occasions. We have first the epidemy of 1806–12; then that of 1823–27, which made itself felt over many parts of England and even in London; next come those of 1845–49 and 1855–60; and, lastly, that of 1866–72. Griesinger mentions, in addition to these, a considerable extension of the disease during the period of the first invasion of cholera, namely, 1830–35.

In Bengal we have records of more or less extensive epidemies of malaria in the following periods:—In 1807–9, reaching Southern India in 1809–11; in 1816, extending to Madras; in 1834, extending to Western Rajputana, Nazirabad, Neemuch, and Mhow; in 1843–44, extending to Scinde, causing a terrible mortality among the troops in Hyderabad in 1843, and at Sukkur in 1844. This last outbreak reappeared in the monsoons of 1845–46. Epidemies, more or less extensive, occurred in 1850–51, 1863, 1866–67, the last corresponding with the European epidemy, and with the first outbreak of malaria in Mauritius. The disease was again epidemic in 1869–70 and 1878–79; in the latter years malaria was also widely prevalent in Mauritius, Madagascar, Cyprus, and even in New England. In the United States similar epidemic extensions have been observed during this century. The most recent one gradually extended over Connecticut, Massachusetts, Rhode Island, Vermont, and New Hampshire. During its prevalence in Connecticut it is recorded that enteric fever, from causing 400 to 500 deaths a year, so decreased that at the height of the

[1] A malarious fever is reported to have been recently epidemic in the Transvaal, carrying off more than 10,000 of the native population. *Lancet*, May 27, 1893.

malarial epidemic in 1879 it caused only 159 deaths. Whatever may be the cause of these outbreaks they are seldom directly to be traced to meteorological causes. Sometimes they have coincided with years of great heat, such as that of 1826 in Europe, and that of 1809–11 in Southern India, but in other instances no such connection has been traced.

In years when malarial fever is epidemic the disease anticipates its usual period of maximum. In those parts of India where fever usually becomes very prevalent in September or October, in epidemic years it begins to show an unusual degree of prevalence as early as August, while in the hill districts, such as Simla, where the maximum fever mortality is usually attained in June, an excess of fever deaths will be observed as early as March in epidemic years. This anticipation of the fever maximum is a sure sign of coming danger, for when it shows itself early in the spring it is proof of an epidemic influence which will manifest itself destructively in the succeeding autumn. This law was clearly enunciated by Sydenham,[1] who says: "If fevers, continued or intermittent, appear unnaturally early, the season that follows will be exceedingly favourable to the development of epidemics,—by early, I mean on or before Midsummer day.[2]

Relation of Malaria to other Diseases. — Endemic malaria has been thought by some to be antagonistic to consumption, but this has not been yet satisfactorily established. Dysentery is occasionally endemic in the same localities as malaria, but this is the exception rather than the rule. It will be found that localities and regions where malaria is most intense are frequently remarkable for their freedom from dysentery. At Fort Gibson in the United States, fever is three times as prevalent as in Jefferson Barracks, but dysentery is much more common at the latter than the former post. Madras is much less liable to fever than Bengal or Bombay, but is more severely affected with dysentery. Béranger-Féraud has noted that in Algeria fever years are often years when dysentery is neither common nor grave.[3] No antagonism has been proved to exist between endemic malaria and typhoid fever. The case is different, however, in respect to epidemic malaria, which during its period of prevalence causes typhoid fever temporarily to diminish in fatality, to reappear again when the epidemic has passed. According to Bryden, "malarial

[1] Sydenham Soc. tranlation, vol. i. p. 271.

[2] For the statistical proof of this law in its application to India, the reader is referred to *Geographical Pathology*, vol. i. pp. 340, 352 *et passim*. The evidence bearing on the relation of phthisis to malaria will be found detailed in vol. ii. in the chapter on the United States.

[3] *Traité Théorique et Clinique de la dysenterie*. Paris, 1883.

and cholera epidemics run a parallel course as regards season, geographical distribution, and in the succession in which provincial areas are covered by them." It has been observed in some cases that epidemies of malarial fever have preceded those of cholera, and have ceased suddenly when cholera has declared itself. In districts where malaria had been present before the advent of cholera, it has sometimes reappeared after the cholera epidemy has passed, or has remained absent for years from districts in which it had been formerly endemic. The relations between cholera and malaria are too manifest to be doubted, but too complex to admit of being reduced, at present, to any definite law. It is recorded, for example, that intermittent fever disappeared entirely from Marienwerder (where it was previously endemic) on the cessation of cholera in 1831, and only reappeared with the return of cholera in 1849; but it did not then disappear with the cholera, as in 1831, but remained the predominating sickness in that locality until 1856, when it again diminished in frequency. Epidemic malaria and epidemic dysentery have frequently been associated together. It rather frequently happens that towards the end of a malarial epidemy the fever becomes complicated with dysentery.

Climatic and Non-Malarial Fevers of Warm Climates.

The dominating position occupied by malaria in tropical pyretology has had the effect of diverting attention from the study of the non-malarial fevers met with in these countries. Malaria has so completely filled up the whole field of vision, that it has shut out from view, as it were, the existence of other forms of fever. It is only within the past thirty years that the existence of enteric fever, as a disease distinct from malaria, has been fully recognised in India. Even at the present day the claim of tropical typhoid to be looked upon as a miasmatic disease is scarcely allowed; for although its existence and importance are now admitted, it is still believed by many to be transmissible only by a germ derived from the intestines of a previously affected person. Relapsing fever, formerly looked upon as malarial, has only in recent years had its own place assigned to it in tropical nosology. During the past few years another febrile disease, now known as Malta fever, has been differentiated from the malarial group, and will probably be found to have a wider distribution than is at present imagined. But we are not to suppose that the process of differentiation has been completed. It is probable that a more careful study of tropical fevers will lead to the recognition of other non-malarial febrile diseases besides those with

which we are at present acquainted. It will only be when we have succeeded in eliminating all these extraneous maladies from the malarial group, that we shall be able for the first time to obtain a clear, unblurred, view of the clinical features of malarial fever. In the meantime, it may not be out of place to glance briefly at some of the, as yet, undefined and imperfectly understood febrile diseases met with in tropical countries, which, in fact, are often looked upon as malarious.

An examination of the Army Medical Reports for any year will show that, in all temperate and non-malarious countries occupied by the British army, a class of continued fevers is recognised,—distinct from ague, enteric fever, typhus, and cerebro-spinal fever. These fevers are also observed to occur among the troops on board ship during their outward passage, which is a strong proof that they cannot be of malarial origin.

This composite class of non-malarial fevers has not been thoroughly analysed, but two groups of febrile disorders can be distinguished as included in it by their clinical and etiological characters. The first group consists of fevers in which the febrile phenomena constitute the most prominent features of the disease. The fevers to which we refer are most common in the summer and early autumn months, and are frequently, although not constantly, observed to follow exposure to the sun's rays, sudden suppression of perspiration, or a chill after exhausting fatigue. They are characterised by their sudden onset, the high temperature of their fastigium, the prominence of vascular and nervous phenomena, and often, although not uniformly, by their rapid defervescence. Lasting for less than two days, they are spoken of as *ephemera;* and when running a course of from three to seven days, as *common continued fever* or *febricula.* Whether the fever or group of fevers comprehended under the names of ephemera and febricula, to which we refer, are due solely to climatic causes, or whether they are not rather to be looked upon as miasmatic diseases, in the development of which meteorological conditions play the part of predisposing or exciting causes, is a point which need not at present be discussed. What it is important to bear in mind is that, whether miasmatic or climatic, they are not malarious.

To the second group of this heterogeneous class of continued fevers are referred diseases in which the purely febrile phenomena are less obtrusive, while gastro-intestinal derangements predominate. These run a course of from one to two weeks, and sometimes assume an irregular remittent type. In their more pronounced forms they are often spoken of as *gastric fever;* and from their tendency to

develop simultaneously with typhoid fever, are, not without reason, suspected of being abortive forms of this disease.[1] It may afterwards be shown that among the fevers included in this group are mild cases of typhoid, and other fevers arising from distinct miasmata.

It would appear improbable *à priori* that these two groups of febrile maladies, which along, perhaps, with others are included under the term common continued fever, should disappear from the pathology of tropical regions. As a matter of fact, they are observed to become more common in the warmer than in the colder non-malarious countries, and according to the Army Medical Reports they are more prevalent in tropical malarious countries than in temperate climates. In the United Kingdom the admissions from this class of fevers in 1883 were 4·4 per 1000; in Malta (Native Regiment), 66·1 per 1000; in Bermuda, 33·4 per 1000. These are non-malarious countries. In Bengal the ratio was 83·0; in Madras, 59·3; in Bombay, 91·0; and in Ceylon, 114·7. In tropical countries these non-malarious fevers are more common among the Europeans than the natives, and amongst those who have recently arrived than those who have become acclimatised.

Ardent Fever.—The first group, comprehending the febricular as distinguished from the gastro-intestinal febrile disorder, are most frequent in dry and arid regions during the warmest months of summer, and they attack by preference those who have been working or marching in the sun, and especially the intemperate.

The simple ephemera and febricula of temperate climates are frequently met with sporadically in warm countries,—when they are often looked upon as attacks of malarial fever, and treated with quinine,—which, although unnecessary, does no harm.

Dr. Arnott described an epidemy of febricula, or ardent fever, as he called it, which casts some light upon the etiology of this affection. The disease is stated to have prevailed among the Bombay Fusiliers at Pesháwar in the months of June, July, and August, when the men were under tents, the temperature ranging from 70° to 114° F., with hot blasts, and thick suffocating clouds of dust,—a temperature oppressive by day and preventing repose by night. The number attacked was 884, without a single death. The symptoms were simply those of febricula,—pungent heat of skin, thirst; red, dry tongue; quick, fast, and strong pulse, severe head-

[1] An epidemy of gastro-intestinal catarrh, with fever, was observed by Dr. Lowe, of Lincoln, at the end of a severe frost in the winter of 1890, when, it appears, cases of a similar kind were very prevalent in London. The symptoms were epigastric pain, anorexia, vomiting, diarrhœa, motions of a whitish-green and yellow colour, tenesmus, furred tongue, a yellowish tinge of conjunctivæ, great nervous prostration. The temperature was seldom above 102° F.—*Internat. Journ. of Med. Sciences*, November 1891.

ache, flushed countenance, restlessness, nausea, and bilious vomiting. These are, in fact, the symptoms proper to what is called "ardent fever" in its milder form; and this epidemy is quoted by Morehead as one of ardent fever. In individual cases of ardent fever, as met with in tropical countries, the cerebral or the gastro-hepatic symptoms are occasionally much more marked. There may be giddiness, sleeplessness, and delirium, terminating in coma; or, on the other hand, bilious vomiting, bilious diarrhœa, and jaundice, with a tendency to death from exhaustion. It will, however, generally be found that when ardent fever becomes complicated with dangerous head symptoms, that insolation has played a leading part in its causation, or that the disease is malarious in its origin. Intense bilious symptoms should also raise a suspicion of malaria.

Epidemies of mild ardent fever, although not common, are occasionally observed among bodies of men subjected, like those at Pesháwar, for a time to the action of a high temperature. In 1878, H.M. ship *Undaunted* had been moored for three months in the harbour of Aden. During the latter part of this time the men complained of an indisposition to eat. They cared for nothing but fluids, and suffered from sleeplessness. There was also a general indisposition to go ashore. At the end of May, 125 cases of continued fever occurred, the symptoms of which were headache, vertigo, injection of the conjunctivæ, dry, hot skin, anorexia, watchfulness, furred tongue, constipation, and languor. The pulse varied from 90 to 110, and the temperature from 101° to 103° F. The average time spent by each person on the sick list was between seven and eight days. It did not relapse, as malarial fever is wont to do. Sporadic cases of this fever are of frequent occurrence among the men of the ships visiting this intensely hot, but not very malarious port. The same type of fever is also frequently observed in the French settlement of Obok on the African side of the Gulf of Aden —on the shores of the Strait of Bab-el-mandeb. Dr. Esclangon, who has described it, believes that it is neither typhoid nor malarious, but climatic in its origin. Its onset is sudden, as a rule, without rigors, skin dry and burning, great pain in back and loins, frontal headache, and is generally accompanied by constipation. In many instances the disease is ephemeral; in cases of medium gravity it lasts from six to nine days, while in its gravest form it may last from twenty to thirty days. Even in a distinctly malarious country, such as Muscat, Peters observed a fever having the symptoms we have described, which was aggravated by quinine, but yielded to purgatives and emetics.

It may, indeed, be difficult to explain satisfactorily in what way

a high temperature gives rise to a febrile explosion, which in most cases passes off within a few days, and while the body is still under the influence of the assumed cause. Many who admit most fully the influence of climatic conditions in modifying the course and in determining the character and type of a fever, refuse to concede to these conditions the power of producing fever. Laveran says that the question of climatic fevers really resolves itself into this: Can external heat give rise to fever? and to this question he replies in the negative. "Il ne suffit pas," he says, "de placer un homme dans une étuve chauffée à 40° on 50° pour lui donner la fievre;"[1] and he contends that, in Algeria, no direct relation can be traced between the frequency of fever and the elevation of the temperature. But it must be admitted that the conditions of the experiment supposed by Laveran, viz. that of placing a man in an oven heated to 40° C. (104° F.), are fairly realised in the case of men confined in a vessel in the harbour of Aden, and also in the circumstances in which the troops at Pesháwar were placed as described by Arnott; yet, as a matter of fact, fever, as we have seen, was produced in these cases. The conditions of life, too, at Obok and Muscat are not very widely different from life in a moderately heated oven, except in one particular, viz. that the temperature of the natural oven, although high, is not constant; and the variations, although they may not be great, are sufficient, in the relaxed state of the system produced by the heat, to determine internal congestions that may very well be supposed to give rise to fever. Apart, however, from all theory, the influence of a high temperature in the pathogeny of the fevers we have described is too obvious to admit of discussion.

Whether we look upon these fevers as climatic or miasmatic, it is of importance to bear in mind the existence of non-malarious ardent fevers in tropical countries. They do not disappear from a region simply because it is malarious; and the same conditions which give rise to ardent fevers may also determine a malarious attack, and materially modify its course.

Gastro-Intestinal Fevers.—The examples we have given above are of diseases related to the febricular group of continued fevers, such as are met with even in temperate climates; but examples of the gastro-intestinal group, to which we have referred, are equally common in warm countries, and when the accompanying fever is of a remittent type they are liable to be mistaken for malarial remittent. Many of these obscure or undefined fevers are probably allied etiologically to enteric fever, yet there are good reasons for suspecting that fevers are included in this group which

[1] *Traité des fièvres palustres*, Paris, 1884, p. 422.

are neither malarious nor enteric. Dr. Joubert, of Calcutta, has observed there a continued fever, which he believes is distinct from enteric, lasting exactly twenty-one days, in which quinine has absolutely no effect, and which for this reason may be assumed not to be of malarial origin. "There is very rarely, if ever, delirium; never the formation of sordes on teeth or tongue; no decided abdominal pain; gurgling may be detected if there be looseness of the bowels—a symptom not often present; there are no rose spots, nor that great loss of muscular strength and that marked debility so common after even mild attacks of true enteric fever."[1] It is extremely important that the nature of fevers presenting features differing from those belonging to the malarial, typhoid, and so-called climatic groups should be carefully studied.

Thermic Fever.—Fayrer has described under the name of *thermic fever* those fatal cases of heat-stroke beginning with coma, or headache followed by coma, hyperpyrexia (temperature, 107° to 108° F.), and often ending in coma within five or six hours from the commencement of the seizure. These cases may sometimes be mistaken for comatose pernicious fevers. When, as is sometimes the case, fever only appears some days after exposure to the sun, and then runs an irregularly remittent course, it is apt to be mistaken for malarial fever, especially when it occurs in a malarial country. The history of exposure to the sun; the early and distressing sleeplessness followed by delirium, are the points that help to clear up the diagnosis.

Fevers with Cerebral Symptoms.—Fevers are not unfrequently met with in warm climates that cannot be classified under any of the recognised forms. I refer to cases running a course of seven to twenty-one days, of a remittent or continued type, in which cerebral symptoms predominate from the beginning, or at least become marked during its progress, without any history of insolation, and in which after death the spleen, liver, and abdominal organs generally are found healthy; the only constant lesions being those indicating minor degrees of meningeal inflammation,—opacity of the arachnoid, and effusion of a fluid, sometimes lymphy, into the meshes of the pia mater. To what specific poison or infection are we to ascribe these fevers? Perhaps in some cases the fever is to be looked upon as symptomatic rather than primitive.

To illustrate this class of fevers, I may be permitted to refer to two cases recorded by Morehead,[2] which he regards as instances of

[1] Fayrer, *Fevers of India*, p. 245.

[2] *Clinical Researches on Disease in India*, 2nd ed., Lond. 1868, p. 87.

inflammation of the membranes or substance of the brain, occurring in the course of remittent fever. The first case is summarised as "Remittent fever, with meningitis, shown by effusion of serum in the cavity of the arachnoid and sub-arachnoid space, and by opacity and thickening of the arachnoid membrane." The patient, a boy aged 7, was admitted into the sick ward of the Byculla Schools on the 6th of June 1838. He was affected with febrile symptoms which did not attract much attention till the 10th, when he was observed to suffer from fever and drowsiness, which after three days was succeeded by screaming, moaning, and strabismus, with dilated pupils, terminating in coma on the seventh day after admission. The viscera of the thorax and abdomen, we are told, were healthy. Perhaps in this instance the fever was symptomatic of meningitis. The healthy condition of the liver and spleen negatives the view that it was malarial remittent. The second case is thus summarised, "Remittent fever admitted after a week's illness; head symptoms chiefly marked by unsteadiness of manner, and latterly by drowsiness. Arachnoid membrane opaque and thickened — increased serous effusion." The patient, aged 16, had suffered for a week from headache and fever, was admitted into hospital on the 9th May 1842. The skin was hot, the face flushed, and the manner undecided. After two days the tongue was noticed to be tremulous, and the bowels to be relaxed. On the following day, slight subsultus was observed. On the morning of the 14th (that is, about the twelfth day of the fever) there was a distinct remission. On the 15th and 16th the febrile exacerbation seemed somewhat checked under the use of quinine; but on the 17th all the symptoms were again aggravated—vomiting, watery evacuations, sunken features, and drowsiness closed the scene. The liver is stated to have been healthy; and as the condition of the spleen is not mentioned, it may be inferred that it presented no abnormal appearance. I have met with a considerable number of instances, somewhat similar to the above, in which severe brain symptoms arose during the course of a fever; in which there was no history of exposure to solar heat, no tubercular inflammation of the membranes or disease of the ear, in which after death no evidence of malaria was discovered. The etiology of these fevers is obscure.

Bilious Remittent Fever.—Much diversity of opinion has prevailed, and still prevails, respecting the nature of the bilious remittent fever of the West Indies. Some regard it as malarial, others as climatic, while many look upon it as an abortive or mild form of yellow fever. A few words on its symptoms may not be out of place.

The mild form of bilious fever is marked by a sudden rise of temperature, which continues high from twenty-four to forty-eight hours, then falls by steps of 1°·5 C. until the normal is reached. The fever is accompanied by slight gastric catarrh, nausea, icterus, and sometimes by bilious vomiting. There may be, in addition, either bilious diarrhœa or constipation. It shows no tendency to relapse.

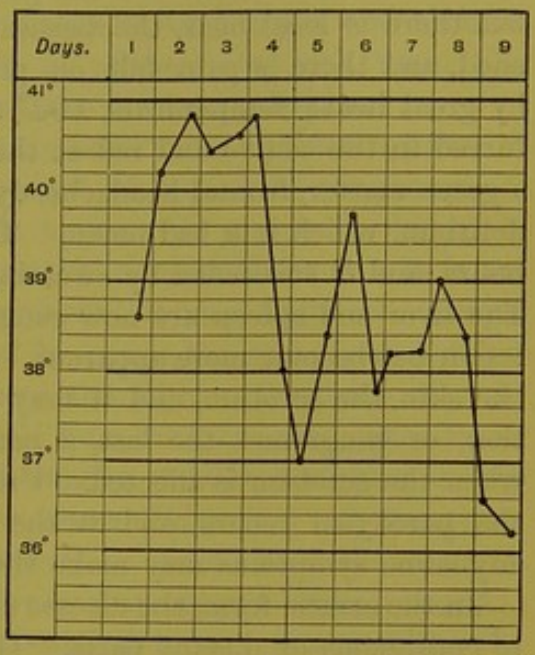

FIG. 11.—Bilious Remittent (Grave).

The grave form runs the course indicated in the accompanying chart (Fig. 11). It is accompanied with great epigastric oppression, nausea, bilious vomiting, and by jaundice appearing about the third day. It is associated in some instances with subconjunctival ecchymoses or petechiæ, and not unfrequently tends to end in collapse. It is characterised by relapses which give rise to anæmia, and often terminates in an obstinate intermittent. The mild form attacks by preference new-comers; the grave form attacks natives as well as Europeans who have been acclimatised; and is met with in marshy localities. Although, so far as I am aware, its malarial character has not as yet been demonstrated by the discovery in the blood of those suffering from it of the malarial parasite, or of black pigment, its symptoms complications, and habitats incline us to regard it as malarial.

Pernicious Lymphangitis.—A pernicious lymphangitis is endemic in Brazil, and is also met with in Réunion, Mauritius, and some other tropical countries, which is believed by many to be a special form of malarial fever. It is certainly often associated with malaria, both as being more prevalent in seasons when malarial fever is epidemic, and as being restricted to malarious countries. Two forms have been described by Dr. Carlos Claudio da Silva in a paper which appeared in the *Archives de médecine navale* (vols. xxxiii.–xxxiv.). The first form, local or circumscribed; the second, erratic or non-circumscribed.

The first form begins without prodromes, with rigors lasting for a few minutes only, or for two or three hours, with a temperature about 104° F., pulse strong, vibrating, and frequent (120 to 130). Generally it is during the rigor that pain or tenderness in the part

that is to be the seat of the lymphangitis is first felt. The glands are swollen, and when the parts affected are superficial, there is redness and local heat. If the glands are deep there is no redness, but there is local heat, the tissues over the affected part are thickened, and there is pain felt on pressure. The rigors are followed by great heat; temperature 104° to 106° F., pulse frequent, tongue furred in the centre and red at the tip, thirst, vomiting of food or of a green matter, frontal headache, dyspnœa, and often there is tranquil delirium, which is a bad omen. This period lasts from four to six hours, and is succeeded by sweating and a remission or intermission. The liver and spleen are now congested and enlarged, and the local lesion has become more apparent. The disease may affect the glands, the skin, the scrotum, and it may terminate by resolution, suppuration, or gangrene—the last, a rather frequent mode of termination when the scrotum is affected. Unless stopped by suitable treatment, the paroxysm recurs, and at the second or third accession, ataxo-adynamic symptoms may make their appearance and prove mortal.

In the erratic form, the disease extends from the point first affected to contiguous or distant parts. Occasionally the local tumefaction appears at some point—say, of the extremities or the mamma, before the outset of the fever. The general symptoms of the paroxysm and the modes of termination are the same as in the circumscribed variety, but the danger is greater. The second paroxysm is not unfrequently accompanied by agitation, hiccup, vomiting, diarrhœa, ending in prostration. Such is a very condensed account of the disease as seen in Brazil. My first practical knowledge of this (or a similar) malady has left a deep impression on my mind. It was in the case of a student who was attending the Civil Hospital in Mauritius. On making my morning visit, he informed me that he was suffering from fever and a swollen gland in the groin. The fever, he told me, was distinctly of the intermittent type, with a cold, hot, and sweating stage. In the right groin there was a swelling, looking not unlike a femoral hernia. The tumour, although slightly tender, was not red, and neither hard nor nodulated, but soft and compressible. Ignorant of the danger which might befall the patient, I recommended him to go to the country for a few days, to a relative who was a medical man. Within two days he was seized with sudden collapse and died. Fayrer has recorded a case very similar to this. The patient, a young man, aged 28 years, was reported to be suffering from a swelling in the groin suspected to be hernia. He was feverish, with pulse at 104° F., having had severe rigors previously. He had great thirst. Bowels acted, voiding greenish matter. The fever passed off next morning. At 2 P.M. next

day alarming symptoms set in; at 4 P.M. his breathing was very hurried, and his body was covered with cold sweat, with a pulse rapid and feeble. He died within half an hour.[1] While acknowledging the close connection between this form of lymphangitis and malaria, I hesitate, until further evidence is adduced, to look upon this disease as a simple manifestation of the malarial infection.

Typho-Malarial Fever.—One other form of fever requires to be mentioned in this place, on account of the great frequency, we may say constancy, with which it appears in camps in malarious countries; this is what is known as typho-malarial fever. It is the true camp-fever of malarious countries. It may begin as a simple remittent or intermittent, preceded by rigors, the symptoms peculiar to typhoid declaring themselves from the fifth to the tenth day; or the typhoid element may show itself from the beginning; the disease commencing without rigors, with headache, giddiness, singing in the ears, and signs of bronchitis. Its course and duration vary greatly in different cases, according as the typhoid or the malarial element predominates. In some cases the typhoid symptoms become accentuated, and death occurs from the twelfth to the fifteenth day by coma, or at a later period by perforation of the bowel and peritonitis It frequently runs a very protracted course, the temperature curve manifesting distinct tertian exacerbations. The following chart

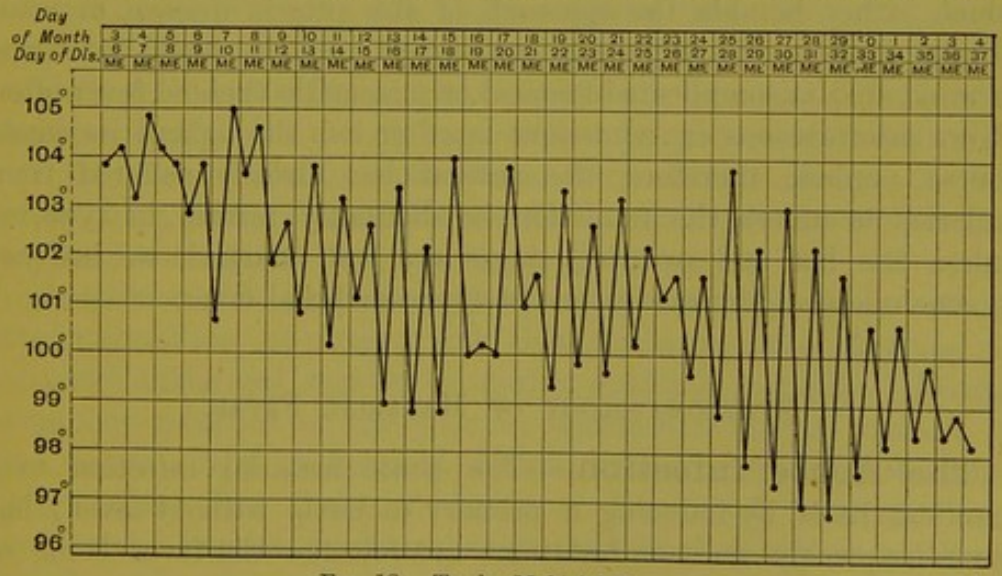

FIG. 12.—Typho-Malarial Fever.

gives the temperature from the sixth to the thirty-seventh day in a case that ran on to the sixty-second day—the third twenty-one-day period. The fever began as a remittent without rigors; but for the first two days the patient, a boy aged 7, perspired freely; after that the skin was dry. From the eleventh day the patient's

[1] *Op. cit.* p. 142.

condition was one of prostration, agitation, and delirium, moving his hands as if in quest of something. From the beginning there was a tendency to bronchitis and diarrhœa, both of which symptoms became more marked as the case proceeded. On the forty-seventh day the temperature began to rise, pleuro-pneumonia declared itself, along with periostitis of the right tibia. After the sixty-second day the temperature fell to and remained normal, and the patient recovered. Here the typhoid element predominated, although the tertian exacerbations indicate the malarial impress. After death in these cases Peyer's patches will show the signs characteristic of enteric fever, while the presence of pigmented bodies in the spleen and liver will testify to the presence of the malarial infection. It would lead us too far to enter into any discussion as to the nature of this disease. I may simply state that I have never seen sufficient reason to justify me in accepting the doctrine of a hybrid germ. I do not use the term typho-malarial in this sense, but simply as a convenient name for a disease in which the typhoid and malarial infections coexist and run their course side by side, the one modifying more or less the course of the other. When the typhoid infection attacks a patient who is suffering from chronic malaria, the disturbance of the system, caused by the development of the typhoid germ, appears to bring about a relapse of malarial fever, which either heralds the approach of the enteric disease, or complicates it more or less during its course.[1] But it should be borne in mind that uncomplicated tropical or miasmatic typhoid fever often shows intermissions or remissions more or less throughout its whole course; unless, therefore, the patient has already suffered from malaria, or unless the remission be distinctly tertian in type, or unless the hot and sweating stages are well marked, we are not to conclude that the case is one of typho-malarial fever.

Symptomatology of Malarial Fever.

The Acute Infection.—The acute malarial infection may manifest itself by inducing a primary cachexia without fever; but more frequently its manifestations are febrile—the fever being of the intermittent or remittent form.

Intermittent fever presents various types, and is either benign or pernicious in its character. The remittent form may be mild or grave. Mild remittent fever is distinguished, according to its

[1] See a case in which a tertian ague contracted some years previously in India, but which had quite disappeared for four years, reappeared during the third week of an attack of typical enteric caught in England. *Lancet*, April 29, 1893.

symptoms, into the simple, gastric, and hepatic varieties. The grave form is marked by the occurrence of delirium and coma, or by the appearance of algid, adynamic, typhoid, or hæmoglobinuric symptoms. Both the intermittent and the remittent forms may become complicated by intercurrent diseases, such as pneumonia and dysentery.

Primary Apyrexial Malarial Cachexia.—Persons residing in a malarious locality are sometimes observed to become anæmic and weak; to lose flesh; to suffer from pains in the back and limbs, from enlargement of the spleen, from intermittent neuralgias, or anasarca, without experiencing febrile attacks. If the patient continues subject to the malarious influence, organic changes are apt sooner or later to develop, and he becomes liable, like those subject to the chronic cachexia, to suffer at a later stage from irregular attacks of fever. When he returns to a healthy climate before his constitution has been impaired, perfect recovery is the usual result; but it occasionally happens that a chill, or exposure to the sun, or some other exciting cause, determines, for the first time, a severe febrile paroxysm, or in some cases a fatal pernicious attack.

This form of the infection has been noticed by several writers on tropical diseases, but its importance has scarcely been recognised. Morehead observes that "enlargement of the spleen and concomitant cachexia may take place from the influence of malaria, *without the intervention of fever;*"[1] and Colin remarks that "in certain cases the cachexia develops without any previous febrile manifestation."[2]

Those who suffer from this cachexia are constantly under the imminence of all the malarial accidents, especially pernicious attacks, and they are liable to suffer from those serious changes in the spleen, liver, and kidneys that we shall afterwards describe as the results of the chronic cachexia.

Sometimes this primary cachexia proves rapidly fatal without the intervention of fever; but this result is only observed in intensely malarious regions or during epidemic periods. Livingstone has recorded an instance in which he observed malaria to give rise to a fatal cachexia in the shipwrecked crew of a Hamburg vessel on the East Coast of Africa. "The men," he says, "were much more regular in their habits than English sailors, so that I had the opportunity of observing the fever acting as a slow poison. They felt 'out of sorts' only; but gradually they became pale, bloodless, and emaciated, then weaker and weaker, till at last they sank more like oxen bitten by the tsetse than any disease I ever saw."[3]

[1] Morehead, *Clinical Researches on Disease in India*, 2nd ed., London, p. 39.
[2] Colin, *Traité des fièvres intermittentes*, Paris, p. 295.
[3] Livingstone, *Missionary Researches*. London, 1857.

Referring to the epidemic fever which ravaged the district of Macacú in the province of Rio de Janeiro in 1829, Sigaud says: "During this epidemy the malarial intoxication gave rise to slow maladies instead of acute fevers; it caused a great number of cachexias, which have been designated by some authors anémie intertropicale or hypoémie. . . . This anæmia is the result of the toxic action of the malarial element; its action is slow, but equally fatal as that which produces algid fevers and pernicious pyrexias."[1] The condition of the blood in these cases has not been examined, but this form of cachexia will probably be found to be associated with the presence in the blood of crescents. Bignami has seen crescents accumulate in the blood and give rise to a fatal cachexia. "Queste forme vanno gradatamente, qualche volta rapidamente accumulandosi nel sangue sino a formare accumuli enormi, determinando una cachessia mortale."[2]

Benign Intermittent Fever or Ague.—The common types of ague are the quotidian, the tertian, and the quartan. The quotidian is characterised by similiar paroxysms occurring after an interval of twenty-four hours; the tertian, by similar paroxysms occurring after an interval of forty-eight hours; and the quartan, by similar paroxysms occurring after an interval of seventy-two hours. The quotidian may be double, two paroxysms occurring daily. The tertian may return every day, the paroxysms on alternate days being similar in respect to hour of onset, severity, and duration; this is called double tertian, or *tertiana duplex*, a type often seen in tropical countries, and often mistaken for quotidian. When the intermission is not complete, we have a remittent of the double tertian type, or, as some prefer to call it, double tertian intermittent with sub-intrant paroxysms—the succeeding paroxysm commencing before the previous one has subsided. Another type of tertian is that in which the fever returns every second day as in simple tertian, but with two paroxysms on the same day. This is distinguished as doubled tertian, or *tertiana duplicata*. The quartan also presents complex forms. It may occur daily, the paroxysms of every fourth day being alike. This is a triple quartan, or *quartana triplex*. Of the four days the third only may be free from fever, the paroxysms corresponding every fourth day. This is the *quartana duplex* of nosologists. When two or three paroxysms occur every fourth day, we have a *quartana duplicata* or *triplicata*, as the case may be.

The period which elapses between the beginning of one paroxysm and the commencement of the next is called the *interval;* the

[1] Sigaud, *Du climat et des malad. du Brézil.* Paris, 1844.
[2] *Ricerche Sull' Anatom. Patolog. delle Perniciose.* Roma, 1890.

intermission is the period between the end of one paroxysm and the beginning of the next.

In addition to these, which are the common types of fever, quintan, sextan, septan, octan, and nonan types have been described, and even bimensual and mensual are said to be met with. The quintan, septan, and nonan are to be looked upon as modified forms of the tertian—one, two, or three paroxysms, as the case may be, being so slight as to escape observation, or entirely suppressed. The quintan and septan are by no means rare. Borius states that the septan type is common in Senegal, and Graves that it is the prevailing and fatal fever of the government of Ufa in Russia. Fevers of irregular intervals are also of frequent occurrence in the chronic form of the infection.

Fevers may change their type either spontaneously or as the result of treatment. Frequently we find a remittent fever assume a quotidian or a double tertian type before becoming converted into tertian; much more rarely, in warm climates, does the tertian undergo the further change into a quartan. Graves records a case in which a double quotidian, during the process of cure, first became converted into a single quotidian, then into a tertian, and finally into a quartan. The reverse process, that of a quartan becoming changed first into a tertian, and then into a quotidian, is certainly extremely rare. In most cases where we hear of a quotidian being converted into a tertian, or *vice versâ*, it is not a true quotidian that is in question, but a double tertian. It is, in fact, a process of evolution or involution that takes place. The change of a quotidian into a tertian, or the reverse, is often sudden; but it also results from a retardation or anticipation of the paroxysm—that is, from the paroxysm coming later and later, or earlier and earlier in the day at each recurrence. Anticipating fevers often turn out troublesome or dangerous.

In temperate climates the paroxysm of quotidian fever begins in the morning, that of the tertian at or before noon, and that of the quartan at noon or in the afternoon. These rules apparently hold good for Algeria and the warm temperate regions, but they do not apply to India. Of 168 quotidians observed by Day at Mysore, the largest number occurred at noon. Only 37 out of the number occurred from 1 A.M. to 11 A.M., whereas no fewer than 108 took place between 11 A.M. and 6 P.M.; the remaining 23 happened between 6 P.M. and midnight. The quotidian thus appears in warm countries at the hours proper to tertians or quartans. Of 24 cases of tertian, 6 occurred about noon, 9 before noon, and 6 after noon, and 3 between 10 and 12 o'clock P.M. Of 7 cases of the quartan type, the paroxysm in 6 occurred from 4 to 6 o'clock P.M.

The hour at which the quotidian paroxysm takes place confirms the view that, in many cases at least, the so-called quotidian is really a double tertian. In temperate climates the average duration of the paroxysm varies according to the type. For the quotidian it is about ten hours, for the tertian about eight hours, and for the quartan about five hours. In the tropics the duration of the paroxysm is more irregular,—the average of all the types seem to be pretty nearly the same, viz. ten hours.

The relative duration of the different stages is also less uniform in warm than in temperate climates; still, as a rule, the quotidian in the tropics has the shortest, and the quartan the longest cold stage.

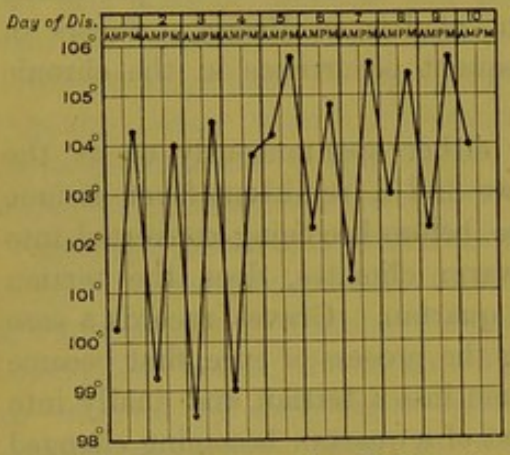

FIG. 13.—Quotidian type (so called) terminating in double tertian remittent.

The types of ague usually met with in tropical countries are illustrated in the accompanying diagrams. I do not find a tracing in my collection of a pure quotidian type, that is, a fever with similar daily paroxysms occurring in the morning, nor do I meet with such a tracing in any of the works on tropical fevers that I have consulted. The temperature curves in the summer fevers, which are regarded by the Italian school as essential quotidians, exhibit great irregularities, with exacerbations at tertian or quartan intervals. The diagram (Fig. 13) represents the temperature fluctuations during the first three days of the disease that is generally looked upon as quotidian; but it will be seen to bear the character of double tertian even at the commencement; and after the fifth day, when the fever assumed the remittent form, the double tertian impress is still more marked.

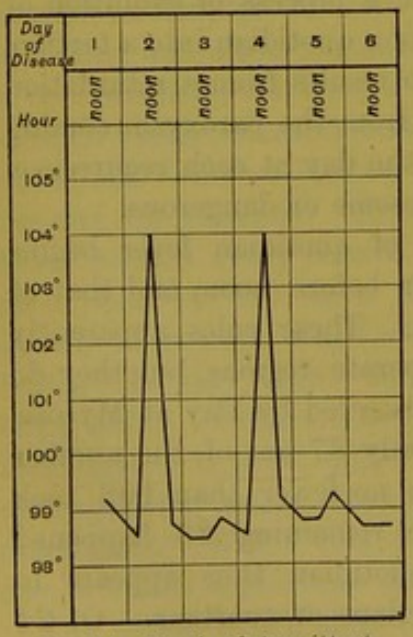

FIG. 14.—Tertian intermittent.

The accompanying figure (Fig. 14) is a tracing of a typical case of tertian. Quinine was given after the second paroxysm, and cut short the progress of the fever.

Double tertian is one of the most common forms of tropical intermittent fever. The case from which the following chart (Fig. 15) was

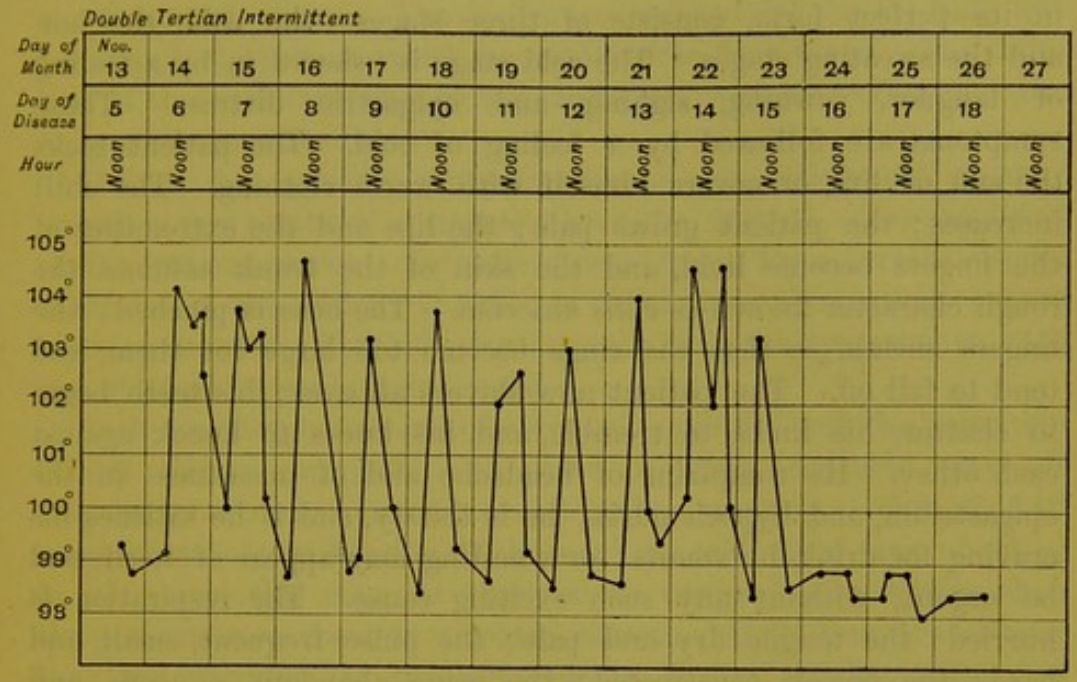

FIG. 15.

derived, was that of a young man who had been for the first time taken with fever five days before he came under observation. The disease probably began as a double tertian remittent. It will be seen that on the morning of the 7th the temperature only fell to 100° F. The fastigium sometimes exhibits a distinct amphibolic period, with a second rise before the definite defervescence takes place. Quinine in full doses given after the fifteenth day cut short the attack.

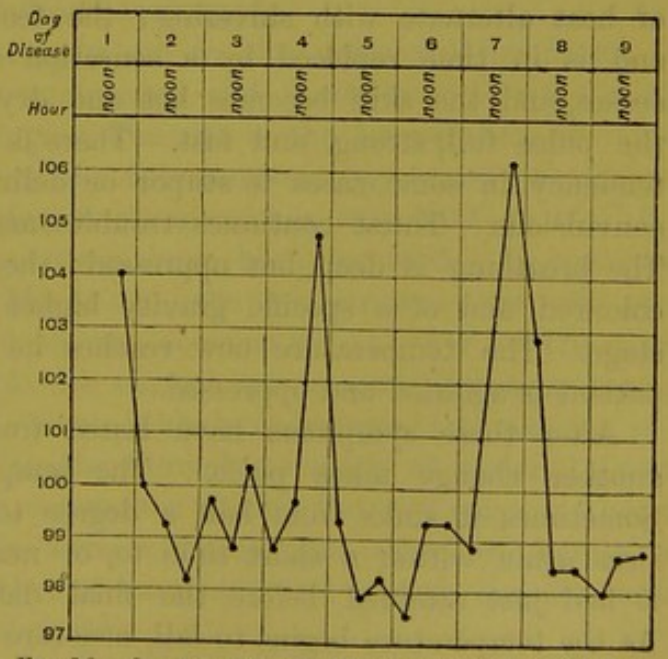

FIG. 16.—Quartan with abortive paroxysms intercalated on the third day.

The chart (Fig. 16) exhibits the quartan type, with an abortive paroxysm intercalated on the third day. This case was under the

care of my former colleague, Dr. Pellereau, to whom I am indebted for the chart.

Phenomena of an Ague Fit.—A paroxysm of intermittent fever, in its perfect form, consists of three stages—the cold, the hot, and the sweating stages. The cold stage is ushered in by a feeling of languor, yawning, sighing, and epigastric distress. These symptoms are followed by a feeling of cold. The patient seeks the sun or fire, or covers himself with warm clothing. The chill increases; the patient grows pale; the lips and the extremities of the fingers become livid, and the skin of the trunk assumes the rough character known as *cutis anserina*. The nose is pinched; the fingers shrink, so that the rings become too large for them, and tend to fall off. The patient now shivers all over; his teeth begin to chatter, his limbs to tremble, and his knees to knock against each other. He complains of headache and of uneasiness in the epigastrium, and hypochondria; he is thirsty, and if he satisfies his craving for drink he vomits; or vomiting may appear of itself, and be urgent, without any such exciting cause. The respiration is hurried; the tongue dry and pale; the pulse frequent, small, and hard; the bowels constipated; the urine abundant, aqueous, and passed frequently. The secretion of milk in nursing women is suspended, and ulcers dry up.

After this condition of things has lasted from half an hour to two or three hours, the patient's state changes. Transitory flushes of heat alternate with shivering; the feeling of cold diminishes, and is in time replaced by a sensation of heat; the trembling ceases, and the skin becomes hot and dry. The face is flushed; the pulse full, strong, and fast. There is severe headache, with a tendency in some cases to stupor or delirium, and in children to convulsions. Thirst continues troublesome. Epistaxis may occur. The breathing is deep, but oppressed; the urine is generally high coloured, and of a specific gravity higher than that of the cold stage. The temperature now reaches its highest point, and the patient is agitated and oppressed.

After these symptoms have lasted from two to eight hours, another change takes place. The temperature begins to fall. Sometimes it sinks from half a degree to two degrees, and then rises again within a short time to, or nearly to, the point which it had just attained before the final defervescence takes place. As the temperature begins to fall, moisture appears on the temples, face, and armpits; and this is followed by a copious sweat all over the body. The pulse diminishes in frequency, and becomes soft and undulating. The headache passes off, the oppression and restless-

ness become less urgent, the thirst subsides; the respiration is slower and easier; the urine, varying in amount inversely as the sweating is more or less abundant, is dark in colour, and deposits a sediment of urates. The patient, exhausted by the fit, falls into a tranquil sleep, from which he awakes free from fever. The duration of the sweating stage is indefinite and variable; often it is the longest of the three.

During the apyrexia the temperature may fall below, or may remain a few tenths of a degree above the normal. The condition of the patient varies. In some cases, except for a certain degree of weakness, the patient is well; in other cases the patient suffers more or less from headache, uneasiness about the epigastrium, with loss of appetite and general malaise.

Such are the phenomena of a typical ague fit; but the paroxysm is not always complete. In warm countries the cold stage is frequently slightly marked, or altogether wanting. Out of 240 cases observed by Waring in Burma,[1] no cold stage was observed in 116; and this stage is observed to become more severe when the patient is removed to a colder climate. Colin found, at Rome, that the elevation of the temperature during the hot stage was greatest in warm weather. The average temperature of the hot stage was 40° C., but during the greatest heat of summer it reached, and frequently exceeded, 41° C.[2] The sweating stage may be represented by a slight moisture only on the skin, or it may be abnormally severe and prolonged.

Temperature.—The temperature in the axilla will be found to rise before the chill has commenced, and it continues to rise during the whole of the cold stage. The rise in the temperature is rapid—in two or three hours the thermometer may stand at 102° or 103° F. At the fastigium it often reaches from 104° to 106° F.; and much higher temperatures have been recorded. In one case recorded by Stephen Mackenzie, the temperature rose on two occasions to 112°·8, and on one occasion to 113°·8 F.[3] The time occupied in the return to the normal line in some instances barely exceeded five minutes. Such high temperatures are curious, but they have no relation to the normal evolution of malarial fever. The temperature of the extremities during the cold stage is always much below normal; in some instances, but these are hardly to be regarded as benign, that of the mouth and rectum has also been observed to fall below normal.[4]

The Urine.—Just as a rise in the temperature is observed

[1] *Indian Annals of Medical Science*, 1853. [2] *Op. cit.* p. 190.
[3] *Internat. Journ. of Med. Sciences*, April 1892. [4] Lorain, *Études de méd. clinique*, 1877.

before the cold stage declares itself, so an augmentation in the excretion of urea commences some time before the chill appears, increases during the cold and hot stages, and diminishes during the sweating stage. This increased elimination of urea, which depends on increased production, takes place even when the paroxysm has been prevented by the administration of quinine. Uric acid and the chlorides are also increased, while the phosphates are diminished during the cold and hot stages, and increased in the sweating stage. When the paroxysm ceases, the urea and uric acid, which, indeed, often begin to diminish during the sweating stage, fall below normal; but Niemeyer has found the excretion of urea increased during the apyrexia of quartan. It deserves inquiry whether this excess represents urea formed during the previous paroxysm, or whether it is caused by a suppressed paroxysm, just as we find the urea increased when the febrile symptoms are checked by the administration of quinine.

The Blood.—The blood will in most cases be found to contain the endo-corpuscular or extra-corpuscular parasites we have described, or leucocytes containing pigment. In severe cases a notable diminution in the number of red corpuscles is observed. In relapses more or fewer of the corpuscles not invaded by parasites are found to be larger or smaller than normal. At the height of the paroxysm, and for some time during its decline, the number of leucocytes in the peripheral circulation is notably increased.

Course.—If the fever be left to itself, the paroxysms continue to recur for an indefinite time; but unless the disease proves fatal, they generally disappear, in time, without medical aid. If the patient remains in a malarious region, relapses are almost sure to occur; the disease then becomes chronic, and the patient cachectic. The quotidian and tertian types now tend to give place to the quartan, and to irregular types of long intervals. Even when the paroxysms are arrested by the administration of the bark or its alkaloids, they still show a strong tendency to relapse. The relapses of the quotidian or double tertian, commonly met with in the tropics, were found by Borius to take place most frequently on the seventh, fourteenth, and twenty-first days.

Forms.—Intermittent fever occurring in healthy Europeans, who have been only a short time in the tropics, often presents a sthenic or dynamic character. The cold stage is severe, the vascular reaction is excessive, with intense headache, sometimes with violent delirium, great thirst, and urgent vomiting, occasionally of bilious matters.

Among the debilitated and badly fed natives, the opposite and more dangerous condition of adynamia is frequently observed,

especially when the disease is epidemic. The cold stage is absent or imperfectly developed. The temperature in the hot stage ranges from 101° to 103° F. The sallow skin testifies to the imperfection of the reaction. The pulse is fast, weak, and irregular. There are giddiness, stupor, and profound prostration, bodily and mental, and the intermissions are marked by symptoms of great exhaustion.

The peculiarities of the severe forms of quotidian and tertian met with both in Europeans and natives are—(1) the rapid rise of the temperature to 104° up to 106° F.; (2) the long duration of the paroxysm and the brief period of apyrexia; (3) the marked oscillations of the temperature at the fastigium; (4) the sub-normal temperature (96°·5 F.) often met during the brief apyrexia; (5) the great anæmia and prostration by which they are followed. It is in such forms that Marchiafava has detected the small amœboid bodies, with or without pigment, and a dark and shrunken state of the parasite-bearing corpuscles.

Herpes and urticaria frequently accompany intermittent and remittent fever. Other forms of eruption are occasionally observed.

Pernicious Paroxysms. — A pernicious fever has been defined as one in which the sudden and unforeseen supervention of some symptom, or train of symptoms (not being an exaggeration of the phenomena proper to the fever itself), puts the life of the patient in jeopardy. Thus, when a patient suffering from intermittent fever suddenly becomes comatose or algid, he is said to be suffering from a pernicious comatose or algid fever. The coma or algidity has been generally regarded as something alien to the course of the fever, as, in fact, a superadded disease; and fevers of this nature have been described as *febres comitatæ* or complicated fevers. This view of the nature of these cases cannot be maintained. The coma or algidity occurring in the course of an intermittent fever is as much a manifestation of the malarious infection as are the vomiting and headache, which usually accompany the febrile paroxysm. Strictly speaking, there is no such disease as a pernicious fever; it is the paroxysm which is pernicious, the fever, during the course of which these symptoms appear, being simple, sthenic, or adynamic, and frequently continues a mild course after the paroxysm has passed off. We shall make use of the term pernicious paroxysm to designate these grave attacks which occasionally develop during the course of an intermittent fever, exhibiting symptoms referable to organic or irritative lesions of the brain and the alimentary canal.

Pernicious symptoms referable to the brain are coma, delirium,

and convulsions; those referable to irritative lesions of the intestinal canal are algidity alone, or combined with cardialgic, choleraic, dysenteric, or sudoral symptoms.

Pernicious symptoms frequently appear suddenly during the course of an intermittent fever that has been running a mild course, and they may either accompany a paroxysm or replace it. They may disappear with the paroxysm, and the fever then resumes its former course; or the pernicious symptoms may persist for one or two days, with or without fluctuations in their intensity; or, passing off with one paroxysm, they may recur with the next, or with some succeeding one; but in whatever way they occur or recur, or whatever may be their duration, they are always dangerous and often fatal. Suddenness of onset is not, however, so constant a character of these paroxysms as is generally supposed to be the case. In cerebral attacks, particularly, some change will frequently be remarked in the patient's manner before the pernicious paroxysm appears; or some peculiarity in the symptoms of the preceding paroxysm or paroxysms may be observed, such as intense headache, somnolence, or muscular tremors. The occurrence of such unwonted symptoms do not necessarily portend a pernicious attack, but they should put the physician on his guard. Colin justly reminds us, in this connection, of the maxim of Hippocrates: "Si quid in morbis præter rationem eveniat, non fidendum."

Pernicious symptoms, although not peculiar to any special type of intermittent fever, occur most frequently in the course of the quotidian and double tertian types, less frequently in that of a simple tertian, and still more rarely in connection with the quartan type.

Further, it must be remembered that pernicious symptoms are by no means peculiar to intermittent fever in any of its types. In my experience, coma, delirium, convulsions, and algidity are as rare in the tropics in connection with frank intermittent fever as they are common in the course of the remittent form. It is to the frequency with which these symptoms occur in connection with remittent fever in tropical countries that their gravity is due. It is in the malarious districts of subtropical climates, and on the table-lands of tropical regions, that pernicious paroxysms in the course of intermittent fever are most frequently observed. We shall proceed to give a brief account of the principal forms of pernicious attack.

1. *Comatose Attack.*—This, which is the most common form of pernicious paroxysm, is distinguished into two varieties, the comatose attack proper, and the apoplectic form. In the comatose variety, the coma may occur primarily, and be the only pernicious

symptom present; or it may be preceded by delirium, or complicated with convulsions. Its intensity varies in different cases from slight somnolence to carus. It makes its appearance during the hot stage, and may either pass off with the fever or persist for hours or even days, with or without a partial recovery of consciousness, and terminate in death or recovery. Graves relates a case of this form that came under his observation which illustrates very well its character when it is uncomplicated. A gentleman, after sleeping well until 4 o'clock in the morning, was awakened by a general feeling of malaise, followed by chilliness, some nausea, and headache. "After these symptoms had continued for an hour, his skin became extremely hot, the pain in the head continuous, and drowsiness was complained of, which soon ended in perfect coma, with deep snoring and insensibility." The patient was treated for apoplexy by bleeding; the fit passed off, and he was perfectly well in the evening. The day but one after, at the same hour, the same symptoms returned, and were removed by the same remedy. A third attack occurred after another tertian interval, when the nature of the disease was recognised, and its further return prevented by the exhibition of large doses of quinine.

2. *Apoplectic Attack.*—The apoplectic form is a modification of the comatose seizure, and differs from it in appearing at the beginning of the paroxysm, which it may be said to replace, and not in connection with the vascular reaction of the hot stage. The ordinary febrile phenomena, in short, give place to a comatose attack. The following case illustrates this form of the disease:—

Apoplectic Attack in the course of Quotidian.

L. N., aged 31 years, has been in Madagascar for about a year, and had his first attack of malarial fever about eight months ago. This lasted for about a week, and he then remained well for about three months, when he experienced a few returns of fever, which seem to have been of the quotidian type. For the past three days he has had fever recurring in the early part of the forenoon, and passing off towards evening. The cold stage is said to have been short and imperfect, the hot stage marked by high fever with intense headache. On the morning of March 3rd, 1875, without having made any complaint of being unwell, and while reclining in an easy-chair, he was observed to be drowsing. This at first attracted little attention, as it was supposed that he was asleep. After a short time his breathing was observed to be heavy, and on attempting to rouse him it was found that he was insensible. When seen at 3 o'clock in the afternoon, his eyes were half open, and the pupil moderately dilated; the skin was slightly warm, but moist; the pulse 95, and the face pale. On being shaken, he showed some signs of consciousness, but was unable to reply, or even to swallow. An enema of 40 grains of quinine was administered, and sinapisms applied to the extremities. In the evening he was still soporose, but on being

roused could understand questions, and was got to swallow 20 grains of quinine.

March 4th. — Temperature and pulse normal — intelligence perfect. Quinine was given in 15 grain doses every eight hours. From this time there were no further recurrences of the fever.

3. *Phrenetic Attack.* — A minor degree of delirium is not uncommon in severe attacks of intermittent fever, especially in the case of those who have been addicted to drink, who have suffered from anxiety, or who have been worn out by brain work. Occurring in these circumstances, it is not to be regarded as the sign of a pernicious attack. Even severe delirium, if it occurs only during the height of the reaction, and disappears on the approach of the sweating stage, is not in all cases to be looked upon as of grave import. Pringle relates that some of the British troops in Dutch Brabant were seized at once with symptoms of an ardent fever and high delirium, which continued for some hours, and then went off with a profuse sweat.[1] If the delirium persists during the sweating stage, and still more if it continues during the apyrexia, there is danger of it ending in coma. The persistence of mild delirium, after the subsidence of the hot stage, frequently presages a severe attack during the succeeding paroxysm.

4. *Convulsive Attack.*—Convulsive attacks of an epileptiform character are exceedingly common in children during the hot stage of fever. They are usually accompanied with some degree of somnolence or coma. These symptoms usually pass off with the fever, and may return with the next paroxysm. I have occasionally observed these convulsive attacks, occurring during infancy, to lay the foundation of chronic epilepsy and mental disorder. Purely convulsive attacks are seldom, if ever, seen in adults. Trismus is frequently present in comatose seizures, but clonic spasms of the muscles of the extremities are more rarely observed, even as a complication of comatose symptoms.

5. *The Algid Attack.*—The algid attack may be a prolongation and intensification of the cold stage, but much more frequently it declares itself during the stage of reaction. The algid symptoms manifest themselves suddenly. The pulse becomes weak and fast, and ultimately imperceptible. The extremities become cold, and in a short time livid and icy. The tongue is cold and pale, and the lips livid. The breath is cold, the voice extinguished, the eyes sunken, and the nose pinched. The thermometer in the axilla or rectum may, however, stand at 103° or 104° F., or, much more rarely, a little over, or below, normal. The intelligence remains

[1] Pringle, *Observations on the Diseases of the Army.* London, 1765.

intact, unless, as sometimes happens, comatose symptoms are also present. When recovery is about to take place, the pulse increases in strength, the skin of the extremities regain their warmth, and the central temperature falls. The duration of the algid state varies greatly in different cases. It may pass off within a few hours, or persist for twenty-four hours or longer. The algid symptoms, unlike the comatose, are never seen to recur in paroxysms.

The following case exhibits some of the principal features of this form of seizure :—

ALGID ATTACK OCCURRING IN THE COURSE OF A DOUBLE TERTIAN. DEATH.

R., a young man aged 25 years, had been trading in a very malarious locality where he contracted fever. Entered hospital on the 5th February for enlargement of the spleen and general debility. He seemed to be improving under tonic treatment, when, on the 10th, he had a paroxysm, marked by severe supraorbital headache, and a tendency to cramps in the lower extremities.

11*th*.—At 9.30 A.M. free from fever, but still complains of occasional crampy pains in the legs. During the forenoon slight rise in the temperature.

12*th*.—At 9 A.M. the patient is in bed. He states that he is well; but on examination his face is seen to be pale, and the extremities are cold. The temperature in the axilla is 102°·3 F.; the pulse fast and weak; no thirst.

Evening.—In the same state, no reaction.

13*th*, *morning*. — Extremities icy cold; skin covered with a clammy moisture; pulse imperceptible; tongue pale and cold; lips exsanguine; breathing hurried; intelligence intact. Died at noon.

AUTOPSY.—*Brain*.—Some congestion of the membranes; brain substance normal. *Liver* enlarged, congested, of a dark colour, and somewhat diminished in consistence. *Spleen* weighs 2½ lbs.; soft and pigmented. *Intestinal canal*. — The jejunum is injected in patches, otherwise normal. *Kidneys* of a dark coloration, and much congested.

If to these algid symptoms there be added an agonising pain at the epigastrium, with, it may be, hiccup and vomiting, we have a picture of the *cardialgic* form of paroxysm.

When, again, in conjunction with the algidity there are cramps of the extremities, urgent vomiting, rice-water stools, diminution or arrest of the urinary secretion, with extinction of the voice and a glazed condition of the eye, the case is one of the *choleraic* form. The choleraic form in its milder grades is often recovered from. It is specially common during the warmest months. In Cochin-China it is one of the most frequent forms of pernicious attack, and it has on more than one occasion assumed epidemic prevalence in India.

When, during the rigor, the patient is seized with colic, followed by sanguineous, mucous stools, accompanied with tenesmus and coldness of the extremities, with a small and frequent pulse and great debility, the dysenteric symptoms passing of on the appear-

ance of a copious sweat, we have presented to us the features of that rare form known as the *dysenteric* pernicious attack.

When the sweating stage is exaggerated and prolonged, with algidity and a tendency to fainting, we have to deal with a case of the *sudoral* or *diaphoretic* form of paroxysm.

A *syncopal* form has been described by most authors who have treated of pernicious intermittents. Fainting is by no means rare in fever, occurring when the patient turns in bed or attempts to walk. I have frequently observed this symptom in the remittent form of the disease. Fainting is also a common accompaniment of algid attacks. Bell described a form of fever observed at Teheran, called Tab-i-ghash or "fainting fever," which appears to have been only a form of the algid paroxysm. "Its usual form," he says, "is an ague in which the cold fit is accompanied by extreme oppression at the heart, and pain when pressure is made on the pit of the stomach. This goes on for some days, when at the commencement of each ague fit the patient becomes insensible. The pulse is not to be felt; he neither shivers nor sweats properly, and his skin is cold and clammy; he has a few spasms and dies, mottled like a man in cholera." [1]

Analysis of Symptoms. — We look upon the parasites occurring in the blood, spleen, and other organs of patients suffering from the malarial infection as the essential cause of the fever. Why in some cases the infection should run an apyrexial course, characterised only by cachexia, is quite unknown. The recurrence of the febrile paroxysms at fixed periods—a phenomenon so characteristic of the malarial infection—is probably, as Golgi has endeavoured to show, related to the evolution-cycle of the parasite. The rise of the temperature in the initial stage is the immediate expression, on the one hand, of the increase in the oxidative processes indicated by the increased excretion of urea and carbonic acid; and, on the other, of a relatively, if not an actually, diminished loss of heat caused by the tetanic spasm of the cutaneous arteries, which also accounts for the lividity and diminished temperature of the extremities. In what way the presence of the parasites gives rise to these metabolic processes which cause the fever is imperfectly understood.

The subjective sensation of cold, notwithstanding the actual increase of the temperature of the internal parts of the body, is probably explained, as Cohnheim suggests, by the diminished heat supply to the thermal apparatus upon which the sensation of heat and cold depends. "The determining element in our sensation of temperature, is the temperature of the thermal apparatus itself." The severity of the chill will be in relation to the degree of the arterial

1 Eastwick, *Three Years' Residence in Persia.* London, 1864.

spasm, the extent of the cutaneous area affected, and the suddenness with which the circulation is arrested. The beginning of the second stage marks the relaxation of the arterial spasm. During this stage the actual loss of heat is greater than normal, but not sufficient to counterbalance the excess of heat production, and for this reason the temperature continues to rise during the first part of this stage. The headache, oppression of breathing, enlargement of the spleen, uneasiness in the pit of the stomach, and right hypochondrium, are due to collateral fluxion during the cold, and to dilatation of the arteries during the hot stage. The headache in some cases may partly result from the accumulation in the capillaries of the brain of malarial microbes and melaniferous cells. The enlargement of the spleen is no doubt largely owing to the vast number of these bodies, and of partially destroyed red corpuscles, with which it becomes engorged during the febrile paroxysm. The increase in the colouring matter of the urine after the paroxysm is chiefly to be ascribed to the destruction of the red corpuscles during the fever.

The cerebral symptoms which we have described are always associated with blocking of capillary areas by parasites, and pigment-bearing microphages and macrophages, and by free pigment. The peculiarities of the different forms of cerebral attack will probably be found to depend on the special area involved; and the longer or shorter duration and intensity of the symptoms may be supposed to be related to the extent and completeness of the capillary occlusion. It has been proved beyond all doubt that in fatal cerebral pernicious attacks, such occlusion of the capillaries by parasitic forms actually occurs; but it is still open to doubt whether their action is entirely mechanical, or mechanical and toxic.

The symptoms of the algid forms point to derangement of the vasomotor system and depression of the heart's action. Experiments show that the alimentary tract is in closer connection with the cardio-inhibitory centre than other parts of the body, and that irritation of this tract, if sufficiently powerful, will produce cardiac inhibition, with pallor of the surface, and accumulation of the blood in the abdominal vessels. That the intestinal canal is the centre of mischief in this form of pernicious attack will appear all the more probable if we observe the character of the disturbances so frequently associated with the algid condition—the cardialgic pain, the choleraic vomiting and purging, and the dysenteric discharges. Bignami has found parasitic thrombosis of the capillaries in the intestinal and gastric mucous membranes of patients dying of the choleraic form.[1] In the irritation caused by the invasion of the

[1] *Op. cit.* p. 48.

intestinal capillaries by parasites and pigment-bearing cells, we look for an explanation of the principal phenomena of the algid paroxysm.

Mild Remittent Fever.—Mild remittent fever is often preceded for a day or two by languor, nausea, and oppression at the epigastrium; but in many cases no premonitory symptoms are observed. The disease may commence either with a distinct rigor, or only with a feeling of chilliness; and sometimes even this is absent. The symptoms of the disease, in whatever way it may have begun, are those of fever—a high temperature—102° to 105° F.; headache; pains in back and limbs; fulness and oppression in the epigastrium; nausea, frequently with retching and vomiting. After a period of from six to eight hours the temperature falls two or three degrees, but does not reach the normal; the skin becomes moist,—in rare cases a free perspiration takes place, and all the symptoms become ameliorated. The fever, constituted by a succession of such exacerbations and remissions, is continued for a period of three or four days in mild cases; for a week or ten days in those of moderate severity; for periods varying from a fortnight to three weeks or longer in the more severe and complicated forms of the disease.

The exacerbations come on at various hours of the day. When the fever follows the double tertian type, the exacerbations of alternate days correspond in respect to severity and to the hours at which they appear. In some unusually severe cases we observe two exacerbations in the twenty-four hours,—one at noon, the other at midnight, with morning and evening remissions; or with exacerbations in the morning and evening, with remissions during the afternoon and night. Whether the disease has commenced with a rigor or not, the exacerbations following the first onset are very seldom ushered in with rigors. There may be a feeling of chilliness, but this is not constant. The exacerbations do not, as a rule, end in sweating. A remittent fever has no real sweating stage. A partial moisture breaks out on the head and neck or armpits, and the skin of the trunk may lose its pungent dryness; but copious perspiration is only met with when the disease is subsiding, and not always then. The length of the remission is variable—usually from two to twelve hours.

When the disease is severe or protracted the remissions tend to become shorter and less distinct, but in the mild remittent they do not completely disappear. The temperature varies greatly in different cases, but it seldom exceeds 105° F., and is generally considerably less. The tongue, which is large and flabby, gets thickly coated as the disease runs on, and becomes brown in the centre, but

continues moist, at least during the remissions. Tenderness on pressure at the pit of the stomach is a frequent, distressing, and persistent symptom. The bowels are often constipated at the beginning, but the stools frequently become loose and bilious as the disease proceeds. If the fever subsides within a week, no enlargement of the spleen may be detected; if of longer duration, it will usually be found to be increased in size. Delirium sometimes appears at the onset of the disease, and passes off during the remission, and may not again recur during its course. Or there may be a slight wandering during the succeeding exacerbations. More frequently, in the mild form, the mind is clear throughout the fever. Jaundice is seldom present in this form. The disease terminates by a gradual abatement of the symptoms, by a copious perspiration, or by being converted into an intermittent.

Gastric Remittent.—The gastric form of remittent fever is most common in the warm season, and is frequently the initial febrile manifestation of malaria in the European on his arrival in a malarious tropical country. It usually begins with a severe rigor, followed by high fever, dry burning skin, hard and fast pulse, violent headache, and pains in the back and limbs. The face is flushed, the conjunctivæ are injected, the tongue is large, flabby, and covered with a thick yellow fur. There is nausea, a sense of fulness in the epigastrium, and often urgent vomiting, which is sometimes bilious, accompanied with constipation, or, less frequently, with diarrhœa. The urine is high coloured, but does not usually contain biliary colouring matters. The fever may exhibit slight morning remissions, with an amelioration of the symptoms, and afternoon or evening exacerbations. The fever is a short one, lasting from three to seven days, but leaving the patient weak and liable to future attacks of remittent or intermittent fever. The following case represents the symptoms and course of a mild form of the disease:—

GASTRIC REMITTENT FEVER, PRIMARY INVASION.

A. B., aged 19, a sailor, had remained for about a fortnight in the harbour of Port Louis, and had landed on several occasions. This was his first visit to a malarious port. On the fifth day after leaving Port Louis, and while at sea, he complained of headache and pains in the back, but the pulse and temperature were normal. Next day (19th April) fever began, and ran the course indicated on the chart (Fig. 17). The general symptoms were those which we have described as characteristic of this variety of fever.

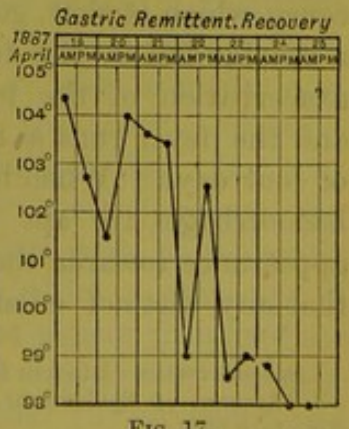

FIG. 17.

Hallucinations are frequently present in the severe gastric remittents of Africa. Cameron the African traveller contracted fever at Unyanyembi, during the first part of his journey. It commenced with severe rigors, and was attended with loss of appetite, giddiness, great prostration, and delirium. Cameron described his sensations to Dillon his companion thus: "The fellows have regularly blocked me in. I have no room to stir; the worst of it is that one of the legs of the great piano is on my head, and the people are strumming away all day. It's all drawing-room furniture that they have blocked me in with." On other occasions he felt as if he were several persons, each of whom was suffering the same torments. This peculiar form of delirium is very common in the initial gastric remittent of Africa.

This form of fever, like the bilious remittent to be presently noticed, is usually found associated with very small amœboid endocorpuscular bodies and melaniferous leucocytes[1] in the blood.

Bilious Remittent.—Bilious remittent assumes two forms —the acute and the subacute.

The acute form of bilious remittent is frequently a fever of primary invasion, lasting usually from four to ten days. Although it is not peculiar to any season, it is most frequently met with in autumn, when malaria is intense. It may be preceded by a premonitory period of two or three days' duration, marked by heaviness of the head, languor, and a feeling of discomfort or tension at the pit of the stomach. Bilious vomiting, the vomited matters in bad cases being dark green, almost black,[2] is a prominent symptom of the disease, to which bilious purging is generally added, along with the early appearance of jaundice. The urine is scanty, contains biliary colouring matters, and is occasionally slightly albuminous. The fever frequently follows the double tertian type, with more or less marked remissions. These symptoms may continue for three or four days, and then disappear, and convalescence, which is slow, goes on uninterruptedly. In other cases, after a notable fall in the temperature, or even a complete apyrexia, an exacerbation of the fever occurs, the bilious vomiting reappears, and the fever runs a remittent course for another period of three or four days. When it runs a prolonged course, epistaxis and other hæmorrhages are apt to occur, and anasarca sometimes makes its appearance towards the close. The liver is tender, but it is seldom that any distinct enlargement of the organ can be made out on

1 See observations 1 to 5 in Laveran's work, *Du Paludisme*, pp. 221–225.

2 "Account of Epidemy of Remittent Fever at Prome," by Murphy, *Med. Times and Gaz.* July 1853.

percussion. The spleen is generally enlarged in the more prolonged forms of the disease.

The subacute form of bilious remittent runs a more protracted course, generally lasting from seven to fifteen days. It frequently begins with one or two paroxysms of ordinary remittent fever, the remissions being well marked. The bilious vomiting and jaundice make their appearance from the third to the sixth day. The spleen becomes enlarged as the disease advances. The fever either subsides gradually, or becomes converted into an intermittent before disappearing entirely.

A certain degree of mental torpor, amounting it may be to stupor, is occasionally observed in bilious remittent. In severe cases a fatal issue is caused by the supervention of coma, or the patient passes into a typhoid state,—a condition which will be presently described.

Analysis of Symptoms.—The most marked feature in the bilious remittent is the hypersecretion of bile, as manifested by the bilious vomiting and purging, the high-coloured urine, and the jaundice. This increased secretion of bile is to be ascribed to the rapid destruction of the red corpuscles of the blood which characterises this form of the disease. The liberated hæmoglobin becomes transformed by the liver into bile, which is partly reabsorbed and deposited in the tissues, giving rise to jaundice, and is eliminated by the urine in the form of urobilin and other colouring matters.

A notable diminution in the number of the red corpuscles is invariably observed in bilious remittent, a distinct loss taking place with each exacerbation. This accounts for the rapidly induced anæmia, and the tendency to anasarca, which are often observed when the disease has persisted for some time. The most reasonable explanation, as yet a hypothetical one, of this hæmolysis, is to assume that the microbes give rise, directly or indirectly, to some toxic substance which acts destructively on the corpuscles.

Grave Remittent Fever.—The simple forms of remittent fever which we have just described, although seldom directly fatal, initiate or accentuate processes which lead to that constitutional deterioration which will be described under the chronic infection. They are thus never to be considered as trivial maladies. But they demand careful attention and treatment for another reason, viz. that during their progress certain symptoms not unfrequently become evolved that at once impress upon them a character of gravity or malignity that places the life of the patient in immediate danger. These symptoms may be ranged under four heads—(1) cerebral symptoms; (2) typhoid symptoms; (3) adynamic and

algid symptoms; (4) hæmoglobinuria. Several of these classes of symptoms may be conjoined in a single case.

1. *Grave Remittent with Cerebral Symptoms.*—A fatal termination of remittent fever is most frequently caused by the supervention of cerebral symptoms, such as delirium, convulsions, and coma. The following two cases will illustrate the manner in which these complications arise during the progress of remittent fever:—

Remittent Fever (Relapse), Hyperpyrexia, Coma, Convulsions. Recovery.

Otto Kash, aged 24. Admitted into the Civil Hospital of Port Louis on the 13th February 1878, having already been twice in hospital within the space of fifteen days, on both occasions suffering from fever. On admission, the patient complained of pain in the abdomen, vomiting, diarrhœa, anorexia, and sleeplessness. The urine was scanty and high coloured. He was treated with cinchonidine, 10 to 20 grains every four hours. The fever ran a very irregular course, ranging from 100° F. in the morning to 104° in the evening, and with little change in the general symptoms till the 27th, when he was reported to have vomited his dinner at 5 P.M. During the evening his temperature rose to 106° F., and he again vomited and fell off his bed. When seen by the resident surgeon at midnight he was insensible, left eye drawn outwards, right eye natural, both pupils dilated. Legs and hands convulsed, skin hot and moist, temperature 106° F. Hiccup. He was got to swallow a mixture containing 20 grains of quinine, with 10 drops of tincture of digitalis in an ounce of decoction of bark; and this dose was repeated every four hours. From this date the patient steadily improved, and was discharged on the 9th of March.

Bilious Remittent, Delirium, Coma. Death.

P. T., aged 16 years. Admitted on the 13th of March, having suffered for three days from fever, which began with rigors, headache, and vomiting. On the day of admission and the previous day, in addition to the bilious vomiting there had also been bilious diarrhœa.

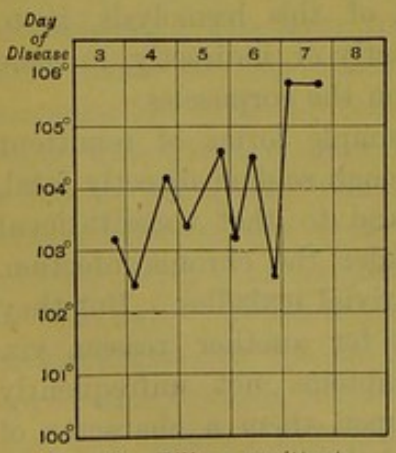

Fig. 18.—Bilious remittent. Delirium and coma.

On admission his temperature stood at 103°·2 F. Complained of uneasiness in the epigastrium, with nausea and frequent vomiting. The spleen was slightly enlarged, and tender on pressure. The liver tender on pressure, but not enlarged. The conjunctivæ slightly icteric.

The fever ran the course indicated in the accompanying chart (Fig. 18). It never came down near the normal, but remained persistently high. Nausea, bilious vomiting and diarrhœa, with great restlessness and debility, were the prominent symptoms for the first two days after admission. On the fifth day of the disease the conjunctivæ were injected, and the patient complained of headache, which persisted during the day and the succeeding night. On the forenoon of the sixth day he became delirious,

tried to get out of bed, and was noisy. On the seventh day of the disease the temperature rose to 105°·8 F. His pulse was fast and small; he was prostrate; passed his motions in bed; became comatose, and died at 3 A.M. on the eighth day.

AUTOPSY.—*Brain* congested, but not visibly pigmented. *Liver* enlarged, softened, deeply congested, and pigmented. *Gall bladder* distended with thick bile. *Spleen* weighs 14½ oz., softened and deeply pigmented. *Kidneys* congested. *Intestinal canal*—mucous membrane of the stomach vascular; small and large intestines healthy. Other organs presented no important lesions.

The delirium and coma often appear in these dangerous cases after the fifth day. In many instances the approach of delirium is marked by a distinct fall in the temperature. When coma is present on the first days of the fever there is usually a history of insolation. When delirium occurs at a late stage it is then of a low muttering form, and is often connected with a typhoid state. It is always to be looked upon as a sign of extreme danger if the delirium augments, or even continues, as the temperature falls.

2. *Typhoid Remittent.*—Typhoid symptoms may appear during the course of any form of remittent fever; but by preference it occurs during the evolution of the gastric and bilious forms, and generally between the third and the ninth day of the fever. In the severe endemic forms of highly malarious tropical countries the typhoid condition is apt to appear at an early period of the fever. After the fever has lasted for a longer or shorter time, the pulse becomes weak and fast; there is great prostration; stupor and sub-delirium appear; the tongue becomes dry and black; sordes collect about the teeth and gums; the skin is sallow or icteric; the lungs are hypostatically congested, and the spleen enlarged. The urine is high coloured, and frequently more or less albuminous; the bowels are usually relaxed, and the motions bilious. Bilious vomiting may occur from time to time during the progress of the disease. In the latter part of its course there may be some œdema of the lower extremities.

The fever by which these symptoms are accompanied is marked by great fluctuations. The temperature may sink to the normal, and remain low with slight oscillations for one or two days, without bringing about the slightest improvement in the state of the patient, then rise again to 104° or 105° F.

This typhoid condition may remain unmixed until death or recovery takes place; but adynamic and algid symptoms are apt to supervene after a few days. The algidity is seldom so marked as in the pernicious algid paroxysm which we have described. When this state of things arises, we have, in addition to the

symptoms proper to the typhoid state, a notable coldness of the extremities, generally accompanied with an axillary temperature not more than from one to three degrees above the normal. The voice of the patient is hardly audible; his expression that of extreme apathy, and the pulse hardly to be felt. From this condition, hopeless as it appears, recovery may take place by a slow amelioration of all the symptoms, or the typhoid or adynamic symptoms may increase and terminate in fatal syncope or exhaustion.

3. *Remittent Fever with Algid and Adynamic Symptoms.*—Algidity may declare itself in the course of an ordinary remittent fever, with the same symptoms as those which characterise the pernicious algid paroxysm. Colin relates a typical instance of this nature. The patient had already been three times in hospital for malarial fever. After two days of intense and continued fever, which commenced without rigors and was marked by vomiting, and by headache so severe as to extort cries of pain, he suddenly became algid, his extremities cold and covered with a clammy sweat. The following day the algidity persisted, without the patient having the least sensation of cold; the urine was suppressed, the voice became extinguished, the intelligence remained perfect. On the third day sub-delirium appeared, while the algidity increased, and the patient gradually sank. The principal lesions were: a transparent gelatinous effusion into the meshes of the pia mater; uniform congestion of the entire intestinal canal, especially of the great curvature of the stomach, and enlargement, softening, and pigmentation of the spleen.[1]

A form of adynamic fever of long duration, sometimes extending over a period of one or two months, which has been described by French authors, remains to be noticed. It is a form which I have not recognised; but it no doubt exists, although it appears to be associated more commonly with the chronic than the acute stage of malaria. It is said to be generally observed in those whose constitutions have been enfeebled by repeated attacks of malarial fever, or who are suffering from malarial cachexia. The fever is irregular in type, continuing high for several days, and then falling to the normal or sub-normal, often to rise again to 104° or 105° F. after rigors. Muscular prostration, shown by a staggering walk and a trembling hand; cardiac debility, manifested by a weak and irregular pulse, and by a tendency to faint; great mental apathy, and profound anæmia, are the leading features of the disease. Typhoid symptoms, however, such as sub-delirium, subsultus tendinum, and a dry and black tongue, may become developed at some stage of the complaint—often, indeed, at an early stage. There

[1] *Op. cit.* p. 251.

may be epistaxis, diarrhœa—often bilious, hæmorrhages, gangrene of the cheeks or gums, or of the genital organs. The disease has been happily described by Haspel as a long marasmus interrupted by "crises."

4. *Bilious Hæmoglobinuric Fever.*—Bilious hæmoglobinuric fever is met with in Madagascar, Mauritius, Senegal, the Gold Coast, Venezuela, French Guiana, Central America, the Southern States of the Union, the West India Islands, and some parts of Italy. Of 2600 cases of fever treated at Nosi-Bé on the west coast of Madagascar, 185, or 1 in 14, were of this form. In Rome it has in some cases been found associated with the malarial parasites; but in two cases no parasites were discoverable.[1] Prout appears to have met with malarial parasites in this form of fever on the Gold Coast.[2] Indeed, it is now well known that hæmoglobinuria and fever may arise apart from any malarial complication.[3] Bilious hæmoglobinuric fever seems to be malarial fever *plus* some unknown condition leading to wholesale destruction of the red corpuscles. None the less does malaria play an important part in predisposing to the attack, in modifying its course, and in determining its issue. Death generally takes place from suppression of the urine, and the resulting coma and convulsions, or from syncope or collapse.

The following case, which I saw along with Dr. Pellereau, exhibits very well the principal features and progress of the disease:—

BILIOUS HÆMOGLOBINURIC FEVER. RECOVERY.

X., aged 24, a native of Pondicherry, came to Mauritius in 1871. Six months after his arrival he had several paroxysms of intermittent fever, which after a time changed to the remittent type. In 1872 he had an attack of fever accompanied with jaundice, and passed urine which seemed to be mixed with blood. This condition lasted for two days. He had a second similar attack in 1874, which also lasted for two days; and a third in 1875. On these three occasions the supposed bloody urine appeared *after the rigors.* After this the patient continued in good health until the morning of 29th October 1877, when at about 4 o'clock he was seized with a severe pain in the right hypochondriac region, which prevented him from breathing freely.

Present condition, 29th October.—Icteric colour of skin and conjunctiva; pulse, 60 and compressible; temperature, 37° C. Heart and lungs normal; spleen much hypertrophied, reaching down into the iliac fossa, not painful on pressure. Slight tympanitis, tongue covered with a thick greenish fur; bilious vomiting and stools. Was ordered calomel followed by a saline.

October 30th, 8 o'clock A.M.—Slight pain and tenderness in right hypochondrium; jaundice deeper; nausea and bilious vomiting; anorexia; pulse, 90; temperature, 38°.

[1] *Riforma med.*, June 1892.
[2] *Brit. Med. Journ.*, 1889, vol. i. p. 998.
[3] *Lancet*, August 1, 1891.

At 9 P.M.—Jaundice not so deep; pulse, 90; temperature, 38°; was ordered hydrobromate of quinine, 1 gramme.

October 31*st*, 8 o'clock A.M.—Patient much better in every respect; asks for food; pulse, 80; temperature, 37°. Was ordered a seidlitz powder, which was followed by four bilious stools.

5 o'clock P.M.—Half an hour after breakfast patient noticed that his urine was getting more "bilious." At 1.30 P.M. had repeated rigors; then at 4.30 passed dark urine. Now (5 o'clock) the jaundice is deeper; skin hot, and covered with perspiration; patient complains of frontal headache; no pain in loins, or anywhere else, excepting very slight uneasiness in right hypochondriac region. Pulse, 100; respirations, 26; temperature, 40°·6; tongue white; no vomiting, but has had several bilious stools. Slight systolic murmur at base. The urine obtained early in the morning was acid, and contained no albumen; that passed immediately before the rigors was alkaline, and contained $\frac{1}{5}$ of albumen; that passed three hours after rigors was like port wine; alkaline in reaction; showing no trace of bile, but containing $\frac{4}{5}$ of albumen.

Examined microscopically, not a single blood cell could be found, but there were a few mucous corpuscles, a large number of granules dark or bright, according to focus, a considerable number of vibrios, and a few bacteria. This refers to the last urine, and it was examined shortly after it had been passed.

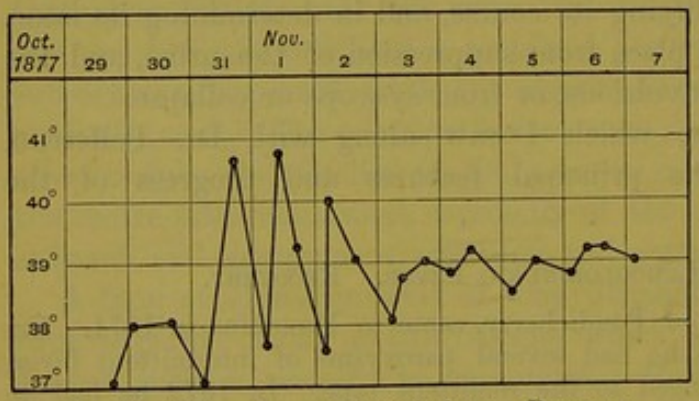

FIG. 19.—Bilious hæmoglobinuric fever. Recovery.

The blood was aqueous; red corpuscles diminished in number, having little tendency to run into rouleaux, with increase of leucocytes. Many of the red cells were more or less deformed.

The patient was ordered 2 grammes of the hydrobromate of quinine, with sulphuric lemonade and ice.

November 1*st*, 8 A.M.—Jaundice deeper; pulse, 84; temperature, 37°·5. No cephalalgia. Pain in hypochondriac region very slight; no vomiting, bilious stools, deafness. Urine scanty, high coloured, acid, no albumen. Specific gravity normal. Continue treatment.

12 noon.—Patient states that he observed his urine begin to get darker, and shortly afterwards, viz. at 10.30 A.M., he had rigors which lasted for an hour. Half an hour after the termination of the rigors he passed port wine urine. During the hot stage he had bilious vomiting and stools. When seen at noon, pulse, 88; temperature, 40°·6. Hydrobromate of quinine, 0·50 centig., was injected under the skin; patient was ordered iced champagne.

5 P.M.—Pulse, 100; respirations, 26; temperature, 39°·3.

November 2*nd*, 8 A.M.—Jaundice much deeper; no itching, great prostration; tongue deep yellow, looking very thin and indented at margins, as is often seen in this form of fever. Liver and spleen tender, but no enlargement of liver or further increase in the size of spleen. Pulse, 96; temperature, 37°·5; urine only high coloured. Treatment by hydrobromate of quinine injected subcutaneously and by enema.

2 P.M.—States that at 10.30 A.M. he had repeated rigors, and that two hours afterwards he passed blood-coloured urine. Pulse, 110; temperature, 40°.

5 P.M.—Pulse, 100; temperature, 39°. Continue the hydrobromate of quinine injections.

November 3rd.—Slight tendency to delirium last night; bilious vomiting and stools; systolic murmur in great vessels. Urine only high coloured, acid, no albumen; sp. gr. 1020. Pulse, 100; temperature, 38°. One gramme of quinine injected below the skin.

1 P.M.—Pulse, 100; temperature, 38°·8. Fit came on at the same time, but slight; urine high coloured, but nothing more.

9 P.M.—Pulse, 110; temperature, 39°; urine good. One gramme of the hydrobromate to be injected beneath the skin.

November 4th, 8 A.M.—Pulse, 100; temperature, 38°·8; jaundice considerably diminished; tongue slightly yellow; ordered purgative.

2 P.M.—Temperature, 39°·2; pulse, 100.

November 5th.—Better. Respirations, 16; pulse, 100; temperature, 38°·5; jaundice almost gone; tongue cleaner and no longer presenting the thin appearance which we noticed; systolic murmur very loud; œdema of tibiæ.

2 P.M.—Pulse, 100; temperature, 39°.

November 6th.—Slight delirium last night. Patient states that he feels better; no pain, vomiting, or diarrhœa. Appetite improving. Pulse, 100; temperature, 38°·8.

2 P.M. } Pulse, 100; temperature, 39°·2.
5 P.M. }

November 7th, 8 A.M.—Pulse, 90; temperature, 38°. From this time the patient's condition improved daily.

Epidemic Manifestations of Malarial Fever.

The following are some of the points in which malarial fever in its epidemic manifestations differs from the endemic disease:—

1. In regions where the tertian and quartan types are endemic, the quotidian type comes to predominate during epidemic periods; and in those countries where the endemic fever is of the quotidian or double tertian type, these are replaced by the remittent and pseudo-continued forms; and fevers of short intervals, or of the remittent or continued forms, are most prevalent at the height of the epidemy.

2. Pernicious attacks—cerebral and algid—become more prevalent in epidemic seasons.

3. The destruction of the red corpuscles is vastly more rapid, and hæmorrhages, profound anæmia, and cachexia are established at an early period of the disease.

4. In severe epidemies, adynamic and typhoid symptoms, the result in part of the anæmia, come to the front, and impart to them their destructive and fatal character.

5. Natives, who are comparatively exempt from the endemic disease, suffer severely during epidemies.

The following notes will serve to illustrate these points.

In the great epidemy of 1866–67, in Mauritius, the admissions from the remittent and pseudo-continued forms were in direct relation to the intensity of the fever. Icterus, hepatic enlargement, diarrhœa, dysentery, and hæmorrhages were frequently present. Rapidly induced anæmia was a marked character of the fever. Comatose and algid seizures were numerous. Dr. Power mentions the prevalence of collapse as a cause of death. "By collapse," he says, "we mean a state of profound anæmia from the blood-poisoning effects of malaria. The symptoms come on with little or no warning. The patient has had one or two attacks of malarial fever (the fever itself not having been necessarily severe); instead of convalescing as usual, the patient becomes weaker and unable to walk. Suddenly, after having perhaps (though not at all as a rule) complained of slight cramping pains in the muscles of the legs, the patient goes apparently to sleep, and, if observed shortly after, is found in a state of profound coma with pupils dilated, gently breathing in slight gasps; body and extremities cold, pulse weak or not at all felt."

Notice here the anæmia, the sudden adynamia,—the patient becomes so weak as to be unable to walk,—the hypothermia, and coma.

In the Haiderabad epidemy of 1843, the fever frequently commenced with vomiting of blood, always of bilious matters, and sometimes terminated by a discharge of blood by the anus. Profuse epistaxis attended it. The disease generally proved fatal by coma, and at a late period by dysentery supervening on a debilitated constitution.

Of the Gazeepur epidemy of 1859, Dr. Garden says: "The patients, after being ill a day or two, were semi-comatose, or entirely insensible, but not delirious; the pulse small and fluttering, and almost imperceptible."

In 1869 an epidemy of fever in the Gangetic valley presented the following characters: "Loss of appetite, pains in the limbs, listlessness or languor for a few days, followed by fever, which lasted for two days, when the patient was left in a cold sweat, and in a state of perfect exhaustion, which, unless stimulants were given, ended in death. Affections of the lungs and copious bleeding from the nose were also attendant symptoms of the disease." Here, again, asthenia, algidity, and hæmorrhages were the dominating features of the disease.

In the Burdwan epidemy, we are told that there were "two or three days' lassitude, loss of appetite, and malaise, followed by fever, without any cold stage; the skin was hot, the head heavy, the bowels confined. Sometimes there was nausea, oftener not. The patient

was stupid and drowsy, with intense prostration and loss of muscular power from the first. Death took place by coma within from three to ten days."

It will be seen that in this fatal fever adynamic symptoms were the most prominent.

A very remarkable, and perhaps the most fatal epidemy of malarial fever on record, occurred among the marines landed at Edam Island, in the Batavian Roads, at the beginning of the present century. Those who remained on board entirely escaped. Every one who landed (seventy-six in all) took ill with the fever, and *at least* sixty-six of these died. The symptoms simulated in many cases malignant yellow fever, often with black vomit.

The principal symptoms were: giddiness, cold chills, vomiting, supraorbital and epigastric pain. Frequently the patient fell down insensible during the paroxysm, his body covered with cold, clammy sweat, except at the pit of the stomach, which always felt warm to the hand. On recovering from this state, there were headache, oppressed breathing, sense of internal heat about stomach and præcordia, while the lower extremities were not unfrequently covered with cold sweats. Eyes protruded, countenance flushed, retching and vomiting of bilious matter, abdomen tense and full. As the disease progressed, the skin often turned bright yellow; in others it became of a leaden colour; sometimes there was vomiting of black bilious stuff, like grounds of coffee. The pupil of the eye was often dilated, and would not contract to light; in other cases there was great intolerance of light. Low delirium was pretty constant from first to last; in some there was furious delirium. Many of the patients were comatose from the first; in others the fever was ushered in with convulsions, delirium, and cold sweats, without any preceding heat of the surface, except at the pit of the stomach, which, as we have said, was burning hot to the touch. Subsultus tendinum often attended the forms with low and high delirium. Deafness was very common. Hæmorrhages from nose and mouth occurred in two cases; the blood did not coagulate, but tinged the linen yellow. Locked jaw also appeared in two cases. Some patients on shore were carried off in eighteen, twenty-four, thirty, and forty-eight hours, but those who were removed on board not till after as many days.[1]

The Chronic Infection.—Occasionally, as we have seen, the patient very rapidly becomes cachectic. Cases, indeed, occasionally come under observation in which anæmia and anasarca appear

[1] Johnson, *Influence of Tropical Climates.* London, 1827. The original history of this disastrous expedition, written by Surgeon Shields, is here given.

at the very beginning of the fever. The following chart (Fig. 20) exhibits the course of a case of this kind observed by Dr. Pellereau, which proved fatal on the fourteenth day. The fever followed the tertian type. More frequently the acute cachexia follows after one or two paroxysms of fever. Cases, however, of this acute kind are only met with in intensely feverish localities or in epidemic seasons.

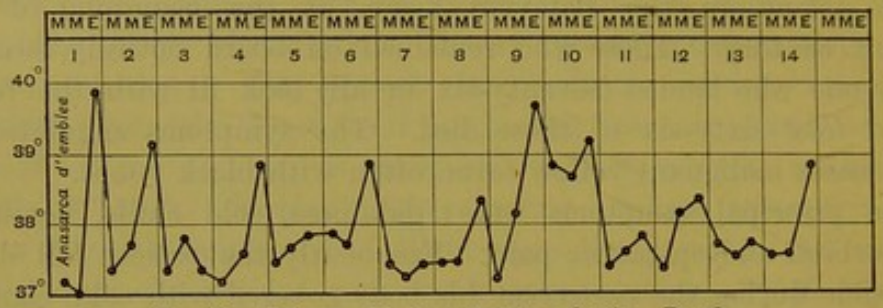

FIG. 20.—Primary febrile malarial cachexia, Death.

In most instances the evolution of the chronic infection follows somewhat this course: After having experienced an attack of malarial fever, in some of its varieties, followed directly or after a time by a relapse either of the remittent or intermittent form, the patient recovers; or, on the other hand, if compelled to reside in a malarious region, he begins to suffer from relapses appearing at irregular intervals. These febrile recrudescences are often determined by a chill, fatigue, exposure to the sun, or by some irregularity in diet. Frequently they arise without any apparent exciting cause. The relapses are irregular in type, in duration, and in the intervals at which they appear. They may be remittent or intermittent, and the type changes in the same and in successive paroxysms. Even if left untreated they often subside spontaneously within a longer or shorter time; but, on the other hand, they occasionally, in these circumstances, show a tendency to run on interminably, as in the case represented in the chart on the following page (Fig. 21). These febrile attacks are accompanied with a feeling of weight, tension, or pain of the spleen, with an enlargement which subsides, but does not disappear, during the intervals of apyrexia. The liver is frequently found to be enlarged; and there may be a dull pain, or a sense of heaviness in the right hypochondrium. During the febrile relapses the urine is high coloured, rich in urea, and, according to Kelsch, it often contains hæmoglobin. The appetite is frequently bad, the digestion slow or painful, and constipation alternates with simple looseness or with bilious diarrhœa.

In proportion as the relapses are frequent and severe, the health of the patient suffers. A severe febrile recrudescence leaves

the patient weak and anæmic; but if the intervals between the relapses are considerable, he regains his strength and colour.

Some patients suffer during the apyrexial intervals from intermittent neuralgias—chiefly of the supraorbital or infraorbital branches of the fifth pair; from intermittent attacks of partial paralysis, anæsthesia, asthma, palpitations, epistaxis, hæmoptysis, or hæmatemesis, along with various intermittent disturbances of nutrition, such as coryza, bronchitis, iritis, etc. These anomalous disorders of sensory, motor, vasomotor, or trophic nerves, mainly occurring in cachectic patients, and not accompanied with marked pyrexia, have been termed masked fevers, or *febres larvatæ*.

From this condition the patient may entirely recover, when he can be removed to a healthy locality. Occasionally, indeed, this state of things may continue for years without any notable change taking place in the patient's condition. In the sub-marshy plains of Vonozongo, on the table-land of Madagascar, at an elevation of about 4000 feet, where malaria in a mild form is endemic, the whole population of some districts suffers from chronic malaria. I have seen patients with indurated spleens stretching across the abdomen to the right iliac crest, pale, thin, and breathless, and suffering from occasional relapses at certain seasons, continue for year after year in the same state, neither improving nor perceptibly losing strength, incapable of great exertion, yet able to move about and look after their affairs.

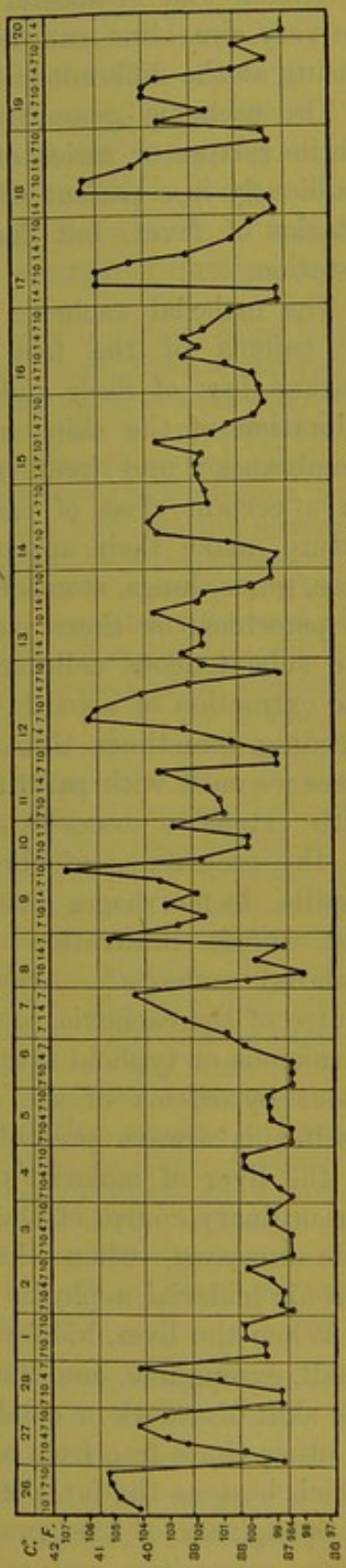

FIG. 21.—Relapses of chronic malarial fever, not treated with quinine.

In most cases, if the patient remains subject to the malarious influence, a grave form of cachexia develops either at an earlier stage, when it is more or less directly the result of the

febrile relapses, or at a later period, when it is associated with functional and structural disease of the liver, spleen, and kidneys. No very clear line can, however, be drawn between the symptoms arising at the different stages of the malady.

The cachexia generally declares itself as a sequel to a severe febrile relapse or series of relapses. Occasionally it develops more insidiously in a patient whose health has been impaired by previous attacks of fever, but independently of any fresh febrile manifestation.

The malarial cachexia is characterised by extreme hydræmia; by œdema of the feet and face, with or without ascites; by marked loss of flesh and strength; by a pale, earthy, or bronzed coloration of the skin; an exsanguine state of the visible mucous membranes; and frequently, in old standing cases, by jaundice. In a certain class of cases hæmorrhages, often of an intractable nature, make their appearance. There may be bleedings from the nose, gums, lungs, stomach or bowels, or into the skin in the form of petechiæ; or there may be more extensive extravasations into the subcutaneous cellular tissue. The simplest operation, such as the extraction of a tooth, will, in such cases, give rise to obstinate bleeding, sometimes threatening the life of the patient. In other cases we meet with parotitis, with gangrene of the mouth and cheeks, with extensive abscesses or carbuncles. Sulzer has drawn attention to the occurrence of chronic optic neuritis, and melanosis of the papilla, hæmorrhages into the peripheral portions of the retina, and diffuse infiltration of the vitreous in those suffering from malarial cachexia.[1] Attacks of fever may supervene during the course of the cachexia, occasionally terminating fatally by developing pernicious or typhoid symptoms. Death, in other cases, is brought about by œdema of the lungs, pneumonia, dysentery, or diarrhœa. Ascites is always present in advanced stages of atrophic cirrhosis of the liver of malarial origin; while albuminuria, which is rare in the ordinary course of the cachexia, is met with, along with intractable anasarca, when the cachexia is complicated with acute or chronic malarial nephritis.

When the liver, kidneys, and heart are atrophied, the liver being small, exsanguine, and chocolate-coloured, the patient becomes weak, the skin of a dark or earthy hue, the urine is sometimes albuminous, at other times free from albumen, and there is frequently dysentery, which hastens the fatal termination.

Sponginess of the gums, purpura, hæmorrhagic blotches on the lower extremities, and epistaxis, in no wise connected with scurvy,

[1] *Klin. Monatsbl. f. Augen.* July 1890.

were not uncommon in the cachexia from which the troops, who were invalided from Cyprus in 1878–79, suffered.

The order in which the symptoms appear, and the way in which they are combined, are never precisely the same in any two cases. The milder form in its daily varying phenomena will be best illustrated by a *résumé* of the symptoms appearing during a single episode in the evolution of the cachexia, in a case which came under my observation. Incidentally, it exhibits a troublesome result which occasionally follows an overdose of quinine.

A. B., aged 35, had indulged rather freely in spirits, without being intemperate. He had contracted fever while in India, where, about a year before he came under my observation, he had been laid up for six months in hospital with fever, enlargement of spleen and ascites, for which he was tapped more than once. After he came to Mauritius, he experienced several attacks of fever about Christmas 1877. Perhaps he had contracted a new infection, for at that period fever was epidemic in the colony. The fever proving obstinate, and the patient's condition becoming threatening, quinine was administered in doses of 80 grains in twenty-four hours, for two days in succession, with the result of temporarily arresting the fever, and at the same time producing complete amaurosis, which lasted for three days. The diarrhœa still persisted, and after a week the fever relapsed, with delirium, great prostration, and profound anæmia. The patient, on the second day of the relapse, fell into a semi-comatose state, during which he passed his motions in bed. He could still swallow food when introduced into his mouth. His pulse was weak and fast, but steady and regular, which was about the only favourable sign in the case. Revulsives were applied to the extremities, blisters to the temples and the nape of the neck, and liquid extract of cinchona and astringents were given to control the fever and diarrhœa. After seeming moribund for two days, the coma disappeared, and, although unable to sit up, he was lifted into a carriage and removed to a healthy district. In a short time he was able to get out of bed. The diarrhœa and fever had ceased, and he was thought to be convalescing satisfactorily. But soon he began to complain of pain in the spleen, with loss of appetite. The urine, formerly abundant, became scanty but not albuminous; there was extreme debility and anæmia, but little or no fever. Within a few days anasarca and ascites set in, and the patient's condition seemed as hopeless as ever. A plaster of ammoniacum and mercury was placed over the enlarged spleen, and a mixture containing 3 grains of quinine, 2 grains of sulphate of iron, combined with small doses of the sulphates of potassium and magnesium were given four times a day. The dropsy diminished day by day, and in about six weeks the patient was able to resume his work as a sailmaker.

Analysis of Symptoms.—The febrile relapses occurring during the earlier stages of the chronic infection are attended with the usual destruction of red corpuscles, but these are renewed to some extent during the apyrexial intervals. The corpuscles are always reduced below the normal figure, and, being deficient in hæmoglobin, are paler than natural. The white corpuscles are also diminished in number, although a temporary increase is observed

during the febrile relapses. The products of the corpuscular destruction are rapidly disposed of by the spleen, liver, and kidneys, which are still comparatively healthy. We have thus a certain degree of anæmia and debility, without any grave cachectic symptoms.

In the more advanced stages the anæmia becomes more intense. The corpuscles, which, in health, number about 4,500,000 per millimètre cube, may fall to 1,000,000, or even to 800,000. The physical characters of the blood become visibly altered. On drawing a drop from the finger it is seen to be watery. The colouring matter rapidly separates from the serum and subsides as a brick-coloured mass. The altered constitution of the blood impairs the nutrition of the capillaries, and this, in conjunction with the hydræmic state of the fluid, gives rise to the anasarca, which, along with the anæmia, is the most striking and constant feature of the cachexia. The same changes probably contribute largely to the occurrence of the hæmorrhages. The icterus depends upon the stasis of the inspissated bile in the minute ducts. The pain or tenderness complained of in the spleen and liver are mainly due to congestion, which seems to give rise to, and to be relieved by, the diarrhœa. The pain is, in some instances, however, the result of perisplenitis or perihepatitis.

During the febrile relapses, the red corpuscles are found to be invaded by parasitic bodies of the same nature as those observed in the acute disease, and pigment-bearing white cells are, in these circumstances, to be met with in the spleen and liver. In the advanced stages of the cachexia, the spleen, liver, and kidneys and other organs become infiltrated with yellow pigment, which, by impairing their functions, interferes with the elimination of the products of blood and tissue changes, and probably hinders the renewal of the elements of the blood, and thus intensifies the cachexia. But how far the final cachexia is to be ascribed to the direct action of the parasites is still doubtful.

Complications and Sequelæ.

Dysentery.—We have already noticed the dysenteric form of malarial fever, in which the bowel symptoms accompany the febrile paroxysm, and disappear during the intermission. The dysentery is, in such cases, a part of the fever, and not a complication.

But dysentery has been observed to follow, and apparently to be caused by, the fever. The troops who had suffered from malaria on the mainland of Mauritius, during the epidemy to which we have alluded, were sent to Flat Island, a few miles out at sea, and healthy.

Here they were attacked with a form of dysentery which is thus described by Power: "The stools were simply of thin, smoky, dark fluid (disintegrated blood and water); no sloughs until some time after the commencement of the disease, and not necessarily then, with no trace of feculent, or, indeed, of any solid matter; great depression; tendency to coldness of the body, but mind quite clear. After death the lesions were either total sloughing of the whole of the internal coats of the larger intestines, or merely a prominent state of all the glands. In this form of dysentery, ipecacuanha was of no avail; but large doses of the tincture of the perchloride of iron succeeded." This seems to me to be the form which has the best claim to be called malarial dysentery.

Fever, again, may be complicated with dysentery; the two maladies attacking the patient at the same time, without our being able to say that the one disease is the cause of the other. The influence of dysentery on the course of the fever is generally, although not always, to aggravate it, and to render it more formidable. The existence of dysentery does not in many cases prevent the usual complete intermission of the fever taking place; but it has been observed, in other instances, to convert an intermittent into a remittent fever.

Pneumonia.—A *febris intermittens pneumonica* has been described, in which the symptoms—objective and subjective—of pneumonia appear during the paroxysm, and abate on the setting in of the sweating stage, to recur with the succeeding paroxysm. The physical signs of the exudation remain stationary during the intermission, to increase during the paroxysm. This, which is rather a form of the fever than a complication, is rare and dangerous.

As a complication (properly speaking) of malarial fever, or of the cachexia, pneumonia is exceedingly common, especially during the cold season, among the native races. The disease is insidious in its onset. The sputa are less viscid, and the pain in the chest less marked than in ordinary pneumonia. As both lungs are frequently attacked, the dyspnœa is usually rather severe; and this and the blood-tinged sputa ought to arouse suspicions, and lead to an examination of the chest. Moore has observed profuse hæmoptysis occurring suddenly and proving fatal in malarious subjects with congestion of the lungs.[1]

Other Complications.—Venous congestion of the papilla and retina, and extensive hæmorrhages in the neighbourhood of the papilla and macula, are met with in the course of acute febrile attacks.

[1] *Indian Annals*, 1867.

Temporary aphasia, occurring during the febrile paroxysm, is met with, but it is rare. Partial paralysis or anæsthesia of different parts of the body have also been occasionally observed. Periodic amblyopia, without any ophthalmoscopic changes, has been observed by Sulzer.[1] Browning has related the history of some cases of bilateral paresis of the lower extremities in children, apparently of malarial origin, and cured by quinine and arsenic.[2] Cases are also on record of complete malarial paraplegia, and of intermittent malarial paraplegia. Gore notices the tendency of fever on the West Coast of Africa to cause spasm and irritation of the neck of the bladder.[3] Ovaralgia of malarial origin has also been observed.

Diabetes, although a rare result of the malarial infection, does occur. Frank found that almost all the diabetic patients he had met with (fifteen in all) had suffered from severe intermittents. Sydenham also notices diabetes as a sequel of ague. Endocarditis has been observed to occur rather frequently in persons who have suffered from ague, but its dependence on the malarial infection has not yet been satisfactorily proved.[4]

Insanity is to be reckoned among the rarer complications or *sequelæ* of malaria. Sydenham was the first to observe its occurrence in malarial fever. "It is," he says, "a form of mania peculiar and *sui generis*. It occasionally follows long agues, especially if they be quartan."[5] It will have fallen to the lot of most who have had experience of mental diseases in the tropics to confirm his observations.

Rupture of the spleen, frequently the result of some trifling accident, but occasionally spontaneous, deserves mention as being of vast importance in malarious countries from a medico-legal point of view. It generally occurs at a somewhat advanced stage of the infection; but it should not be forgotten that it may also happen during the acute stage. Indeed, one case has recently been recorded in which spontaneous rupture occurred within seven days of the appearance of the fever.[6]

Pathological Anatomy.

The Acute Infection.—*Melanin.*—This pigment is of a dark-brown, reddish-brown, or black colour, and occurs as rounded granules, or extremely fine rodlets, either in a state of fine division

[1] *Klin. Monatsbl. f. Augen.* July 1890.
[2] *Internat. Journ. of Med. Sciences*, December 1891.
[3] *Med. Hist. of West African Campaigns.* London, 1876.
[4] See an article, containing a condensed bibliography of the subject, by Fisher in *Lancet*, April 1, 1893.
[5] *Med. Observations*, chap. v. par. 53.
[6] *Lancet*, December 24, 1892.

or agglomerated so as to form larger irregular masses. This substance, which is derived from the colouring matter of the blood, resists the action of mineral acids, even when concentrated or boiling; but it is acted on by alkalis, which change its colour to a light brown or yellow, and it is readily dissolved by ammonium sulphide. If it contains iron, this metal is not indicated by the usual tests (Kelsch).

Malarial melanin[1] is probably formed from the hæmoglobin contained within the corpuscles through the agency of the malarial parasites, and is thus to be regarded as, in a sense, a specific form of pigment. Hæmoglobinæmia, whether arising as the result of disease or artificially produced, does not give rise to melanæmia. However the pigment may be formed, it undoubtedly originates within the circulation, and is generally found within the blood vessels included in white cells or in the endothelium of the capillaries. It is met with in the proper tissue of the spleen and the medulla of the bones; in other organs, such as the liver, brain, and kidneys, it is retained within the vessels of the part.

Yellow Pigment is found infiltrating the cellular elements of the tissues of patients who have succumbed to the acute or chronic form of the infection. It is not peculiar to malaria, but is met with in any condition which gives rise to extensive destruction of the red corpuscles, with discharge of hæmoglobin into the blood plasma, being specially abundant in pernicious anæmia. The hæmoglobin thus set free appears to be transformed into yellow pigment by the protoplasm of the cells of the tissue in which it is deposited. It is met with in the form of exceedingly fine or of somewhat coarser granules, or as larger round or irregular masses, insoluble in water or alcohol, and not affected by acids or potass. It always contains iron; but when of recent formation, this metal is in a state of combination, which does not permit its detection by reagents; the older formed pigment darkens or becomes black when treated with ammonium sulphide.

The Blood.—After death, from the acute infection, the colour of the blood is usually natural to the naked eye; rarely it appears dark from the presence of pigment. The number of the red corpuscles is always diminished; they are often reduced to a half or even to a fourth of their normal number. They are found in some cases to be swollen and pale; in others, small, and of irregular outline; frequently they appear to be normal as regards size. In most instances, when death has resulted from an attack of grave or pernicious fever, more or fewer of them will be found to contain endo-

[1] The term melanin might be conveniently restricted to denote the black pigment of malaria.

corpuscular amœboid bodies, pigmented or non-pigmented, and are often wrinkled, smaller than normal, and of a dark yellow colour. After death from a pernicious attack, the leucocytes are generally found to be increased in number; in other circumstances their number may be normal or diminished. More or fewer of them will be seen to contain granules of black pigment. Large mononucleated and polynucleated white melaniferous cells are quite abundant in the blood of the vena porta and splenic vein. Besides these melaniferous leucocytes, the other parasitic bodies, spherical or crescentic, which we have already described, with more or less free pigment, will generally be found in the blood.

The Spleen.—The spleen is always, perhaps, more or less enlarged. If in some cases it is found of the normal size and weight, this is no proof that it was not enlarged during life, for Hunter has observed that a contraction of the organ sometimes takes place immediately after death.[1] Its weight, which is normally about 6 oz., is often found to reach 12, 24, or even 30 oz. Its capsule is thin, and easily ruptured. The colour of the organ varies from a dark red to a dark brown or black tint. Its consistence is always diminished, and it is frequently reduced to a soft pulp or a jelly-like mass.

On microscopic examination, the venous sinuses are seen to be greatly dilated, and the pulp tissue is found to be filled with an enormous number of red corpuscles, leucocytes, and phagocytes, separating and breaking up the elements of the pulp. It is indeed the great number of phagocytes scattered over the field of the microscope that attracts attention. Many of these contain granules of black pigment, some of them masses of yellow pigment, while others, again, contain red corpuscles, which have lost more or less of their colouring matter, and within which are occasionally to be seen amœboid bodies, pigmented or non-pigmented. Bignami has observed segmented bodies, in the characteristic form of rosettes, contained within the phagocytes. When the patient has died of a pernicious attack, a considerable number of the red corpuscles are often found to contain parasites, which are, as a rule, pigmented. Two or three of these may even be found within a single corpuscle. Alongside the altered blood cells and leucocytes there will often be observed extra-corpuscular parasitic bodies—spherical, crescentic, oval, or fusiform, with granules of free pigment. The blood of the splenic vein is particularly rich in pigment and pigmented bodies. The Malpighian corpuscles and the fibrous trabeculæ are generally free from pigment, and otherwise normal.

The Liver.—The size and weight of the liver are usually only

[1] *Lancet*, Dec. 17, 1892.

slightly augmented; but in the Mauritius epidemy of 1867, enlargement was usually well marked, the organ sometimes attaining nearly double the normal weight. Its consistence is normal or somewhat diminished. It is frequently of a dirty brown colour; but if the hyperæmia has passed away, it often presents a chocolate or slaty hue. The gall bladder and bile ducts are distended with thick bile. The vena porta and the interlobular veins are generally seen to contain free pigment and pigment-bearing leucocytes, macrophages, and free parasites. The accumulation of pigment in the interlobular veins is sometimes so marked that the periphery of the lobules is surrounded by a distinct black zone. The intralobular capillaries are dilated, their endothelium swollen and pigmented, and in most cases they contain numerous pigmented bodies, similar to those which we have described as occurring in the spleen. These may be, indeed, so numerous as to occlude the capillaries. Exceptions, however, appear to occur in which pigment may be absent. Bignami found the liver in a fatal case of the algid pernicious form to be exsanguine and free from any trace of pigment. It never happens that the black pigment is found within the hepatic cells; these, however, are not unfrequently more or less charged with yellow pigment. In many cases the hepatic cells appear healthy. In pernicious cases they have been found in a state of cloudy swelling, in some places atrophied, and particular groups of cells necrosed. The condition of the red blood corpuscles found in the liver varies greatly in different cases. Sometimes the greater part of them are normal, in other instances a considerable number of them contain parasites. The connective tissue of the organ not unfrequently shows signs of commencing interstitial hepatitis.

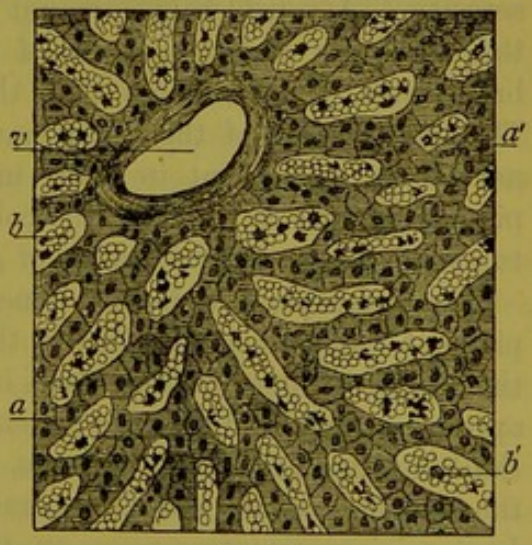

FIG. 22.

a, *a'*, Hepatic substance.
b, *b'*, Capillaries containing blood corpuscles and pigmented elements.
v, Central vein of lobule.

Fig. 22 (after Laveran) shows the appearance presented by a section of the liver in a case of pernicious fever (170 diameters).

Kidneys.—The kidneys are usually normal in weight and consistence, but are slightly enlarged. In the hæmoglobinuric form, however, they are augmented both in size and weight. In ordinary and recent cases the colour of these

organs may be quite normal, or there may be brown or slaty patches on the surface or in the cortical substance. In the hæmoglobinuric form we meet with subcapsular ecchymoses and interstitial hæmorrhages into the cortical substance, or with the discolorations which we know to result from these conditions.

The small vessels and capillaries are frequently found to contain pigmented bodies, but seldom in large numbers. The glomeruli are often distinctly pigmented. Sometimes white pigmented cells, and more rarely endo-globular parasites, are seen in the small vessels on a level with the Malpighian corpuscle, or even in the capillaries of the corpuscle itself. The epithelial elements of the convoluted tubes are in recent cases healthy; in those less recent, they are swollen and cloudy, the tubes themselves containing hyaline casts, charged with yellow pigment in a fine state of division, or in larger granules; and this obstruction of the tubules, both in their cortical and pyramidal portion, with yellow pigment is specially marked when death has resulted from the hæmoglobinuric form of fever. There is often a slight increase in the connective tissue, if the disease has been of some standing. In cases of pernicious algid fever, Bignami has found a true glomerulitis, with a scanty albuminous exudation.

The Brain and Nerve Centres.—When the patient has died of a comatose attack, the meninges are usually observed to be deeply congested, and the meshes of the *pia mater* to contain an opaline serosity. A considerable amount of serum may also be found in the ventricles and at the base of the brain. The substance of the brain often presents more than the usual number of bloody points. The grey matter of the brain may appear, to the naked eye, to be normal in colour, but in many instances it is more or less deeply pigmented, and in this case the coloration may, or may not, be found to extend to the central masses of grey matter in the brain and cord.

A microscopic examination generally reveals the presence of free pigment and pigmented bodies in the smaller vessels, and especially in the capillaries, even in those cases in which to the naked eye the brain appears normal. Where the patient has succumbed to an algid attack, the brain has been found anæmic, alterations in the endothelium of the vessels absent, and the parasitic forms in the blood scanty. In pernicious comatose cases, on the other hand, the capillaries in particular areas may be blocked by pigmented bodies, while those of other areas are comparatively free. As a rule, in cases of pernicious cerebral attacks, many of the red corpuscles in the vessels of the brain contain amœboid bodies. Speaking of the appearances met with in such cases, Bignami says: "The cerebral vessels are usually so rich in parasites, that it is difficult or impossible in some capillary

areas to find a single red corpuscle of a normal appearance. In some instances these endo-corpuscular parasites are exclusively non-pigmented, in others pigmented; frequently both kinds are met with in the same case. The endothelium of the capillaries is often swollen and pigmented, and shows signs of fatty degeneration."[1] Some of these appearances are represented in the accompanying figure (Fig. 23), after this author.

The Lungs. — The lungs to the naked eye generally appear healthy, or, at most, show signs of hypostatic congestion; occasionally we meet with signs of lobar or lobular pneumonia. Microscopically, the small vessels — arterial and venous—and the capillaries of the alveoli are seen to contain phagocytes, but of a smaller size than those met with in the spleen and liver, in which are included pigment and red corpuscles. The phagocytes are specially numerous in the small veins (Bignami). Pigment-bearing leucocytes and pigmented bodies are also present in the vessels.

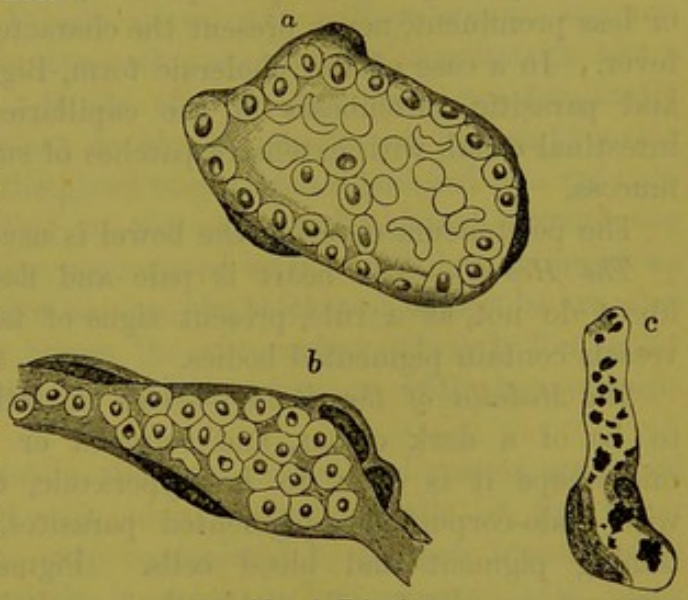

FIG. 23.—$\frac{1}{12}$ Zeiss immers.

a, Transverse section of cerebral vein; parasite-bearing corpuscles lying against the wall of the vessel.
b, Cerebral capillary, in case of pernicious comatose, all the corpuscles containing parasites.
c, Capillary containing free pigment.

The red corpuscles are found to be invaded by the amœboid forms met with in the blood of other organs; but the number of corpuscles containing parasites, compared with those that are normal in appearance, varies greatly in different cases. The alveoli are free from parasites and macrophages. When chest symptoms have been present during life, and sometimes when no such symptoms have been observed, the alveoli may be filled with serous, more rarely with fibrinous, exudations, or they may be the seat of hæmorrhagic extravasations.

Intestinal Canal.—In the cerebral forms of attack the mucous membrane of the stomach and the large and small intestine often appears healthy to the naked eye; and the same is not unfrequently the case even when the patient has died of the algid form

[1] Bignami, *Ricerche Sull' Anatom. Patolog. delle perniciose.* Roma, 1890.

of fever. But in many cases of the algid and choleraic forms the mucous membrane, particularly that of the stomach and small intestine, is congested, of a reddish-brown colour, and small hæmorrhagic spots may be noticed here and there. The solitary glands of the small intestine are often somewhat prominent. Unless when enteric fever has complicated the malarial malady, which it not unfrequently does, Peyer's patches, although they may be more or less prominent, never present the characteristic lesions of typhoid fever. In a case of the choleraic form, Bignami found pigmentary and parasitic thromboses of the capillaries of the stomach and intestinal canal, with extensive patches of superficial necrosis of the mucosa.

The peritoneum covering the bowel is usually healthy.

The Heart. — The heart is pale and flaccid, but the muscular fibres do not, as a rule, present signs of fatty degeneration. The vessels contain pigmented bodies.

The Medulla of the Bones. — The medulla is frequently found to be of a dark colour, and softened or diffluent. Under the microscope it is seen to be hyperæmic, the small vessels filled with endo-corpuscular pigmented parasites, and macrophagi containing pigment and blood cells. Pigmented bodies and free pigment are also found outside the vessels in the medullary tissue itself.

The Chronic Infection. — *The Blood.*—During the febrile relapses which mark the early stage of the chronic infection, endo-corpuscular and extra-corpuscular parasites and pigment-bearing microphages and macrophages, such as occur in the acute disease, are to be met with. The crescentic bodies are of rather frequent occurrence in this stage of the malady. In the advanced stages of the cachexia, parasitic forms may be entirely absent. The mass of the blood is reduced, and the numbers of the red and white cells diminished. In some cases, instead of 4,500,000 red corpuscles per millimetre cube, the number falls to 1,000,000, or even less. The red corpuscles are swollen and pale, showing a deficiency of hæmoglobin. The leucocytes are diminished in number both absolutely and relatively to the red corpuscles, but during the recrudescences of the fever their number suddenly undergoes an augmentation (Kelsch). The serum of the blood is watery, and it is probable that the albumen is proportionally less than in health.

The Spleen.—This organ is always greatly enlarged. In old standing cases it frequently stretches across the abdomen to the right iliac crest. It is generally indurated, its capsule thickened,

often with white patches or fibrous deposits of varying size, indicating previous attacks of perisplenitis. But it should not be forgotten that the enlarged spleen of chronic cachectics, especially in those living in intensely malarious countries, may be softened, with extravasations of blood in its substance, and the capsule so thin that spontaneous rupture occasionally takes place. The indurated state of the gland is everywhere that which is most frequently observed in chronic cases, and I have never met with a spleen at once enlarged and softened in the cachexia occurring in countries where the infection is mild. Bands of false membrane are frequently found attaching the spleen to the diaphragm or to neighbouring organs. The colour of the gland varies from a pale red to a reddish-brown or black, according to the amount of pigment present, the pigmentation being more pronounced when death has followed an acute febrile relapse. On section, the thickened trabeculæ are seen as white bands running across the cut surface, which is frequently mottled with dark grey or black patches, or with hæmorrhagic extravasations.

Examined microscopically, the pulp sinuses and vessels are found to be dilated, engorged with blood, and the walls of the latter thickened. The splenic pulp, compressed by the new fibrous tissue and by the dilated vessels, is more or less charged with black pigment, and many of the cells contain yellow pigment either diffused through their protoplasm, or occurring in larger or smaller granules. When death has resulted from a pernicious attack, the parasitic forms already described will be met with in the vessels and pulp. The thickened trabeculæ are infiltrated with numerous small embryonic cells. The Malpighian bodies are unaltered.

The Liver.—The liver in chronic cases is usually considerably enlarged, congested, of a firm consistence, and of a dark-red colour. The section may be smooth, but in many cases it is slightly granular. The capsule is generally thickened, and occasionally presents whitish patches or fibrous bands, the result of a former perihepatitis.

The small vessels and capillaries are dilated and distended with blood. The hepatic cells, which are often charged with yellow pigment, may be hypertrophied, or show signs of hyperplasia; they are sometimes fatty, and there is generally more or less increase in the connective tissue of the organ. The pressure of the dilated vessels may give rise, in certain spots, to atrophy of the hepatic cells, while, in other places, a different lesion may be observed, the hypertrophied connective tissue narrowing or occluding the vessels. Black pigment and parasitic bodies may be absent from the blood of

the liver, except when death has resulted from, or followed closely upon, an acute febrile attack, in which case the capillaries are sometimes found stuffed with pigmented leucocytes and large white cells, which, in particular areas, may be so numerous as to obstruct their lumen. The bile ducts are patent; their walls healthy or thickened.

A slight but distinct increase in the connective tissue of the hypertrophied liver, scarcely amounting to cirrhosis, is frequently observed. This form of diffuse interstitial hepatitis is occasionally sufficiently marked to be justly described as hypertrophic cirrhosis. The organ is firm on section, and is visibly infiltrated with dense fibrous tissue, not in annular bands surrounding groups of lobules, but diffused throughout the gland. In most of these cases the liver is considerably increased in size and weight, in some instances, however, it is normal in size or even slightly atrophied. In this form of hypertrophic cirrhosis, unlike that described by Hanot,[1] jaundice at an early, or, indeed, at any stage, is generally absent. Only occasionally is a slight icteric tint developed. More frequently we meet with the usual atrophic form of cirrhosis, in which distinct annular bands of fibrous tissue surround groups of lobules.

After death from chronic cachexia and melanæmia, the liver is occasionally found to be atrophied without being cirrhotic. It is small, exsanguine, and of a chocolate colour. There may be a slight increase in the connective tissue, but never to any large extent. The most marked pathological condition to be observed is the occlusion of the capillaries in certain areas, with large white cells, which contain black or yellow pigment. The kidneys and the heart are also found to be atrophied and pigmented.

The small vessels of the liver are sometimes found to have undergone amyloid degeneration.

Kidneys.—The kidneys are generally found to be enlarged and congested, of a dark red colour, smooth on section, and firm in consistence. The small arteries and the capillaries (especially in the pyramids) are dilated and distended with blood containing numerous leucocytes, some of which may be found to carry pigment. The epithelium of the convoluted tubes is swollen, of a brown colour, from being charged with yellow pigment, the diameter of the tubes themselves enlarged. The connective tissue is usually normal, or only slightly increased; but occasionally the kidneys are found to be cirrhotic and atrophied. The epithelium in the early stages of the cirrhotic process may not be much altered, but as the disease advances there is always more or less tubal atrophy and epithelial degeneration.

[1] *Étude sur une forme de cirrhose hypertrophique du foie.* Paris, 1876.

Diagnosis, Prognosis, and Prophylaxis.

Diagnosis.—The discovery in the blood of pigment-bearing leucocytes, of endo- or extra-corpuscular parasites, or of free pigment, during the course of a fever, is conclusive evidence of its malarial character.

No fever is to be regarded as malarial in which melanæmia does not exist in some degree; but pigment cannot always be detected in the peripheral blood of patients who are found after death to have suffered from malarial fever. While the presence of pigment in the blood is thus conclusive as to the malarial nature of a febrile disease, its absence from the peripheral blood, examined on one or two occasions, is not proof of its absence from the circulation.

When, after death, the spleen is found enlarged, diffluent, and pigmented (black pigment), a retrospective diagnosis of malaria is justified, whatever other conditions may have been present during life.

Conversely, if after death from fever the spleen is found to be normal in size, colour, and consistence, and destitute of black pigment, the disease has not been malarious.

The symptoms of simple intermittent fever are so characteristic that it is seldom that any difficulty will arise in regard to its diagnosis. It will be sufficient to remind the reader of the possibility of mistaking urethral fever, the febrile attacks occasionally caused by the passage of gall stones, pyæmia, or hectic fever, for a malarial intermittent. Graves mentions a case of multiple circumscribed abscess of the liver, in which both he and Sir Henry Marsh overlooked the essential disease and treated the patient for ague; and Frerichs tells us that in a case of calculi in the hepatic duct, the symptoms led him to a diagnosis of ague, and the real nature of the disease was only discovered after death.[1] In young children the disease is so irregular in its course that its true nature may be misunderstood both by the parents and physician. Rigors are frequently absent. The child becomes cold, pale, pinched, and drowsy, and as the hot stage sets in, it is seized with convulsions or becomes soporose, and then falls into a perspiration. Occasionally the cold stage may pass unobserved, and the attention of the parents be first directed to the patient when he is convulsed or comatose. When a child living in a malarious country exhibits such symptoms, the malarious nature of the seizure should always be suspected. In children the spleen becomes distinctly enlarged at an early period of the disease, and this will aid the diagnosis.

The gastric and bilious remittent forms of malarial fever may

[1] See also article by Osler on "Fever of Hepatic Origin," *Johns Hopkins Reports*, 1891.

be readily mistaken for simple continued or gastric fever. The temperature furnishes no certain indications, unless, indeed, the remissions are unusually well marked. The locality and season in which the disease appears should here be taken into consideration. If it occurs in a non-malarious locality, and in a healthy season, it will probably turn out to be some form of febricula; if in a malarious locality, and during an unhealthy season, the diagnosis of malaria will probably be justified by its being followed by relapses. If an examination of the blood reveals the presence in the circulation of pigmented or parasitic bodies, all doubts will be at an end.

Insolation, alcoholism, meningitis, cholera, rupture of the spleen, and perforation of the bowel have repeatedly been mistaken for pernicious comatose and algid attacks. Apart from the results furnished by an examination of the blood, it is only by a careful consideration of the history of the case, of the habits and profession of the patient, and of the character of the locality in which he lives, that we will be able to form a judgment as to the real nature of the disease with which we are dealing. When a patient falls down unconscious during a march in a hot sun, or is found comatose in his cabin while passing through the Red Sea, a diagnosis of sunstroke or heat-stroke may be made with little risk of error. But when, as in a case that came under my observation, an intemperate patient, who had been subject to frequent attacks of fever, is admitted into hospital suffering from convulsions, following what was supposed to be a paroxysm of intermittent, apparently induced by insolation on the racecourse, the exact nature of the case will only be cleared up by the results of treatment, or by the autopsy if it should prove fatal. It may be a case of alcoholism, or the result of insolation, or it may be a pernicious attack of malarial fever. If the disease yields rapidly to a full dose of quinine injected under the skin, its malarious nature is rendered, at least, highly probable.

The diagnosis between remittent fever and tropical typhoid fever often presents great difficulties. The presence of rose-coloured spots would, of course, settle all doubts in favour of typhoid, for these are never found in malarial fever; but, then, in tropical typhoid these spots are more frequently absent than present. The presence of a herpetic eruption about the mouth or face is strongly in favour of the malarial character of a disease. Severe bronchial catarrh, on the other hand, is more common in typhoid than in malarial fever. Deafness and muscular trembling, not induced by quinine, appearing in the earliest stage of the disease, are also somewhat characteristic of tropical typhoid.

When the diagnosis is doubtful, it may in some rare cases be

justifiable to withdraw a drop of blood from the spleen by antiseptic puncture. The presence in the splenic blood of pigment and blood parasites is not likely to be wanting if the fever is malarial; they are never present in the blood of those suffering from typhoid fever. The two diseases may, however, coexist in the same individual.

Prognosis.—The considerations which determine the prognosis have reference—(*a*) to the age and habits of the patient; (*b*) to the season and locality; (*c*) to the symptoms present in the individual case.

(*a*) In tropical and subtropical countries the highest malarial mortality falls on infants under one year of age. In the United States more than one-fourth of the total deaths ascribed to malarial fever occurs in children under one year of age. In Bombay the proportion is still higher. The ratio of deaths from remittent fever to the number living at a given age is also highest in infants and in persons over 60 years of age. Although the actual number of deaths from malarial fever at intermediate age-periods is high, the proportion which it bears to the living at these ages is comparatively low. The prognosis of malarial fever at the extremes of life is thus less favourable than at other age-periods.

The habits of the patient materially affect the prognosis. The dissolute and intemperate fall easy victims to the infection. It is doubtful if the habit of opium eating tends to increase the liability to or the danger of the disease. Some even hold it to be prophylactic.

(*b*) In healthy seasons the prognosis is favourable; in epidemic seasons any case may become dangerous, and the prognosis should be guarded.

The same considerations apply to healthy and malarious localities.

(*c*) Algidity, adynamia, drowsiness, vertigo, or fainting occurring in the course of a fever, should always be viewed with suspicion. The ictero-hæmoglobinuric form is never devoid of danger. Guiòl states that out of 185 cases of this form treated in Madagascar, 49 died, or about one in four.

The prognosis in the chronic form depends very much upon the functional integrity of the liver and kidneys. Anæmia and anasarca, clearly dependent upon febrile relapses, are not of grave import; but when they appear at a later stage of the disease, and are the result of organic disease, they are of bad omen.

Dysentery and pneumonia occurring in cachetic subjects are frequently fatal, and always render the prognosis less favourable.

Prophylaxis.—Prophylaxis may be considered with reference —(*a*) to site and season; (*b*) to the measures to be adopted for improving the health-conditions of malarious localities—rural and

urban; (*c*) to the means to be employed in order to diminish individual susceptibility to the infection.

(*a*) In selecting a site for a temporary camp or station, marshy grounds; those which, although superficially dry, have water standing within a few feet of the surface; lands subject to periodical overflow; grounds likely to become temporarily marshy during rains, and the dry beds of summer torrents, should, if possible, be avoided. For permanent stations, camps, or cantonments, still greater care should be exercised in selecting a site. The medical history of a locality should be inquired into. However promising a site may seem, if its history shows that it is malarious, it ought to be avoided. During the insurrection in Jamaica, the position and surroundings of Port Antonia seemed so favourable, that regardless of its previous notorious insalubrity it was recommended as suitable for a permanent military station. When reoccupied in 1866 by the 2nd battalion of the 6th Regiment, the admissions for paroxysmal and continued fevers rose to 1700 per 1000, and the deaths from paroxysmal fevers alone to 25 per 1000; and it had speedily to be abandoned.

The health of the native community should be considered before fixing upon the site of a station. If the inhabitants of a locality are anæmic, and suffer from enlargement of the spleen, the place may be expected to prove highly insalubrious for Europeans.

When an outbreak of fever in a new settlement can be traced to excavations connected with its foundation, there is good reason to hope that after a few years the fever will subside, as has happened in the instance of Hong-Kong.

In malarious countries no unnecessary excavations should be made, and those that are absolutely necessary should so far as possible be carried out during the cold season. When a choice is possible, the healthy season should be selected for explorations and expeditions in malarious countries. Neglect of this obvious precaution has in innumerable instances led to unnecessary loss of life and health.

(*b*) Drainage and cultivation are the only effectual means of banishing malaria from a country. The good results which have been obtained in some malarious countries from extensive plantations of the eucalyptus,—a tree of extremely rapid growth,—is owing to its effects in draining and drying the soil. The sanitation of malarious towns demands, *first*, subsoil drainage to carry off underground humidity; *secondly*, the careful paving or asphalting of streets and courts, and the covering by concrete of the areas on which houses are built, in order to prevent, on the one hand, the saturation and

contamination of the soil by the soakage of rain and waste water; and, on the other hand, the contamination of the air of the town and dwellings by soil emanations.

The very simplicity of these measures often leads them to be ignored in favour of costly schemes of "sanitary improvement" which increase the evils they are intended to remedy.

Although it has not been proved that drinking water is the most common medium of infection, the possibility of malaria being diffused in this way cannot be denied in presence of the observations to which we have referred in a previous section. Instances are not wanting in which, other conditions remaining unchanged, the introduction of an improved water-supply to a fever-stricken village has been followed, not only by a marked improvement in the general health of the community, but by a notable diminution in the prevalence of malarial fever. Whether this result is to be explained by the suppression of a source of the specific infection may, to some, appear open to doubt; but such instances prove, none the less, the importance of a pure water-supply in preventing or mitigating the malarial infection.

(*c*) In travelling through a malarious country it is a safe precaution to make use only of boiled water. Night marches should be avoided, as well as travelling during the warmest hours of the day; and it is important to partake of a light breakfast of tea or coffee before setting out in the morning. It is safer to accept the humblest quarters in a village or town than to camp out in the open. Over fatigue is to be avoided; flannel should be worn next the skin, and wet clothes should be changed as soon as possible. A regular action of the bowels should be insured by diet or medicine, —constipation and diarrhœa are alike to be avoided. Quinine is not an absolute preventive of fever, but its use undoubtedly diminishes the liability to the malarial infection. It should, therefore, be given as a prophylactic to persons who have to traverse, or to reside for a short time in, a malarious country. When it is a question of a few days' excursion up a marshy African river, or of one or two days' sport in a malarious jungle, 20 to 30 grains of quinine should be given daily in divided doses. If the journey through, or residence in, the malarious region extends over a period of two or three weeks, large doses such as these should not be employed. It will be enough, in these cases, to give 2 to 5 grains, three times daily; and these comparatively moderate doses are not without a prophylactic effect. I have never found any good to result from the employment of arsenic as a prophylactic agent. It is right, however, to state that others have had satisfactory results from its

use. Dr. Ricchi's experiments on seventy-eight persons in the malarious district of Bovino in 1883 showed that those who used arsenic as a prophylactic either escaped entirely, or only suffered from slight attacks, while those who did not take arsenic suffered severely.[1] It will, I think, be found that when arsenic acts as a prophylactic it is in preventing relapses of the chronic infection, not in warding off the primary attack.

Treatment.

Symptomatic Treatment.—When the attack is mild and of the intermittent type, and when the season is one in which the fevers are not exhibiting any great intensity, little requires to be done during the paroxysm. In some cases it will be sufficient to order the patient to bed and await its subsidence before having resort to quinine. In most cases, however, something has to be done to relieve the immediate sufferings of the patient. During the cold stage the patient should be warmly covered, and heat or friction applied to the extremities. The patient is to be restrained from satisfying his inordinate craving for liquids; a cup of weak, warm tea is perhaps the best drink he can take.

In the hot stage the patient is to be lightly covered, his body sponged with tepid water, while cool and refreshing drinks may be given in moderation. The headache often complained of during this stage is generally relieved by the application of cold evaporating lotions, or of cloths wrung out of iced water. If the congestion is great, pounded ice may be applied to the head. Waring states that he found the application of hot-water stupes to the head to be more effectual in relieving the violent headache than cold evaporating lotions;[2] but I have had no experience of their use. When the hot stage has passed off, nothing should be done either to check or increase the sweating. It occasionally happens that the vomiting during the cold stage is so severe as to demand treatment. This symptom is frequently relieved by a large sinapism over the epigastrium. When this does not succeed, in the case of adults a small enema, containing 15 to 30 minims of laudanum, may be administered; or 1 to 3 minims of the acetate of morphia (1 in 10) may be hypodermically injected. In children, opiate remedies should be avoided. The vomiting of the hot stage will often be mitigated by the patient sucking small pieces of ice, or by the use of effervescing draughts containing, if need be, 30 to 60 minims of the compound tincture of chloroform. In this stage

[1] *Lancet*, Dec. 10, 1892. [2] *Indian Annals of Medical Science*, Oct. 27, 1853.

effervescing draughts tend not only to soothe the stomach, but likewise to diminish the fever and hasten the sweating stage.

These are the chief points in the management of the symptoms proper to the paroxysm itself; but two questions, relating less to the phenomena of the fit than to the condition of the patient and the after course of the case, often arise at this stage. We have to ask ourselves whether an emetic should be administered, and whether a purgative is to be given? If the patient's stomach is loaded with undigested food, or if there are much nausea and retching, or if it seems likely that we have to do with a bilious fever of primary invasion, an emetic of ipecacuanha, or of mustard and water, at the beginning of the fit is indicated, and is often highly serviceable, reducing the nausea and in many cases mitigating and shortening the paroxysm. When the bark was used for the cure of ague, it sometimes failed to act as an antiperiodic until an emetic was given. Heberden says: "Cortex, quamvis rite sumptus, interdum parum efficax est; quo in casu suspicio erit, ventriculum sordibus onustum vim remedii impedire. Itaque vomere oportet; quo facto, febris non raro cedit." This difficulty is not so much felt now that quinine has been substituted for the bark; but emetics properly used are not without their value in the treatment of malarial fever. They are not to be given, however, as a matter of routine. The mildest only should be employed, and they should not be unnecessarily repeated. Antimonial emetics frequently administered have with good reason been suspected of favouring the appearance of algid symptoms. Their use is, therefore, contra-indicated.

Purgatives may not be required at all. It is never necessary, and in the severe forms of fever it would be highly dangerous, to delay the administration of quinine until a purgative has been given and has acted. As Dr. Boling has remarked, "Cases originally violent, almost invariably die while *preparing* for the quinine, and those of moderate severity become worse under the *preparation*."[1] Yet a purgative is in most cases of undoubted service. After its use, quinine is not only better tolerated, but the curative action of the specific is promoted. This, at least, was the conclusion at which I arrived from a series of observations made in the wards in the Civil Hospital of Mauritius to test the value of the practice during the epidemy of 1879. I found that when a purgative was given at an early stage of the fever, whether of the remittent or intermittent form, quinine acted more certainly and promptly in subduing the disease. In mild cases castor oil, a seidlitz powder, or a dose of Epsom salts will suffice; but in the severer cases

[1] *Amer. Journ. Med. Sciences*, July 1846.

of remittent or sub-continued fever, 10 grains of calomel, followed, if need be, by a saline; or 5 grains of calomel combined with compound jalap powder or rhubarb, will prove much more useful than simple salines or laxatives. Mercurial purgatives are specially serviceable in the bilious remittent form, and, when the bilious disorder persists, their repetition may become necessary. But in most cases, after the bowels have been freely opened, nothing more is necessary than to obviate constipation by the use of any mild laxative.

Attempts have been made to arrest the progress of a paroxysm by a bleeding practised at the commencement of the cold stage. Mr. Twining found that a bloodletting to the extent of 12 to 15 oz. generally prevented the recurrence of the hot and sweating stages; and he believed that this practice tended to obviate ulterior visceral engorgements. As no one at the present day advocates such a practice, it is unnecessary to discuss its merits or demerits.

Opium has been administered during the cold stage with the object of rendering it shorter and milder. Waring, during his practice in Burma, found that, given in a full dose at the commencement of the cold stage, opium had an undoubted influence in mitigating this stage, but that it did not materially affect the after course of the disease.[1] Lind strongly recommends the administration of an opiate during the hot stage; and from his experience of its effects in upwards of 300 cases, he states that it shortens and abates the fit with more certainty than an ounce of bark; that it generally gives a sensible relief to the head, takes off the burning heat of the fever, occasioning a profuse sweat, and often produces a soft and refreshing sleep.[2] But it may be reasonably urged against this practice, that opiates are altogether unnecessary in any stage of mild intermittent, while in severe cases, with a tendency to coma, they might prove dangerous.

Specific Treatment.—When we have to deal with a case of mild fever of the intermittent type, 5 to 10 grains of quinine are to be administered as soon as the paroxysm has subsided. As the disease most frequently follows the quotidian type in warm countries, and may consequently be expected to recur about the same hour on the following day, the dose should be repeated so that 15 to 40 grains be given before the period when the next paroxysm or exacerbation is due. The smaller dose may suffice in countries where the fever is mild, and is often as much as can be well borne by delicate, nervous, or young patients; but in the severer cases

[1] *Loc. cit.*

[2] Lind, *Essay on Diseases of Europeans in Hot Climates*, Lond. 1768, p. 317.

often met with in malarial countries, the larger dose will be required. Several moderate doses, repeated at intervals of from three to six hours during the intermission, is more efficacious than a single large dose. The first of these should be administered as soon as possible after the paroxysm has subsided.[1] For children under 1 year of age, $\frac{1}{3}$ of a grain to 2 grains; in those from 1 to 2 years of age, $\frac{1}{2}$ a grain to 3 grains may be given; for older children the dose will be proportionately greater. These doses are to be repeated, increased or diminished, according to the urgency of the symptoms and their effects on the system.

Children, as a rule, can take comparatively large doses of quinine without suffering from headache, vomiting, or delirium; but, on the other hand, two doses of 4 grains, repeated at intervals of three hours, are reported to have proved fatal in a child of 6 years of age.[2] The effects of the remedy should, therefore, be carefully watched in the case of children.

The first stage of cinchonism is characterised by buzzing in the ears, a sense of fulness in the head, restlessness, and sometimes by vomiting and diarrhœa. Well marked ringing in the ears, with some dulness of hearing, may be accepted as proof that the system is sufficiently under the influence of quinine; and when these symptoms are established it is unadvisable, at least in ordinary cases, to increase the dose, or to repeat it oftener than is necessary to maintain this state of the system. Although enormous doses—in one case an ounce taken at once—have been recovered from,[3] yet serious toxic symptoms have followed the administration of strictly medicinal doses of 10 to 20 grains,[4] so that resort ought never to be had to heroic doses unless the symptoms are urgent.

The object of the treatment we have indicated is to prevent the recurrence of the paroxysm, or failing this, to render the succeeding one milder, if the case is one of frank, or distinct, intermittent; or, if the case be of the remittent form, to reduce the violence of the succeeding exacerbation, or to convert it into an intermittent of the quotidian or tertian type. Should the paroxysm return, or the exacerbation recur, quinine must be repeated in the same, or in an increased dose, according to circumstances, during the intermission or remission, until the paroxysms cease to recur or the fever is subdued. When this result has been achieved, quinine in smaller doses, say

[1] For an interesting inquiry into the period of the fever during which quinine can be most advantageously given, the reader is referred to an article by Golgi in the *Trans. Soc. Med. Chir. di Pavia*, Jan. and Feb. 1892.

[2] *Amer. Journ. Med. Sciences*, April 1847.

[3] *Med. Times and Gaz.* April 23, 1864.

[4] *Bull. de Thérap.* lxiii. p. 234.

from 3 to 5 grains, should be continued for three or four days, after which the liquid extract of cinchona may be substituted for it with advantage on account of its tonic as well as its anti-periodic properties. As the disease in warm countries often shows a decided tendency to relapse about the seventh, fourteenth, twenty-first, or twenty-eighth day, it is prudent to give one or two moderate doses of quinine a day or so in advance of these dates, to prevent relapse.

In the remittent form, when the remissions are distinct but short, quinine may be given in two doses of 15 grains each during the remission; and the first of these should be administered as soon as the fever is reduced, and the skin shows signs of becoming moist.

When the disease approaches to the continued type, the remissions being indistinct or absent, quinine should be given in full doses every six or eight hours, without reference to the slight fluctuations of temperature by which these cases may be characterised. When the fever is mild, we may safely wait for the remission or intermission, which is undoubtedly the best time for exhibiting quinine; but in countries where malaria in its more intense forms prevails, it is safer not to wait for an intermission or a remission, but to give quinine at once in whatever stage of the fever the patient may happen to be when first seen. Should a well-marked remission or intermission subsequently occur, this is to be taken advantage of for repeating the remedy.

In grave and pernicious fevers, certain symptoms, as we have seen, come to the front, menacing the life of the patient, which demand the symptomatic treatment to which we shall presently refer; but it should never be forgotten that these symptoms are, in the main, the effects of an infection for which we have only one specific remedy; and that, whatever may be the symptoms, our first object is to bring the system as rapidly as possible under the influence of quinine. It is in such cases that quinine has occasionally to be administered hypodermically; the administration of the drug by the mouth is, as a rule, to be preferred; but hypodermic injections must be resorted to—(*a*) when the existence of coma or convulsions prevents its administration by the mouth; (*b*) when the vomiting is so severe that it is rejected as soon as it is swallowed; (*c*) when the symptoms are of so menacing a character that it is necessary to bring the system, as rapidly as possible, under the influence of the drug. The most soluble salt of quinine is the acid hydrochlorate, which is soluble in its own weight of water. When this is at hand, it should be preferred; and in malarial countries it would be well to have it in readiness. When the acid hydrochlorate is not to be had, we make use of the sulphate of quinine with the addition of

half its weight of tartaric acid, dissolved in distilled water. Whichever solution is used, it will often be found necessary in grave cases with comatose or algid symptoms to inject a quantity representing 10 grains of the salt. The injection should be made into the subcutaneous cellular tissue of the upper or lower extremity, not into the skin, otherwise sloughing or abscess is apt to occur; and it is better not to inject the whole at one place, but by several punctures at different spots. The solution should be fresh and the needle clean.

Intravenous injections of quinine have lately been suggested and used by Baccelli.[1] He uses the following formula: hydrochlorate of quinine, 1 gramme; chloride of sodium, 75 centigrammes; distilled water, 10 grammes: of this solution he injects 40 to 50 centigrammes into the veins of the neck. Such heroic treatment should certainly be reserved for desperate cases; but its value in some instances is not to be ignored. When quinine can be given by the mouth a dose of 15 to 30 grains is often required in grave or pernicious attacks. Whether administered by the mouth or hypodermically, the doses stated may require to be repeated should the symptoms persist. But when an improvement takes place, more moderate doses should be resorted to, to prevent the recurrence of the attack. In some cases it is convenient to administer the drug by enemata.

In comatose attacks the symptomatic indications require the application of ice to the head, the administration of an active cathartic or drastic, and the use of sinapisms to the extremities. It has been recommended to apply 15 to 20 leeches to the mastoid process when the face is flushed, and there are signs of active cerebral congestion. It is only in cases where these signs are well marked, and when no manifest improvement has followed the administration of quinine and purgatives, that resort should be had to local depletion to the extent and in the manner indicated.

The same line of treatment will be called for in acute phrenetic cases, with a hard pulse, injection of the eyes, and flushing of the face, indicating congestion of the brain. When the delirium is not associated with signs of active congestion tending to coma, then 15 to 20 drops of tincture of opium may be given with benefit. Laveran states that hydrate of chloral has been of great service in his hands in combating the delirium occurring during attacks of pernicious fever, and in delirium arising during the course of simple malarial fever in persons given to alcoholic excesses. Both opium and chloral ought in all cases to be given with great circumspection.

In algid attacks it is necessary to stimulate the patient. Sina-

[1] *Gazzetta degli ospitali*, Feb. 1890.

pisms, turpentine stupes, or rubefacient frictions to the extremities are to be resorted to in order to restore the peripheral circulation; ammonia, ether, or brandy should be given in moderate but frequently repeated doses, along with beef-tea, in order to stimulate the heart's action. If these measures fail, 20 to 30 minims of ether should be injected hypodermically. When diarrhœa accompanies the algid state, camphor with small doses of laudanum should be given along with the other remedies we have mentioned. Convulsive seizures, especially those of an epileptic character, will often yield to 20 or 30 grains of the bromide of potassium, given three or four times a day, in addition to the specific treatment.

Quinine is less to be relied upon in the bilious hæmoglobinuric than in any other of the graver forms of malarial fever, and the reason probably is that this is not a purely malarial disease, and is, besides, a form that occurs most frequently in those who have already reached that chronic stage in which quinine has lost something of its control over the infection. Nevertheless, all who have seen much of the disease in its headquarters advocate recourse in the first instance to quinine administered in full doses, and this I think is the safest practice. The tincture of the perchloride of iron may often be advantageously given alternately with the quinine.

Calomel in 20 to 30 grain doses is generally given at the outset of this form of fever, and it may be repeated in the same or in smaller doses once or twice during its course. Given in this way it is believed to remove accummulated bile, and to permit the liver the more readily to eliminate the hæmoglobin in the circulation. Whatever may be the true explanation of its action, its value as a purgative in this disease, as in bilious malarial fevers generally, is undoubted. I, as a rule, content myself, however, in this form with the use of smaller doses combined with other purgatives. Dry cupping to the loins and warm hip-baths are employed to relieve the renal congestion which frequently occurs during the progress of the fever. It is to be kept in mind that relapses are not uncommon in bilious hæmoglobinuric fever. The patient should therefore be carefully watched during convalescence; the liquid extract of cinchona being administered, the bowels carefully regulated, a mild but nourishing diet prescribed, and *exposure to chills and severe muscular exercise carefully guarded against.*

When quinine is administered by the mouth we, as a rule, use the sulphate, the action of which, in most cases, leaves nothing to be desired. The hydrobromate is to be preferred, however, in the treatment of nervous patients, of pregnant women, of those suffering

from dysmenorrhœa, in those cases in which the sulphate causes intense headache, and for children. The valerianate has been recommended as a good preparation for girls approaching puberty. The other alkaloids of cinchona and their salts are so inferior as febrifuges to quinine, that they should never be employed in any of the severer forms of the disease. The mixed alkaloids sold under the name of quinetum are a useful medicine, which may be given instead of quinine in all but the grave and pernicious forms.

In the treatment of the acute infection there is no substitute for quinine. Dr. Prout has shown that antipyrin, given in doses of 30 grains during the paroxysm or exacerbation, and repeated, if necessary, in an hour or an hour and a half, will relieve the headache, the oppression, and the pains in the limbs from which most patients suffer, and rapidly reduce the temperature.[1] Used in this way, or preferably in 5 grain doses, repeated if necessary, antipyrin may be of service. It does not dispense with the necessity of giving quinine, but it brings the patient into a condition in which quinine may be given with advantage. But valuable as this drug may be in mild cases, no remedy but quinine has any claim upon our confidence in the graver forms of the disease; and in these its administration should not be delayed in order to give any other remedy whatever. I do not think it necessary to inquire into the value of the long list of substitutes for quinine which have been used and recommended for malarial fever. Even their advocates admit their inferiority, and their use is no longer justified by the high price of quinine.

Experiments have shown that while the schizomycetes and algæ are little affected by quinine, the protozoa, to which the malarial parasite is believed to belong, are extremely sensitive to its action. By adding a solution of a salt of quinine to a drop of blood containing the different forms of the parasite, the movements of the flagella cease, and the amœboid hæmatozoa assume their cadaveric forms (Laveran). The effects of quinine upon these parasites is more satisfactorily proved by their disappearance from the blood of fever patients after the administration of a few doses of the medicine. Yet in most instances the fever is arrested for some days before the parasites entirely disappear from the blood. The crescentic bodies are excessively refractory to the action of quinine. The occurrence of relapses after the use of the remedy, proves that in some form or other the parasite may survive its action and remain latent for long periods, and under favourable conditions multiply and invade anew the system. Quinine, so far from diminishing, actually increases, according to some observers, the activity of the leucocytes that

[1] *Internat. Journ. of Med. Sciences*, Dec. 1889.

take so active a part in the destruction of the parasites, and in the removal of the pigment from the circulation, so that Vandyke Carter suggests that the action of quinine might be explained by its stimulating effect on the phagocytes.[1]

Treatment of the Chronic Infection and of the Malarial Cachexia.

The febrile relapses which mark the early stage of the chronic disease are amenable to quinine administered in the ordinary way. In the intervals of fever our principal object must be to maintain and improve the health of the patient by hygienic and dietetic means, and by the administration of tonic remedies. In many cases benefit results at this stage from the use of arsenic, which is often harmful in the acute disease. Five to eight minims of the liquor arsenicalis are to be given after meals, three or four times daily, for a week or ten days, when its use should be suspended for a period, and then be recommenced anew. While the use of arsenic is suspended, the liquid extract of cinchona or the citrate of iron and quinine may be given.

The anæmia and dropsy which follow rapidly on the acute disease, will generally yield to a mixture containing 2 or 3 grains of quinine, the same amount of the sulphate of iron, combined, if the bowels are sluggish, with small doses of the sulphate of magnesia, and from 5 to 10 minims of dilute sulphuric acid. If the dropsy should persist, a teaspoonful of the infusion of digitalis may be given, twice a day, in addition to this mixture, with the best results.

The hæmorrhages which occur in the course of the malarial cachexia require the use of the tincture of the perchloride of iron in full doses, generally with the addition of a few grains of the sulphate of quinine. When this fails, turpentine given in doses of half a drachm every four or six hours will often succeed. The strangury which occasionally follows the use of turpentine readily disappears when its administration is suspended. If there be a scorbutic taint, fresh lemon juice should be given, and the diet carefully attended to.

Recent hypertrophy of the spleen usually disappears gradually under the influence of the remedies indicated for the cachexia. Occurring at a somewhat more advanced stage of the disorder, the application of the biniodide of mercury ointment over the gland is to be resorted to. Maclean recommends that a portion, about the size of

[1] *Op. cit.* p. 159.

a nutmeg, should be rubbed in with a spatula over the swollen organ, the patient exposing the part to the sun's rays, or to the heat from a fire, as long as he can bear the smarting which quickly follows. A few repetitions of this process will generally succeed in effecting a marked reduction in the size of the organ. He adds that this treatment acts just as energetically on enlarged malarial livers. I have frequently followed this plan in hypertrophy of the spleen, and have found it useful in comparatively recent cases. Laveran recommends the cold douche, applied in jets over the splenic region. In more obstinate cases Faradism deserves a trial. But none of these, or any other method with which I am acquainted, will be of much service in the large, old standing, and indurated spleen of chronic cachectics. Localised pain or tenderness at some point of the hypertrophied organ is most readily relieved by the application of a small blister to the painful spot.

In malarial cachexia the essential point is the withdrawal of the patient from the source of infection, by removing him from the malarious locality; and this should be done, if possible, before the health has been seriously impaired. The change should not be made so as to land the patient in an English winter. A sea voyage will often effect a marvellous improvement in cachectic subjects; and a few months' residence in a dry, bracing, equable climate will in most cases suffice to complete the cure. When the liver as well as the spleen is enlarged, a resort to the mineral springs of Carlsbad in Bohemia, Kissingen in Bavaria, or Wiesbaden in Nassau, should be recommended. The particular spring should be carefully selected with reference to the special features of the case.

When dysentery occurs as a complication of fever, a full dose of quinine should be given; and after a sufficient time has elapsed to allow of its absorption, ipecacuanha is to be administered in the usual manner. It may be better, in some cases, to administer the quinine hypodermically, so as not to interfere with the anti-dysenteric treatment. When the dysentery is not a complication, but a direct result of the malarial infection, ipecacuanha may fail entirely to arrest the dysentery. This was the experience of Power in the treatment of the dysentery which attacked the fever-stricken troops in Mauritius during the epidemic fever to which we have already referred. In this instance ipecacuanha was of no service, while the tincture of the perchloride of iron in large doses gave good results. In this form of the disease, a few grains of quinine should be given along with the iron, even if no fever be present, and external derivatives should be sedulously applied, and the strength of the patient sustained by suitable nourishment.

Pneumonia may attack a patient labouring under the malarial infection, or it may occur as a part of the malarial process. In the former case quinine or cinchona is to be given as a subsidiary, in the latter as the main remedy. In both conditions a stimulant and restorative treatment is indicated, and all depressing remedies are to be avoided. Reliance is chiefly to be placed on the use of ipecacuanha wine with camphor and the carbonate of ammonia, along with small doses of the tincture of digitalis if the heart's action is feeble. Large sinapisms should be applied to the chest and back, and repeated frequently, but not so as to cause blistering of the skin. Stimulants will often be necessary at an early stage of the complaint.

CHAPTER V.

TROPICAL TYPHOID FEVER.

BY SURGEON-CAPTAIN H. R. WHITEHEAD, F.R.C.S., ARMY MEDICAL STAFF.

Tropical Typhoid Fever.—Synon. Tropical enteric fever.

Definition.—A continued fever, usually lasting for three weeks or longer, and accompanied by inflammation and ulceration of the agminated and solitary glands of the intestine, with enlargement of the mesenteric glands and spleen.

A rose-coloured eruption and diarrhœa are frequently present.

Geographical and Racial Relations.

Distribution.—Typhoid fever is a disease which seems universal in its distribution. With the diffusion of more accurate knowledge of its course and post-mortem appearances, its recognition has followed in all quarters of the world. Not many years ago the very existence of this fever in India was denied. We now know that it causes more deaths among Europeans than any other disease in that country, and that it exists not only among the European races, but also among the native population. What has happened in India has occurred many times since in the geographical history of this fever. Increased knowledge has shown that it is common in various countries, in which previously it was supposed not to have existed.

The great probability is, that many of the fevers which are now returned in tropical and subtropical countries as "simple continued fever," will be found in reality to be mild or abortive attacks of this disease.

In nearly every part of the world, the prevalent diseases of which have been carefully studied, we gather from the literature of the subject that typhoid fever has been recognised, or that indications

exist to show that it is extremely probable that the disease really does form one of the specific fevers of these countries.

We are not prepared to admit that climate *per se* has any influence on its production. It is met with alike in countries, such as Iceland, the mean temperature of which is extremely low, where it is prevalent and has appeared in epidemics of marked severity, and in every phase of climate, until we reach the intense heat of tropical countries.

Surgeon-Colonel Welch, Army Medical Staff, in a valuable monograph, being the Alexander Prize Essay of 1881, has attempted to group the prevalence of typhoid in the various stations of the British army, and to compare the liability of the troops to the disease in accordance with locality. We are not disposed to accept the conclusion that he arrives at, being more inclined to attribute an increased prevalence and death-rate inversely to the length of residence in a foreign climate, and to the age at which the soldier is subjected to such change of climate. We quote Welch's table, however, as being an important addition to our knowledge of this subject.

Group.	Stations.	Admissions per period per 1000 of strength.	Deaths per 1000 of strength.	Proportion of Mortality to Admissions per 1000 of strength.
Countries approximating to native climate.	Home. Canada and Nova Scotia, Australia, New Zealand, Japan.	·81	·20	257
Mediterranean, subtropical.	Gibraltar, Malta, Ionian Isles, Cyprus.	3·75	1·14	309
Western tropical isles.	Bermudas, West Indies.	7·20	1·85	257
Eastern tropical isles.	Ceylon, Mauritius.	1·54	·90	586
India.	Sea-coast, Plains, Table-lands, Hill stations.	2·41	1·01	421

In taking a general view of the geographical distribution of typhoid fever, we are struck by the diversity of countries, climates,

and races affected. The disease is well known over the whole of Europe, the Mediterranean isles, and Egypt. In India it is accountable, as we have before remarked, for more deaths among the European military population than any other disease. Epidemic cholera causes a greater mortality in a short time, but does not give rise to the same steady annual mortality as typhoid fever.

In Burmah the disease was recognised by Scriven as early as 1853; in Ceylon, by Massy in 1865. The French physicians, at a later period, drew attention to the fact that the disease is common in Cochin-China. Our information about the diseases which exist in the interior of China is meagre, but we know that the Chinese ports are affected by this fever.

Hirsch states that in the Australian continent, Tasmania, New Zealand, New Caledonia, and other islands of the Pacific Ocean, typhoid fever takes one of the foremost places among their acute infective diseases in respect of frequency and malignancy.

From Réunion, Mauritius, and Madagascar, we have reliable information that this disease is met with. The French troops have suffered from it severely in Algiers, and their experience in this respect coincides with our own in India on many material points.

Over the whole of the African continent, wherever Europeans have resided for any length of time, we find the disease noted as present, and the cause of many fatal epidemics.

In the United States it is extremely prevalent, and the reports of the American Civil War furnish us with accurate knowledge of the enormous extent to which it existed among the troops.

There have been frequent outbreaks of tropical typhoid in the West Indies and Bermudas, in Cayenne, Brazil, and other countries in South America.

If we turn to the Army Medical Reports, for a series of years we find that no station at which British troops are called on to serve has a complete immunity from this scourge. Although possibly absent for years, yet sooner or later the disease has occurred at every station.

It will be interesting, while considering the general prevalence of this disease, to glance more particularly at its distribution in India, for several reasons.

In the first place, this vast peninsula from north to south embraces a great variety of climates. Again, the returns are accurately compiled by skilled observers.

The conditions under which the soldier lives are well known. We know that in the great majority of cases his surroundings are good, for it is impossible to speak too highly of the admirable

barracks and hospitals in the plains of India. In fact, everything which can be devised to promote the welfare and efficiency of the soldier in India is done; and although we are well aware that many insanitary conditions surround our cantonments, yet, as far as the actual dwelling-place of the soldier goes, nothing can be said except in its favour. Most of the accidental causes of this disease would thus seem to be eliminated.

In India unrivalled opportunities exist for observation, and many of the facts which are known about the tropical variety of this fever are due to the researches of medical officers in that country. The following table gives the admissions and deaths per 1000 from typhoid fever in the three Presidencies for a period of ten years:[1]—

Period.	Bengal.		Madras.		Bombay.	
	Admissions per 1000.	Deaths per 1000.	Admissions per 1000.	Deaths per 1000.	Admissions per 1000.	Deaths per 1000.
1879,	5·3	2·28	3·9	1·42	3·1	1·75
1880,	8·7	3·07	2·6	1·36	9·5	5·76
1881,	6·3	2·62	·9	·58	4·2	2·83
1882,	7·3	2·90	4·0	2·09	5·1	1·90
1883,	8·1	2·52	8·6	2·86	5·7	1·55
1884,	12·6	3·31	11·4	1·67	9·1	2·05
1885,	14·0	4·05	6·4	2·19	7·3	2·54
1886,	21·6	5·70	11·9	3·85	12·3	4·16
1887,	13·4	4·90	11·5	2·98	11·3	3·40
1888,	16·2	4·15	7·4	2·26	11·6	4·04

From this table it will be seen that although Bengal heads the list with the greatest number of admissions and deaths, the other two Presidencies also suffer severely. If we take the stations and provinces of Bengal in detail, we find that no certain laws exist with regard to its distribution according to climate.

The hill stations of India furnish a very large admission and death-rate. No doubt this is, in some measure, due to certain difficulties which arise in the sanitation of these stations. Wherever we have a large number of men inhabiting a comparatively small area, special precautions are needed in connection with the removal of excreta and the purity of the water supply. We shall probably find that greater attention to the sanitary conditions of some of the hill stations will be followed by a corresponding reduction of the prevalence of the disease.

[1] This and most of the other tables are from the Annual Reports of the Sanitary Commissioners with the Government of India.

The following table shows the admissions and deaths from typhoid fever in the military divisions in Bengal:—

TABLE SHOWING THE DISTRIBUTION OF TYPHOID FEVER IN THE BENGAL PRESIDENCY IN 1888.

	Average Strength.	Number of Cases.	Admissions per 1000.	Number of Deaths.	Deaths per 1000.	Percentage of Deaths to Cases.	Percentage of Deaths to Cases in 1876–85.
1. Gangetic Provinces,	6,728	179	26·6	31	4·61	16·76	32·15
2. Hill Stations,	6,731	157	23·3	33	4·90	21·02	27·10
3. Quetta District,	2,223	49	22·0	12	5·40	24·49	Not available
4. Rohilkund and Meerut,	5,214	102	19·6	28	5·37	27·45	39·12
5. Agra and Central India,	3,010	38	12·6	17	5·65	44·74	43·26
6. Punjab,	13,418	142	10·6	43	3·20	28·87	36·42
7. Convalescent Depôts,	1,874	18	9·6	8	4·27	44·44	28·57
8. Bengal Proper,	1,745	7	4·0	5	2·87	71·43	43·90

Race Liability.—When we turn to the incidence of this fever among the natives of the countries to which our observations extend, we are confronted with many difficulties.

Let us take, for example, the facts that are known of the relationship of the disease to the native population of India, which comprises a great variety of races. There can be no doubt, as we have said, that typhoid fever attacks the natives of India. Post-mortem examination has quite convinced us of this; but it does so to a very limited extent, as far as our present knowledge goes. Nor are we able, from the material at our disposal, to apportion out to the various races their respective degrees of liability to this fever. It would appear, however, that the meat-eating races suffer to a greater extent than those races which feed almost exclusively on vegetables and grain.

In the Indian Native army the mortality from typhoid fever is about 0·1 per 1000; in the Indian jails it is about the same. Compared with the admissions and deaths among the European army in India, the difference is enormous.

The following table shows the admission and death-rates per 1000 among these three classes:—

Classes.	From 1877–1886.	
	Admissions.	Deaths.
European Troops,	8·9	3·15
Native Troops,	0·2	0·10
Jail Population,	0·2	0·10

In Bombay, in the year 1885, out of 21,850 deaths among the natives from all causes, fever accounted for 6648, and only 10 of these are ascribed to typhoid fever.

It must not be forgotten that the deaths from "fever" among the natives are much more numerous than among the Europeans, and it is a question whether many cases of typhoid fever do not pass unrecognised. When we take into consideration the difficulties in the way of procuring post-mortem examinations among the natives of India, it lends additional force to the suspicion that many cases may remain undetected. Among the theories which have been advanced to account for the comparative immunity of the adult native population, it has been suggested that this disease is really extremely common among native children, and that the comparative freedom which adults appear to enjoy is due to an immunity conferred by a previous attack in early life.

We well know that the children of natives in India suffer much from "fever," accompanied in many instances by diarrhœa; but we hardly think it possible to accept this theory of immunity without very definite proofs, which at the present time are not forthcoming.

On turning to the United States it has been found that the white race suffers more than the negro, and the negro more than the Indian. The proportion of deaths from typhoid fever to 1000 deaths from all causes among these races is as follows:—White races, 33·9; coloured races, 31·7; and Indians about 22.

Hirsch states that the relative immunity of acclimatised persons from typhoid fever is not dependent on peculiarities of race or nationality, whether in temperate climates or in the tropics. He points out in support of this view that negroes in their own country are found to suffer much less than new-comers of other races, but when they are removed to America they suffer very severely. Lewis mentions that hundreds of negroes died of typhoid fever in Central Alabama in the winters of 1835–36 and 1837.

In British Guiana, typhoid fever was at first considered to be absent; but Dr. Ferguson has shown (in the *British Guiana Medical Annual*) that the disease is prevalent among the coloured races, chiefly made up of negroes. On Dupont's authority we learn that typhoid fever is observed in French Guiana at all seasons and among all races, viz. European, Negro, and Indian.

Davidson states that in Madagascar it is one of the most common diseases of the Central Plateau, and attacks the Hovas, a Malay race, very severely.

On the same authority, some districts, at least, in China, suffer largely from this fever. Pekin, Shanghai, Foochow, and Formosa

come under this category, while in Swatow, Amoy, Chin-kiang, Hankow, and Kin-kiang it is rare. The returns from the Civil Hospital at Hong-Kong prove that typhoid fever is more common in that colony amongst Europeans than natives. In 1886, 590 cases of admission among Europeans for all diseases gave 8 cases of typhoid fever; while 380 admissions of Chinese gave only one case.

Although it is at times present among the white troops stationed there, the years 1888–89–90, with an average strength of 1346, gave no admissions for this disease. Upon the whole we must conclude that the Chinese are no more exempt from typhoid than the negro.

In Arabia, the Arabs along the coast suffer considerably from it. Palgrave states, however, that it is quite absent from the pathology of the plateau. The disease is common among the natives of Khartoum.

In Egypt, with a strength of 3209 white troops, there were 123 admissions in 1890, with 23 deaths, being in the ratios of 38·3 and 7·17 per 1000 respectively. Nor is the disease unknown, although we have it on the authority of Dr. Sandwith, of Cairo, that it is rare among the native population.

In Senegal typhoid fever is rare, but it is met with occasionally both among Europeans and natives.

On the Gold Coast, Dr. Prout, the Government medical officer, states that he has never seen or heard of a case on the Coast. Mense says the same of the Congo.

When we get up to the interior, east of the Great Lakes, we find that it is exceedingly common among the blacks, that is, the natives of the negro race. Dr. Pruen, who has studied the disease in these parts, gives an explanation of the cause of this fever which deserves attention. He believes that the "first showers" moisten the accumulations of refuse lying about, and wash the decaying matter into the nearest stream, which serves as the water-supply of the natives. He further remarks on the prevalence of the disease on the marshy shores of the Victoria Nyanza.

Drago reports that typhoid fever has not been recognised at Zanzibar. It seems difficult to believe that it does not exist there; it must, however, in any case, be comparatively rare.

It is very rife in Australia; but whether it existed among the natives prior to our colonisation, or whether it even now exists among them outside the limits of European settlements, seems doubtful.

Davidson thinks, from the account of the *Diseases of the Aborigines of Australia* by Smith, that it is probable that the disease was

endemic there before the arrival of the first settlers, and that it is found among them at the present time.

Dutroulau and Boyer have observed the disease among the natives of New Caledonia; they differ as to its prevalence. Dutroulau states that it is very common; Boyer, on the contrary, only met with two cases during a somewhat prolonged residence in this island.

In the Sandwich group the natives suffer a good deal; and although the number of deaths returned under this heading is very small, yet numerous cases appear under the heading of "fever"; and as these islands are not malarious, Davidson suggests that probably many of these deaths from "fever" are really from tropical typhoid fever.

It is endemic among the natives of the Marquesas Islands, and is also common enough in the Society Islands. Numerous French writers testify to this.

In summing up our knowledge on the geographical distribution of this disease, it becomes evident that it is ubiquitous, and found in all gradations of climate. No race is exempt from it, and the natives of a place suffer less than the foreigner. We shall discuss this point at more length hereafter.

Etiology.

Nature of the Disease.—It is curious that although this disease is prevalent throughout the civilised world, we know so little comparatively about its etiology. The exact nature of the infection which produces typhoid fever is still disputed.

At the present time the majority of observers believe that this fever is generated by a poison introduced into the body from without. We know, at least, that this is one means by which it is induced; although we shall draw attention, at the proper place, to certain facts which would seem to point to the possibility of an autogenetic origin.

We have to discuss the etiology of a disease which, as far as our present knowledge goes, is more fatal to Europeans, of the soldiers' age, in tropical and subtropical countries than any other febrile malady, and which is more liable to attack them in these regions than in temperate climates. It is said by some to be on the increase; but this is probably only apparent, and due to a better recognition of the disease.

We know with certainty that this fever can be most readily produced by infection derived from the stools of patients suffering from the disease, and carried by water, milk, and air contaminated

by these discharges. Some, however, hold the view that typhoid fever can be generated *de novo* by insanitary surroundings, and may be due to changes of a putrefactive or fermentative character taking place in collections of filth and excrementitious matter not mixed with typhoid dejecta. Before proceeding to discuss the etiology of the disease in tropical countries, it will be useful to glance at our knowledge of the causation of typhoid fever in Europe, and the two theories which have been advanced to account for its origin and spread in temperate climates.

The main issue involved is, whether this is a specific disease absolutely dependent on a specific poison or not.

Budd believed that typhoid fever is a disease purely specific in its character, and that however difficult it may be to account for its primary origin, or to explain the commencement of some epidemics, yet unless the specific germ on which it depends be present, the disease known as "typhoid" fever could not exist. Murchison's theory, on the other hand, was, that it could be produced by insanitary conditions, and especially from the products of fermentative changes in filth and excreta. Moreover, that it may be due to sewer gas, or the mixture of excreta, not derived from a previous case of typhoid, with the drinking water, and that it was not necessary to assume the existence of any specific germ.

Murchison's theory, which has been known as the "pythogenic" theory, has been widely supported, but it would seem at the present time to be losing ground.

These two opposite views have been for many years before the profession, and we are still unable to state confidently which is the correct one.

On the discovery of the bacillus, which was alleged to be, and which probably is, the actual parasite of typhoid fever, great hopes were entertained that much more light would be thrown on the etiology of this fever, and that the question would be definitely settled.

The feeling among authorities at the present day seems to be gaining ground that this is a disease caused by a specific micro-organism, the introduction of which into the body is necessary for the development of the disease. But, as we shall have occasion to remark in discussing the bacteriological origin of typhoid fever, it has not been possible to induce, beyond all shadow of doubt, typhoid fever in animals by inoculation with cultivations of the bacillus,[1]

[1] A disease not unlike typhoid fever has been produced by inoculation of animals with cultivations of Eberth's bacillus, but points of difference between the disease so induced and true typhoid fever have invariably been noticed. For a good review of this subject up to the present date, see an article in the *Lancet*, March 4, 1893, by Dr. A. C. Lathom.

and therefore we are unable to say with absolute certainty whether the so-called bacillus of typhoid is really pathogenetic, or only incidentally, although constantly, present in the disease.

We are certainly inclined to believe, from our knowledge of the dependence of other infective diseases on specific micro-organisms, that typhoid fever will also be found to be dependent on a specific bacillus, and we believe that this specific micro-organism is that which has been isolated and described as Eberth's bacillus.

Difficulty in tracing the Source of the Infection.—We must not, however, shut our eyes to the extreme difficulty of accounting for all the phenomena of its outbreaks in tropical climates on this assumption. Many of the epidemics in Europe have been actually traced to the fouling of water or milk by typhoid stools. The supporters of Murchison's theory must admit that this is certainly one means by which the disease is spread. They, however, point to a large number of epidemics in which, after the most careful search and complete investigation, no such cause could be demonstrated.

Were it only now and then impossible to trace the disease to infection from a previous case, we should, no doubt, be inclined to lay the blame of failure solely at the door of the investigator. Our experience in India and the colonies, however, has convinced us of the great difficulty of determining, in a very large percentage of cases, the origin of this disease. This seems to point to the probability, in these countries, of some other way of infection besides the direct method, from the stools of pre-existing cases. By this we mean that it is often impossible to demonstrate a continuous chain of infection from the stools of recent cases as the cause of a given outbreak. This may, in fact, be looked upon as the etiological peculiarity of the tropical form of the disease.

We are repeatedly called on to account for sporadic cases, or for the origin of epidemics, where it is impossible to trace infection either from a recent case in the community or from importation.

We shall attempt in the sequel to show that diffusion of fæcal matter by the wind is by no means an unlikely means by which the infection is spread in some of these obscure outbreaks.

The absence of sewers in most Indian stations, and the impossibility, therefore, of leakage from these into wells and reservoirs, removes one of the commonest causes of this disease in European countries.

A great deal of our knowledge of the tropical variety of typhoid fever has been gained from the writings and experience of medical

officers in India, and we shall have occasion to refer frequently to facts gleaned from these sources.

The supporters of Murchison's theory of the pythogenic origin of this disease, have, we admit, a very strong case in India.

In the first place, we have throughout the whole of India vast populations living in absolute ignorance, and complete defiance of all the rudimentary laws of hygiene.

The ground around most Indian villages and towns is simply a vast latrine, and has been so for centuries. The smell from such localities is very perceptible for a considerable distance. The water supply is usually obtained from surface wells. In the rains, these surface wells are filled with water which has dissolved the surface impurities, and the waters of these wells are rich in organic matter. The natives of India also perform their ablutions on the margin of the wells, and the drainage back of the water into the wells is of the most insanitary kind. In this way a great deal of the drinking water of the country is contaminated by fæcal matter.

If typhoid fever were a common disease among the natives, it would be very easy to account for the presence of the specific bacillus in such drinking water; but as we have before stated, the natives, like those of other typhoid haunts, enjoy what appears to be an almost complete immunity, although we cannot help suspecting that the disease is more common amongst them than is supposed. As regards the soldier in India, who is especially liable to attack, his sanitary surroundings, in cantonments at least, are very carefully supervised. The barrack accommodation is excellent, and the dry earth system of latrines, if carefully carried out, would seem to reduce the chances of infection from this source to a minimum. We cannot, however, keep our soldiers constantly in cantonments; and we quite believe that a considerable danger exists in the use of aërated waters and other drinks which he buys in the bazaars, and which may be concocted of suspicious waters.

We do not think that the great difficulties which surround the causation of this disease in the tropics are thoroughly appreciated in Europe. When we come to investigate its origin in warm climates, we find that the conditions under which it appears are often entirely different from those which we are accustomed to in Europe.

Occurs on Virgin Soil.—How can we explain such well-known facts concerning tropical typhoid as its occurrence among troops who have been stationed for some time at places hitherto unoccupied, and who have not themselves imported the disease? Sir Thomas Crawford, late Director General of the Army Medical Department, for instance, points out that cases of typhoid fever

occurred in our army in Afghanistan at nearly every post occupied by our troops, and that many of these positions were occupied, it is believed, for the first time by human beings.

Our experience in South Africa was the same. Similar experience has been met with in the United States.

Hunt, in his remarks on the disease in North Queensland, says that cases of typhoid fever occur in remote stations, far removed from the routes of traffic, where no typhoid was ever known.

The French have noticed the same fact with regard to the occurrence of the disease in their army in Algeria.

Identity of Typhoid in Tropical and Temperate Climates.—In attempting to explain such phenomena, we must, in the first place, ask ourselves if the disease we call typhoid fever in tropical climates is the same as that with which we are familiar in Europe?

Their identity must, we think, be admitted; the post-mortem appearances and clinical history are the same in both, although the symptoms are occasionally modified. Some writers have attempted to prove that typhoid fever may exist in two forms; the lesions found post-mortem, however, being the same. In the one variety they class the cases presenting exactly those symptoms that are met with in Europe, and which can often be traced to a pre-existing case. In the second variety, which they state is the more common form in tropical countries, the following peculiarities are observed, which have been thus formulated by Webb in the *American Journal of the Medical Sciences*, April 1882:—

"1st. The cases are often sporadic, and cannot be traced to infection from water or air polluted with anything derived from a previous case.

"2nd. The rose-coloured spots are generally absent.

"3rd. Constipation is quite as common as diarrhœa.

"4th. These cases seldom present the normal thermal curve, but often begin as a remittent or intermittent fever."

Webb insists that sporadic cases of this nature occur "with none other than the ordinary miasmatic influences to account for their origin, and all absence of evidence of an infectious character."

Although we are quite prepared to admit that a class of cases is often met in tropical climates which seems to present these differences from the ordinary classical type of the disease, yet we do not think there is sufficient reason to describe these cases as belonging to an etiologically distinct variety, nor do we agree with the deduction that they are the result of ordinary miasmatic influences.

We must further inquire whether the lesions found in typhoid fever are found in this disease alone. Is it possible that tropical typhoid fever is simply a form of malarial fever, or that the lesions found in this disease are simply due to long continued high temperature?

In India the highest fever mortality among the natives is ascribed to malarial fevers, while among Europeans the death-rate from typhoid fever takes the first place. Are these two fevers essentially the same, and only modified by race and length of residence in the country? There can be no doubt but that these two fevers are perfectly distinct; and although they present many points of similarity, yet they have points of most essential difference, which will be dwelt on more at length under the heading of *Diagnosis.* We may state here, however, one point of importance, viz. that long protracted cases of true remittent fever present no signs of ulceration of Peyer's patches.

Some observers have been inclined to regard the disease as one of climatic origin. We can hardly entertain this view when we reflect that typhoid fever is met under such widely different climatic circumstances. It exists alike in hot and cold, in moist and dry climates, and seems to flourish in every quarter of the globe.

Media of Diffusion.—Believing, as we do, that typhoid fever is really due to a specific organism, we must consider what factors are concerned in the diffusion of this micro-organism.

As we have hinted before, the difficulties in the way of proving with certainty the transmission of the infection in tropical countries is great.

Sewers do not exist in the greater part of India, and therefore no contamination of the water-supply by this means is possible. Again, as there are no sewers, the disease cannot be introduced into habitations by sewer gas.

There are three chief ways by which it has been proved that this disease can be disseminated, viz. water, milk, and air.

The water supply in the majority of stations in India is from wells. A great deal has been done lately in the way of introducing a pure supply by pipes to some of the large stations.

In cantonments the wells are, as a rule, carefully built, covered, and preserved, and it is difficult to see how the poison can be diffused by this means as long as the supervision is strict.

We are well aware that the subsoil water, passing through ground infected by typhoid emanations, has the power to carry the germ of the disease from some distance. Supposing that typhoid dejecta were thrown on the soil, or buried in proximity to the

source of the drinking water supply, we could well understand this means of fouling the water.

The excreta in most Indian stations are, however, buried at some distance from cantonments. The chief risk, so far as water is concerned, arises from the fact that our soldiers are not always in cantonments; their visits to the bazaars, and their excursions into the country for sporting and other purposes, give them occasion to use water from impure sources.

As typhoid fever is endemic along most of the channels of European communication, a constant danger of infection is present at rest camps, and on the line of march at the various halting-places. The milk supply, often a source of danger in Europe, does not appear to be a usual method for the conveyance of this disease in India. It is a well-established fact that the women and children, who naturally are the principal consumers of milk, are not by any means the greatest sufferers from this disease, in fact rather the contrary. While, then, we think that the spread of infection by these two media, viz. water and milk, is not only possible, but extremely probable, we must not forget that medical officers in India have experienced much difficulty in tracing epidemics and sporadic cases to these causes. We now come to consider the third method of diffusion, namely, by the air.

While not in a position to prove that this is a common way by which the specific bacillus is spread, we cannot help thinking that air may readily play a very important part in its diffusion.

In many parts of India the soil in the dry season is of a peculiarly light, porous character, and is carried about by the winds in clouds. We have only to call to the minds of residents in India the not uncommon phenomenon of a "dust storm."

The dejecta of typhoid patients are buried in shallow trenches, and covered often by only a few inches of soil. True, these emanations have probably been treated by so-called disinfectants when the nature of the disease has been recognised; but are the disinfectants in ordinary use really germicides? We have considerable doubts on this point.

We know that the poison of typhoid fever can exist for a considerable time outside the body. Surgeon-Captain Firth, who read a very able paper at the Hygienic Congress at Portsmouth, 1892, on the relation of soil to enteric fever, and whose views on the matter we entirely share, stated that from his experiments he was able to determine the presence of Eberth's bacillus in the soil six months after he had artificially infected it by typhoid dejecta. Other authorities support the view that the poison can retain its virulence for considerable periods outside the body. Dr. Cayley, in

the Croonian Lecture, 1880, quotes the well-known instance related by Von Gietl: "In a village free from typhoid fever an inhabitant returned suffering from the disease, which he had acquired in a distant place. His evacuations were buried in a dunghill. Some weeks later, five persons who were employed in removing the excreta from the heap were attacked by typhoid fever. Their alvine discharges were again buried deeply in the same heap, and nine months later one of two men who were employed in completing the removal of the excreta, was attacked and died. Here we have distinct evidence that the poison retained its power for nine months."

Murchison, in his work on "Fevers," gives an instance in which the infection supplied by six cases was spread over a period of eight years. If, then, it is an established fact that the soil can become infected by the bacillus, and that this can be transported by the wind, then, given a congenial soil in which the bacillus can retain its vitality when it is deposited, a probable means of the dissemination of this disease becomes at once evident.

Many observers have thought that soil plays an important part in the production of this disease in the tropics; the heat, the amount of moisture, and the porosity of the soil being the factors chiefly concerned. Some, again, have thought that the typhoid microbe is essentially a micro-organism belonging to the soil, and capable of leading an independent life, and of reproducing itself in the earth. If this should be proved, there would be no difficulty in understanding why the disease should arise independently of the introduction of the germ by a person suffering from the disease, and appear in the most diverse localities where previous cases of the disease had never been observed.

Magne has made some attempt to classify soils in relation to their liability to the disease. The outcome of his investigations seem to be that typhoid fever is more prevalent on the recent formations.

Relation to Ground Water. — It will be necessary to glance at Pettenkofer's views on the subject of the influence of the ground water on the prevalence of typhoid fever. He attributes a direct connection between this disease and the fluctuations in the level of the ground water. He states that outbreaks of typhoid fever occur when the ground water is lowest, and especially when it falls rapidly after having risen to an unusual height. It will be seen that Pettenkofer is inclined to believe that the specific germ resides in the soil, and that certain conditions of heat and moisture are favourable to its development.

In England the changes in the level of the ground water are

numerous; but no connection has been traced between these changes and the prevalence of typhoid fever. In India, where the question has received attention, the weight of evidence seems against the theory brought forward by Pettenkofer.

Susceptibility.—Evidence would seem conclusively to prove that a varying degree of susceptibility must exist among individuals to the attacks of the specific fevers.

If a given number of people are subjected to the same chances of infection, only a limited number will be attacked by the disease. This is certainly true of typhoid fever, and a special predisposition would seem to exist in some families.

The chief predisposing causes of tropical typhoid fever would appear to be age, length of residence in a tropical climate, and the season of the year.

Age.—Age seems to exercise a very considerable influence on the liability to attack. Early life is comparatively free from the disease, and up to the age of 15 the number of attacks and deaths are comparatively few. In India the death-rate per 1000 of strength for children (up to the age of 15) is 1·32. The following table, taken from the Annual Report of the Sanitary Commissioner with the Government of India for 1888, shows the relationship between typhoid fever and age:—

Mortality from Typhoid Fever and Ratio of Liability to it, at Different Ages.

	Under 25.		25 to 29.		30 to 34.	
Year.	Deaths per Mille.	Percentage of Liability.	Deaths per Mille.	Percentage of Liability.	Deaths per Mille.	Percentage of Liability.
1879,	6·17	54·1	2·73	23·9	1·78	15·6
1880,	6·25	56·26	3·15	28·35	1·09	9·81
1881,	4·56	59·84	1·57	20·60	0·79	10·37
1882,	4·32	56·18	1·55	20·16	0·78	10·14
1883,	4·34	66·36	1·50	22·94	·70	10·70
1884,	4·61	62·05	1·83	24·63	...	...
1885,	5·16	61·06	2·30	27·22	·74	8·76
1886,	7·44	63·05	3·08	26·10	1·02	8·65
1887,	5·37	54·24	2·63	26·57	·68	6·87
1888,	5·46	61·21	2·36	26·46	·84	9·42

In studying this table, the influence of age on this disease is most apparent. It is evident that the period between 20 and 25 years of age is the most fatal in hot climates for this disease. Of the cases in the army in India, 64 per cent. occur in men under 24 years of age; 21 per cent. between 25 and 30; and 12 per cent.

from 30 to 34. We must not forget, however, that this is distinctly a disease of early manhood in Europe as well as in India. From the age of 40 to 50 and upwards the disease is very rare.

Length of Residence.—A still more important factor is the length of residence in a tropical climate. A table taken from the same source as the previous one illustrates the fact that those who have recently arrived in the country are much more susceptible than those who have lived there for a number of years, and that the liability to attack and death diminishes with each year of residence.

TABLE SHOWING THE DEATH-RATES OF THE EUROPEAN ARMY FROM TYPHOID FEVER AT DIFFERENT PERIODS OF RESIDENCE IN INDIA, TOGETHER WITH THE RATIOS OF LIABILITY TO IT FOR THE YEAR 1889.

Year.	Mortality from Typhoid Fever and Rates of Liability to it at different Periods of Residence in India.					
	1st to 2nd years.		3rd to 5th years.		6th to 10th years.	
	Deaths per Mille.	Percentage of Liability.	Deaths per Mille.	Percentage of Liability.	Deaths per Mille.	Percentage of Liability.
1889	11·65	57·02	4·20	20·56	2·43	11·89

The deaths from this disease in the first and second years of residence in India are more than double those which occur between the third and fifth years, and nearly five times those between the sixth and tenth years; while the liability to attack diminishes in the same ratio. European soldiers in India are thus seen to suffer more in the first years of residence than after they have become acclimatised.

In the first place, they arrive at a susceptible age; and, in the second place, the novelty of their surroundings, the alterations in their habits and food, and their transference to a hot climate, all tend to produce an irritable condition of the whole system—especially of the digestive tract—very favourable to the reception and development of the specific poison of this disease.

The great influence exercised by age and length of residence in tropical climates is admitted by all observers in India, and the same exists in other tropical climates.

It is interesting to note that when the native of India emigrates to other countries, he undoubtedly suffers more severely than in his own country. Surgeon-Colonel Welch states that the disease has been

observed among native troops sent to Hong-Kong, North China, and Singapore.

Relation to Season.—Another factor we have to discuss is the seasonal prevalence of typhoid. A very important fact becomes evident, namely, that this disease is more common in all parts of the world, from which we can obtain trustworthy information, in summer and autumn, than in spring or winter. In those countries which have a distinctly marked hot and cold season, the disease is at its maximum in the hot season.

Hirsch states on various authorities that in Iceland, out of six epidemics, the disease invariably reached its height in late summer and autumn. In Malta and Italy the greater number of epidemics occur in autumn; and, in the Cape, of four epidemics, three made their appearance in summer and one in autumn.

In Algiers, of three epidemics all happened in summer.

In Tunis, Japan, Cochin-China, and New Caledonia, the same rule holds good.

The greatest prevalence exists in summer and autumn in the Bermudas.

Jamieson, in the *Medical Reports* from China, notices that this disease is most prevalent in summer.

The relation to the seasons of the year in India again demonstrates the same rule. The annexed table gives the percentages for twenty years. It is taken from the Report of the Sanitary Commissioner with the Government of India.

Quarterly Percentage of Admissions for Typhoid Fever in India (by Presidencies) in 1870-89.

Presidencies.	January to March.	April to June.	July to September.	October to December.	Total.
Bengal, . .	13	40	29	18	100
Madras, . .	15	19	49	17	100
Bombay, . .	16	20	41	23	100

It will be seen that the second and third quarters of the year are those in which the disease is most prevalent, and that in Bengal the second quarter of the year is the one in which the majority of the cases occur. This seems to be a peculiarity of Bengal.

The other two Presidencies show the mass of the cases in the third quarter. It will be interesting in this connection to note that

in India remittent fever and ague are more prevalent in the third and fourth quarters of the year.

To sum up shortly our present knowledge of the etiology of this disease, we believe that in tropical countries, as in Europe, it is caused by a specific micro-organism, and cannot originate *de novo;* that recent arrivals in a tropical country are more susceptible to the action of the bacillus than those who have resided some time in a warm country; that age plays, as in Europe, a very distinct part, and that climate *per se* has nothing to do with the causation of the disease.

The same channels of infection through which the disease is conveyed in temperate climates, with some notable differences in respect to their comparative importance, to which we have drawn attention, are those by which it is propagated in hot countries. Certain conditions of heat and moisture of the soil play a very important *rôle* in maintaining the vitality of the bacillus, which may be diffused, to a much larger extent than we are at present cognisant of, by the winds. The best hypothesis on which to work with regard to the prevention of the disease, is that it is of bacillary origin, and that collections of filth and excreta form an excellent nidus for the bacilli to live and develop in.

Symptomatology.

Incubation.—It will be necessary to discuss shortly the period of incubation of this fever. There is no disease in which this period is so ill defined. The general impression among the best authorities in temperate climates is that it is generally a comparatively long one, which varies from ten to fourteen or twenty-one days, or even longer.

Typhoid fever has frequently been traced to its source in England, where epidemics have been studied so minutely, that it has, in some instances, been possible to ascertain the exact time which had elapsed between the ingestion of the poison and the onset of the acute symptoms. Much information on this point will be found in the Croonian Lectures delivered by Dr. Cayley in 1880. He quotes the various periods of incubation in many well-authenticated cases. The time has been found to vary considerably. Cases are on record where the disease appears to have commenced almost immediately after exposure of the patient to powerful emanations from sewers or cesspools; but instances of this kind are rare.

If authorities have differed as regards the incubation period in temperate climates, they have done so to a much greater extent in

tropical climates. Surgeon-General Marston, whose work on *Typhoid Fever* will well repay perusal, is inclined to think that the period may be a much longer one than is generally supposed. He quotes, on this subject, a case recorded by Parkes in the *Army Medical Reports for* 1872. The evidence suggests that in this particular instance the incubation period lasted for twenty-nine days.

Marston brings forward evidence in connection with the Egyptian Campaign, from which he has reason to believe that the period may be much longer, and in some cases may extend over many weeks. Surgeon-Colonel Hamilton, on the other hand, believes the period to be a very short one in hot countries. He assigns as a reason that the body is frequently in a feverish condition, owing to improper diet, excessive use of stimulants, and the effects of heat. He concludes that the average period of incubation does not last above a week, and is frequently less. Our own experience leads us to the belief that the period of incubation is most variable in hot countries; but exact information on this and many other points in connection with this disease is much needed. On the whole, we think the most usual period of incubation ranges from ten to fourteen days, although it may vary considerably on either side. The commencement of the disease is most insidious, and in many cases its symptoms are equivocal and its progress variable. Instances are not uncommon in which a person has kept about during the whole course of the fever, and it may not have been till a relapse occurred that the true nature of the complaint was recognised.

The period of incubation is usually only marked by a feeling of malaise and prostration. In those who have suffered much from the effects of malaria, it is common enough for the disease to be ushered in by attacks of ague. From this peculiarity cases of typhoid fever are at first often diagnosed as merely attacks of fever due to the malarial poison. It is not till late in the disease that the true character of many of these cases is recognised, and in not a few cases the diagnosis has been made for the first time on the post-mortem table.

Onset and First Stage.—The exact period from which to date the onset of the acute symptoms is often hard to determine; in countries where fevers of all kinds are common, the early symptoms of the disease may easily be overlooked. We must remember that this is essentially a continued fever, and although it is marked by great fluctuations of the temperature, it is comparatively rare to find the temperature approximating to the normal during the earlier stages of the disease. The mere fact that it does so must not, however, exclude this fever from the diagnosis. Our experience in hot climates teaches us that the temperature does occasionally fall

to the normal during the first week (see Figs. 4 and 5). Nor must we lose sight of the fact that the disease is sometimes of an exceedingly mild type, and that the fever may abort at, or at some period prior to, the fourteenth day.

These abortive attacks of typhoid fever are frequently returned as "simple continued fever,"—a term which seems to be a very elastic one, and under which many of the various continued fevers due to a specific cause are wrongly placed.

We have already mentioned that the exact onset of the disease is frequently ill defined. The usual course is for the patient to experience for a few days a general feeling of depression and malaise. The sense of mental and physical prostration is often very well marked. One of the earliest and most constant symptoms is headache. This is usually very severe and frontal in character. It continues for the first week of the fever, and, taken with the great prostration complained of, becomes a valuable aid to diagnosis.

Restlessness, sleeplessness, and loss of appetite are also early symptoms. The appearance of a person at the commencement of an attack of typhoid fever may be rather characteristic; the patient appearing heavy, dull, and apathetic, and having a hectic flush on the cheeks. The initial symptoms are sometimes marked by rigors, or by a feeling of chilliness, alternating with flushing and heat; but many cases begin simply with a rise of temperature, without any well-marked rigor.

During the first week the temperature is high, and sometimes rises in the characteristic manner so frequently met with in Europe, the morning temperature being followed by a rise of two degrees in the evening, and sinking one degree the next morning, till at the end of the first week the temperature has risen to 104° or 105° F., and often higher, in severe cases. It may remain fairly steady at this point for some days; and in the third week, where no complication exists, the temperature may become intermittent, or gradually fall till it reaches the normal on the twenty-first day of the disease.

The fever may, however, last much longer, and the primary attack may be followed by relapse after relapse. We must not in tropical climates expect the same regularity of the temperature curve as is usually observed in temperate climates, or such as Wunderlich has depicted in his charts. The temperature chart in tropical typhoid is frequently by no means typical of the disease as observed in Europe, and the differences observed in individual cases is often great.

To illustrate the course which the fever frequently pursues in the tropics, we have introduced a series of six charts, kindly supplied by Surgeon-Colonel Martin, Army Medical Staff, who collected them

from cases which occurred during an epidemic of this disease in Secunderabad. These charts will serve to indicate some of the peculiarities of the thermometric course of the tropical disease.

Chart 24 is peculiarly instructive. The case was that of an officer. After frequent consultations, it was decided that the disease from which he was suffering was not typhoid fever. Acting on this diagnosis the patient was allowed some solid food on the thirtieth day of the disease. Death occurred from perforation on the thirty-third day of the fever. Post-mortem examination demonstrated the presence of four small ulcers, three of which were healing; the fourth had given way.

Charts 25 and 28 show the very irregular and ill-defined course which the fever sometimes follows.

Chart 26 shows a relapse occurring on the twenty-eighth day of the disease.

Chart 27.—In this case the temperature dropped to subnormal on the sixth day of the disease.

Chart 29 shows a fairly typical course of the fever. The case ended in death from perforation on the thirty-first day.

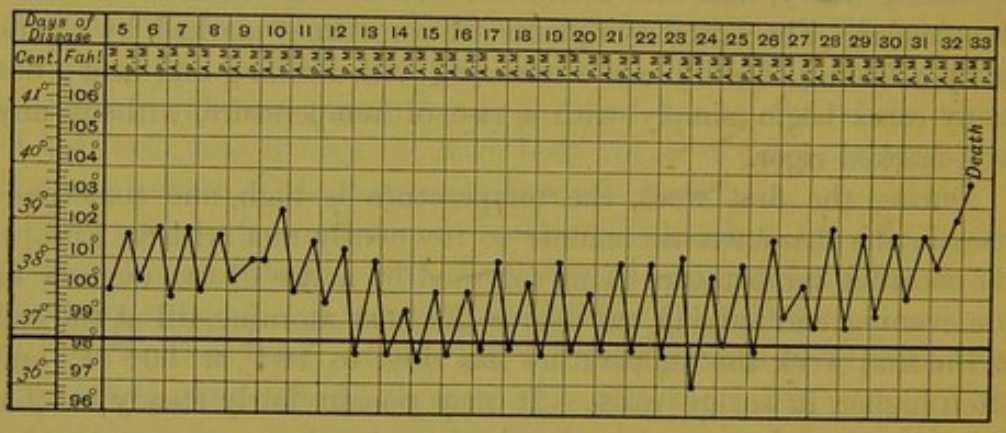

FIG. 24.

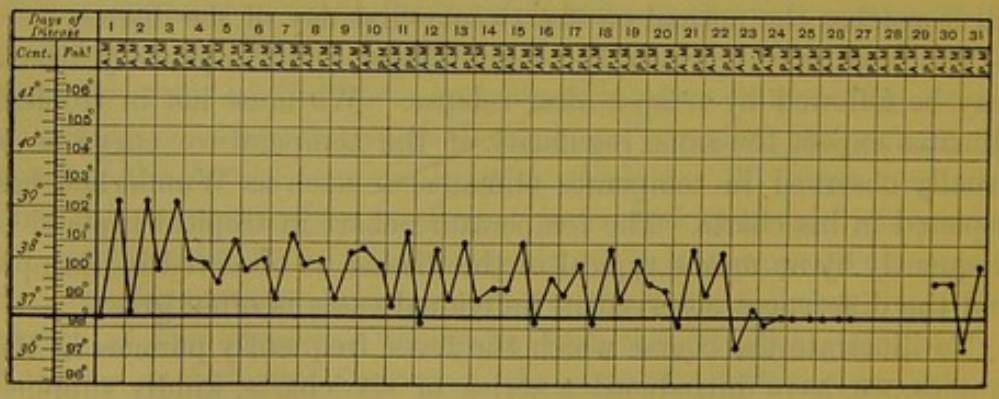

FIG. 25.

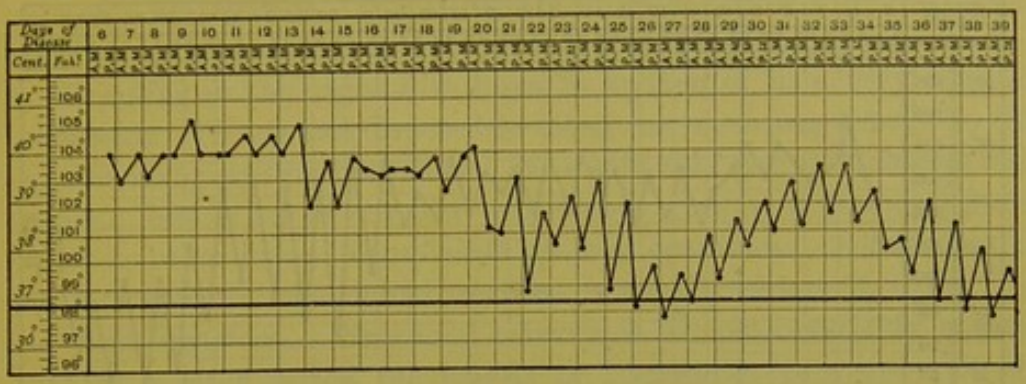

FIG 26.

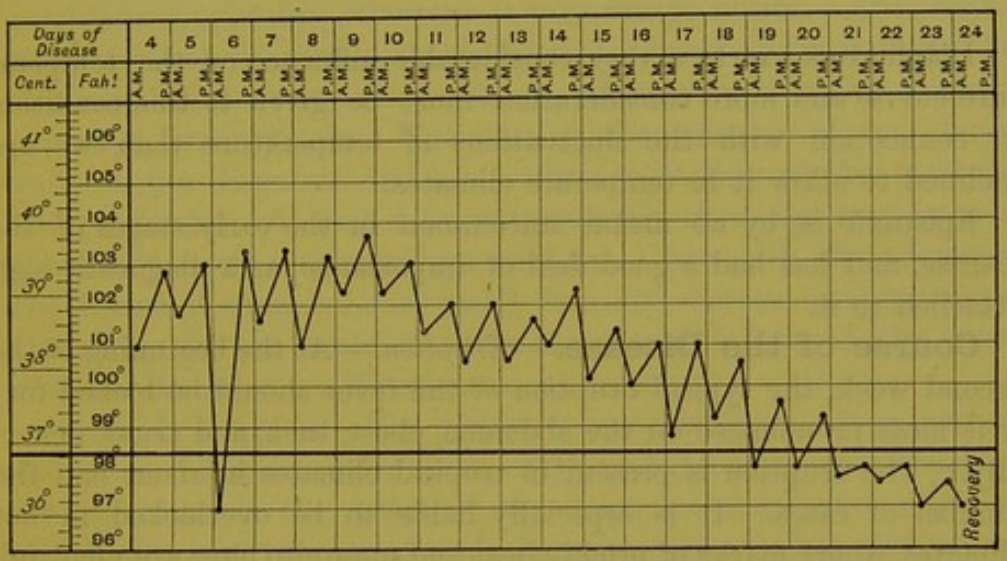

FIG. 27.

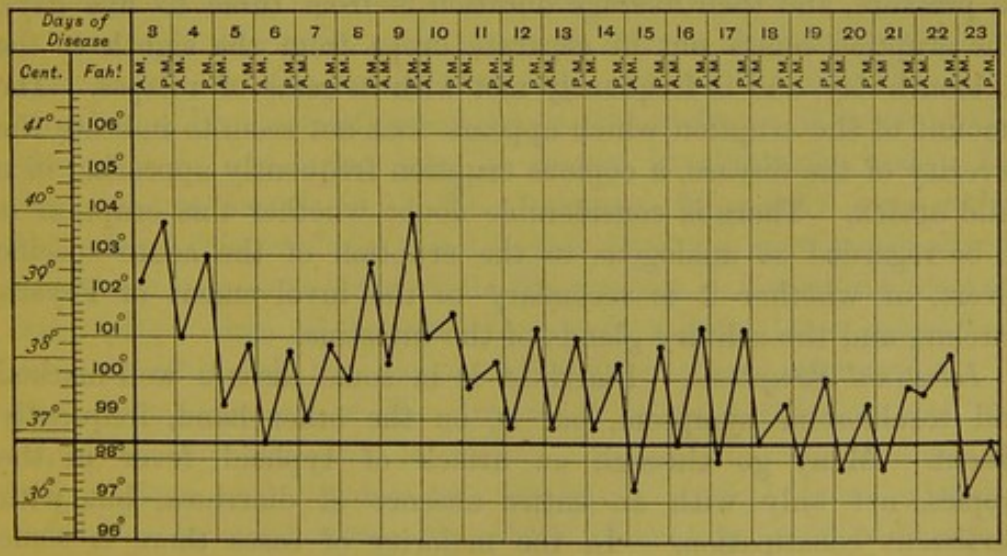

FIG. 28.

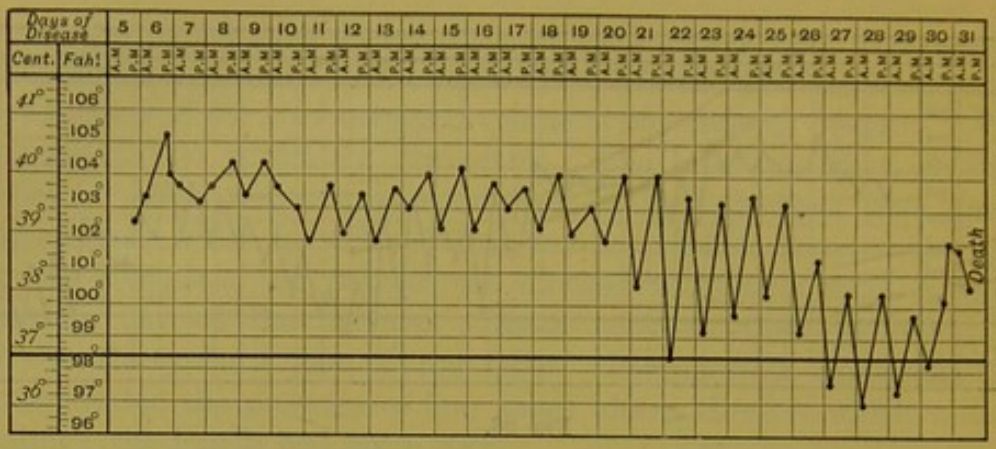

FIG. 29.

Diaphoresis exerts a very considerable influence on the temperature curve, and more consideration must be given to this condition in connection with the fluctuations of temperature than we are inclined to allow it in temperate climates.

Epistaxis is by no means uncommon in the early stages of the disease, and has had a good deal of importance as a diagnostic sign attached to it.

Course of the Disease.—*Eruption.*—At the beginning of the second week, the typical eruption of the fever should be looked for; it is most pronounced on the abdomen, chest, back, and trunk generally. The eruption is present in tropical climates in about half the number of cases. It is especially liable to be overlooked in hot climates, where marks of prickly heat and mosquito bites are plentiful. The eruption is exactly similar to that seen in Europe. The spots are raised, of a rose colour, and lenticular in form. They disappear on pressure, and their average duration is from three to five days. The eruption comes out in separate crops from the eighth to the sixteenth day, and is frequently met with in cases of relapse. The amount of the eruption which appears does not seem to indicate the severity of the disease, a copious eruption frequently appearing in a mild attack. There is considerable doubt whether this eruption is to be regarded as analogous to the eruption of the acute specific fevers, or whether it is secondary to the involvement of Peyer's patches, and the solitary glands of the intestine.

Intestinal Symptoms.—Diarrhœa is in some cases a well-marked and troublesome symptom, but is, on the other hand, frequently absent. Many go through an attack of typhoid fever in the tropics, not only with an entire absence of diarrhœa, but with persistent constipation. In the majority of cases there is some looseness of the bowels, and the stools may assume the so-called

pea-soup appearance. If so they are of light yellow colour of a consistence of pea-soup, and have an alkaline reaction.

Gurgling in the right iliac fossa is spoken of as a very constant symptom. We are not disposed to attach as much importance to this sign as many writers have done. No doubt this symptom is generally present, but as far as our own experience goes, it is frequently met with in other bowel complaints in hot countries. Tenderness on deep pressure in the right iliac fossa is usually observed.

Chest Symptoms.—Bronchial catarrh is almost invariably noticed, and at a later period pneumonia or hypostatic congestion of the lungs forms an important feature of the disease. The base of the lungs must be carefully examined from time to time, and this condition sought for, otherwise it may escape notice. The respirations are not, as a rule, much hurried, unless there is some concomitant inflammation of the lungs or bronchial tubes, or a marked degree of hyperpyrexia. The state of the pulse is of considerable value in the diagnosis, and has a distinct bearing on the treatment and prognosis. At first it is comparatively slow, and may be under a hundred beats a minute in a mild case; but with the advance of the disease it often becomes small, fast, and dicrotic or intermittent; the strain on the heart is badly borne, and failure of the heart's action is frequently a direct cause of death.

General Symptoms.—The disease is essentially one of an asthenic character. The muscular fibres of the heart become weakened and lose their tone; the action of the heart requires, especially when the disease is advancing towards its termination in hot climates, the most constant attention.

Delirium, at first absent, or only amounting to slight confusion of ideas, becomes later an almost constant symptom. The delirium is usually of the quiet variety, and is due to imperfect nutrition of the brain. In the worst cases the patient towards the end of the disease lies perfectly unconscious; the abdomen is much swollen, the motions and urine may be passed involuntarily, and a fatal issue seems imminent. We, however, frequently see patients rally from this condition; and in this fever we must never abandon hope, nor stay our efforts, while life remains. Surprising recoveries take place, when all hope of other than a fatal issue seems futile. The condition of the tongue affords us some information. At the commencement of the disease, it is, as a rule, covered with thick creamy fur, and is moist. As the disease advances, the edges clean and become red, leaving a central broad streak of white or brownish-white fur, which ultimately becomes brown and dry.

The lips and teeth are covered in the later stages of the disease with sordes.

The abdomen, which at first may be flaccid and natural, at the end of the second or commencement of the third week may become enormously distended by gases, to such an extent, indeed, as to require active treatment, owing to the pressure exercised on the heart and lungs by such distension.

The above is a short account of the more prominent symptoms which mark the commencement and progress of the disease, and which are, in the main, identical with those we are accustomed to meet with in temperate climates. We must, however, note as points of difference the comparatively rarer existence of diarrhœa, the frequent absence of the eruption, the irregular temperature curve, and the greater likelihood to relapses, in the tropical disease.

The special factor we have to regard when discussing the symptoms is the influence of climate. We wish it to be distinctly understood that we hold that climate has no influence in the origin of the disease. As may be readily understood, this disease, which is fatal enough in temperate climates, becomes more deadly in tropical climates. Not only has the patient to fight against a long continued and exhausting fever, but he has to combat, at certain seasons of the year, the exhaustion dependent on great heat. It is not our intention to go more into the detail of the symptoms, which may be studied in any of the standard text-books, and which differ in no material way from those met with in European countries, except in their aggravations by the climatic causes, in the manner we have just mentioned.

Complications.—We must now glance at some of the more common complications which may arise during the course of the disease, and the sequelæ which are occasionally found after the termination of the fever.

The complication which most usually calls for active treatment is *diarrhœa;* this may become excessive, and weaken the patient considerably. When it does so, the physician must step in and prevent undue waste of strength from this cause. We do not think it advisable to interfere unless the motions exceed four or five a day, and are accompanied by exhaustion. *Constipation* is a complication which often gives great trouble. It may arise from an almost exclusive milk diet.

Hæmorrhage is always a grave and serious symptom. Early in the disease it has not the same dangerous import as during the separation of the sloughs. When copious hæmorrhage occurs in the third week, it denotes that the ulcerative process has im-

plicated some large vessel, and the prognosis is decidedly bad. Hamilton states that in the Lucknow Station Hospital, out of 105 cases of typhoid fever in 1886, this complication was hardly ever observed, and no deaths took place from this cause. In 1887, however, out of 80 cases, there were numerous attacks of hæmorrhage, some very severe, and several fatal. While during 1888, out of 60 cases, hæmorrhage was noted in only two instances.

Peritonitis may either be general, from perforation, in which case the condition is well-nigh hopeless; or local, from implication of the peritoneum at the base of an ulcer. The frequency of *pneumonia* has been alluded to; this may either be of the hypostatic variety, or more acute in character, with inflammatory consolidation. It is nearly always present to some degree in the third week. The physical signs, and increased rates of pulse and respirations, usually show the advent and advance of this condition.

Hyperpyrexia is probably more frequently met with in hot than in cold climates. This condition should be most carefully guarded against; frequent thermometrical observations should be taken night and day, and energetic efforts made in the manner to be described later, to prevent the numerous evils which follow in its train.

Sequelæ.—The sequelæ of typhoid fever are various. Many observers in Europe have remarked the length of time over which complete convalescence extends. Debility, palpitation of the heart, and intense nervous prostration very frequently follow this fever.

This *great prostration* and *protracted convalescence* is especially marked in those who are recovering from a severe attack in hot climates. We may anticipate so far as to say that the best possible cure is removal from the heat to residence in a temperate climate. A sea voyage is probably the best of all tonics. Whenever it is feasible, a patient who has passed through a severe attack of typhoid fever in the tropics should be sent home to Europe, and remain there for some months. It is not fair to subject such a patient to the enervating effects of a hot climate while still in a low state of health. The hill stations in India and other hot countries may be advantageously employed in this way.

Abscesses of connective tissue and *periostitis*, with occasional exfoliation of bone, are sometimes met with as sequelæ.

Thrombosis of the veins of the leg and thigh is by no means uncommon.

In patients with a tubercular predisposition, *tubercle* in some of its protean forms is liable to be developed during convalescence from typhoid fever.

Insanity also, which is often of an evanescent nature, has been recorded among the sequelæ.

Treatment.

Diet and Nursing.—In a simple, uncomplicated case, no medicinal treatment may be necessary, and no drug can in any way cut short or lessen the severity of the disease. Occasions may arise, and complications occur, which call for the exhibition of drugs; these must necessarily be treated on their own merits. A purely routine treatment by medicine is much to be deprecated.

We have to treat a long continued fever, calling for the greatest possible care and attention, to enable the patient to hold his own against the disease. It is certainly one of the first duties of the physician to place his patient in the best possible surroundings, with a view of giving him the best chance to fight against the disease successfully.

We should, therefore, select a large airy room, with good ventilation, and also, what is perhaps just as important in the tropics, a room in which the temperature can be kept down to a suitable level by thermantidotes, punkahs, tatties, or other artificial aids. If practicable, it is advisable not to choose a room which gets the full glare of the sun for many hours in the day.

The bed should be placed in an easily accessible spot; and in the large rooms in tropical houses, the best position will generally be found to be the centre of the apartment, and, of course, underneath the punkah, if this is used.

The bed should be of moderate size, so as to be easily manipulated, and permit the patient being attended to without difficulty. If possible, the mattress should be of wire, and over this a well and evenly padded hair-mattress spread. In the later stage of the fever, it may be necessary to use a water bed to prevent bedsores.

Mosquitoes and flies are very troublesome; if a punkah is in use this will generally be sufficient to keep them off, if the fringe is hung low enough. If not, mosquito curtains should be employed, or a special attendant told off with a fan.

Whenever the services of a skilled nurse can be obtained these should be procured. Tropical typhoid is essentially a disease in which the help and co-operation of a trained nurse is of the first importance. It will be of the greatest possible value and comfort to the patient, and will relieve the medical officer from a load of anxiety and responsibility.

Regularity in the administration of food, stimulants, and medicine

is of the greatest moment. The ventilation and temperature of the room require the most careful management; and many other points which have much to do with the comfort of the patient and his ultimate recovery can only be efficiently and satisfactorily dealt with by a trained nurse. One of the main difficulties that we are often confronted with in the tropics is this very want of proper nursing. It is not too much to say, that this lack of good nursing has a most direct bearing on the high mortality.

The diet is a point which demands great consideration. The principles which must guide us in this particular are laid down in every text-book. Cow's milk seems by universal consent to be the article best suited for this purpose. When good cow's milk can be obtained, our anxieties on the score of diet are relieved. Goat's milk is sometimes used, but is not to be recommended if cow's milk is obtainable. From three to four pints of good milk are sufficient for a daily allowance. We must constantly test the milk to assure ourselves that it is really pure, and of proper nutritive value.

The specific gravity of the sample should be from 1026 to 1035, and should yield from 8 to 10 per cent. of cream. The taste, colour, and smell must be noted, and no sediment should deposit on standing. The milk should be boiled to prevent it turning sour, kept in a cool place outside the sick chamber, and well protected. About 4 oz. should be administered in a cup or feeder at a time, and given at short intervals. A lump of ice may be added to each cupful. The milk may be diluted with water, lime water, or barley water, if a large drink is required. Soda water may also be used for the same purpose, but it is not so good on account of its liability to increase flatulence and cause distension of the abdomen. Care should be taken to examine the stools regularly to see, among other things, whether any quantity of the milk is passed undigested; this is a common cause of diarrhœa. If this is the case, we should lessen the quantity of milk, and mix what is given with lime water. The milk may, if it is considered advisable, be peptonised before it is given. Condensed milk is useful if fresh milk is not procurable. The ordinary condensed milk is very sweet, and cannot be taken, as a rule, for any length of time. If the use of condensed milk is a necessity, we should try to obtain the unsweetened variety, which is more palatable, and can be continued longer without occasioning nausea or disgust. From one to two pints of good beef-tea or chicken broth may be given during the twenty-four hours, and the quantity of the milk proportionately reduced.

Much discussion has arisen as to the advisability of giving beef-tea on account of the supposed difficulty of digestion and its liability

to cause diarrhœa. Our own idea is that it is decidedly beneficial, and relieves the monotony of a wholly milk diet. If found to disagree it must be discontinued, and one of the meat juices or extracts substituted for it. Meat juice made by digesting finely-chopped beef in water, to which a few drops of hydrochloric acid have been added, and expressing the juice between folds of muslin, makes an excellent substitute. Valentine's and Brand's essences may also be given, and are excellent preparations. If the patient become unconscious, and food cannot be administered in the ordinary way, recourse must be had to nutritive enemata.

Stimulants.—A good deal of difference of opinion has arisen on the necessity for stimulants in the treatment of this fever. The question must be discussed without prejudice, and with a wish solely to arrive at the exact value of alcohol in this disease.

In the case of a young person who has previously enjoyed good health, and is suffering from a mild attack, we are of opinion that stimulants will probably not be required, at any rate in the early stage of the fever. We are prepared to go further, and to advise avoidance of stimulants, especially in hot climates, unless the condition of the patient clearly demands their use. In severe cases, when there is great exhaustion and prostration, the exhibition of stimulants is urgently demanded. The pulse must be our guide in deciding whether recourse should be had to them or not; while this remains moderately slow and full, and there is evidence that the heart is doing its duty well, there is no occasion for their use. When the pulse becomes fast, soft, and possibly dicrotous or intermitting, we thoroughly believe that stimulants are of the greatest service. Good brandy or wine should be employed, and should be ordered in fixed quantities as a medicine, and should never be given except in exact accordance with the order of the medical officer. The use of stimulants is especially called for when the lungs become engorged at the later stages of the fever. In cases where the patient is almost unconscious, and food is taken with difficulty, especially if the heart is flagging, their use should be pushed. The nurse should keep, not only an accurate account of the stimulants used, but also of the food given during the twenty-four hours, the amount of sleep, the effect of drugs, and other important circumstances.

Means of Combating Hyperpyrexia.—The temperature often gives us cause for great uneasiness; this may rise to 106° F. or higher. With the rise of temperature all the evil effects of pyrexia become apparent—restlessness, delirium, insomnia, and exhaustion. When the temperature rises over 103° F., especially if it seems on the rise still higher, steps should be immediately taken to reduce it.

In our opinion, it is useful to keep the temperature as far as possible at a much lower level, and check at once any tendency to rise. The exhibition of quinine as an antipyretic has not met with the success that at one time was hoped for.

Antipyrin and antifebrin are rather double-edged weapons in this condition, and should be used with great caution, if employed at all. Our own feeling is against their use in this disease. Their tendency to reduce the fever by copious diaphoresis tends distinctly to lower the general condition of the patient. In any case, these remedies are out of place in the later period of the fever, when the patient is much debilitated.

Cold applied locally is by far the best method of reducing the temperature. The treatment by the cold bath has been strongly advocated by some of our ablest physicians, and is no doubt of very great value. Dangerous pyrexia is checked, and the whole condition of the patient is changed for the better by the immediate and great reduction of temperature effected by this method. The pulse and temperature must be continually taken when this remedy is applied, and the tendency to collapse carefully avoided. It must also be remembered that the temperature of patients subjected to this treatment falls for some considerable time after they are taken out of the bath. Our difficulty in the use of the cold bath in hot countries is usually one of ways and means. In a London hospital, where plenty of skilled assistance is at hand, and special apparatus available for lifting and lowering the patient into the bath and raising him again, the method may be practicable. How seldom in the tropics are we in a position to enable us to pursue this treatment without great danger to the patient. We can, however, by placing the patient on a waterproof sheet, and covering him with towels, wrung out of cold water and constantly changed, make a decided impression on the temperature. The towels should be laid on from head to foot, and around each limb separately. Ice may be placed in the water if required. The wet pack is frequently recommended in this condition, but is not so satisfactory as the foregoing method. Ice placed in the vicinity of the body has a great effect in reducing the body heat, and seems to us to be exactly the remedy which, being easy of application and certain in its results, is most valuable. A very useful way of applying this is as follows:—

A cradle is placed over the body, which should extend from the neck to the feet. To this cradle is attached a large tray, about 4 to 6 inches deep, filled with blocks of ice mixed with salt, and hung to the cradle just clear of the body. A waterproof sheet and then the bedclothes are placed over the cradle and tucked in at the foot

of the bed and on each side, leaving the head only exposed. It is necessary that the patient should be very lightly clad, in thin pyjamas or night-dress, or, better still, naked. This method has been used at Netley, by Professor Henry Cayley, with the most encouraging results, the temperature of the patient being almost completely under control. Sponging of the various parts of the body with cold water, to which a little vinegar or eau de Cologne has been added, adds greatly to the comfort of the patient. This will be often sufficient to insure a quiet and refreshing sleep, when the patient is suffering from restlessness, the result of high temperature, and should certainly be tried in preference to drugs when the temperature is high and the local methods of applying cold in the manner above mentioned are not available.

Treatment of Complications.—The most frequent complications calling for active treatment by drugs are the following:—

Diarrhœa.—If the motions are more than four or five in the twenty-four hours, and accompanied by exhaustion, we must interfere.

We have alluded to the irritation caused by undigested milk, and we should satisfy ourselves, in the first place, that the diarrhœa is not due to an improper diet. Beef-tea is often blamed as a cause of this condition. A pill of acetate of lead, grs. iij, and extract of opium, gr. ½–gr. i, taken every four hours is very beneficial, or the lead and opium pill of the Pharmacopœia may be used. The effect of the opium must be carefully watched. Dover's powder, in 5 grain doses, is also a reliable preparation. A starch and opium enema is of great value,—30 drops of the tincture of opium may be added to 2 oz. of thin starch. Turpentine, sulphuric acid (dilute), tannin, chalk, and opium powder have all been recommended.

Constipation is best treated by soap and water enemata, which are repeated as often as required. It sometimes happens that this is not sufficient, and small doses of castor oil may be required. One to two teaspoonfuls of the oil may be given for the dose. Great caution requires to be exercised in the use of purgatives. The proper use of enemata is usually sufficient to obviate the necessity for purgatives by the mouth.

Hæmorrhage.—If this occurs, our first aim must be to stop the peristaltic action of the gut. Opium, either in the solid form or in that of tincture, is especially useful. The application of ice locally over the cæcum may produce the desired effect. The ordinary astringents are not of such great value in this condition. Turpentine in 20 drop doses, and the liquid extract of ergot in drachm doses, repeated frequently, have each been used with good effect. The hypodermic injection of ergotin is well spoken of.

Perforation.—In this condition opium must be given with no sparing hand, and feeding carried on altogether by the rectum.

The question of the performance of abdominal section and suture of the gut, must present itself to the mind of every thoughtful practitioner as justifiable in some cases. We imagine, however, that the cases in which it would hold out any prospect of success, must be very rare.

Peritonitis is usually secondary to perforation, and demands the free use of opium.

Pneumonia and other lung complications must be treated on general principles. A stimulant expectorant, containing small doses of carbonate of ammonia, will be found of service in the hypostatic variety; in this condition the free use of alcohol is of great benefit, increasing the force and diminishing the frequency of the pulse, and thus promoting the circulation through the lungs.

The position of the patient should be changed at times,—he should be encouraged to lie on his side for short periods. The dorsal position favours the tendency to hypostatic congestion of the bases. Hot fomentations with or without turpentine, locally, and the application of warmth by cotton-wool jackets, will be of service.

Sleeplessness is a complication, for the relief of which our aid is not unfrequently required. In the majority of instances, when we reduce the temperature of the patient, we procure sleep. In cases in which the insomnia seems due, not so much to pyrexia as to an irritable condition of the nervous system, the bromides of potassium or ammonium, from 10 to 20 grains for a dose, may do good; and bromidia, in ½ to 1 drachm doses, is well reported on. Hyoscyamus is also a valuable sleep-producing drug. Morphia is very useful in some cases, chiefly those in which pain is present, or to overcome extreme irritability and restlessness, but must be used with caution. It is best administered hypodermically, commencing with ♏ij of the pharmacopœial injection, but seems less suitable in the majority of cases than the remedies already mentioned.

Excessive thirst, which is generally complained of in the early stages of the disease, may be considerably relieved by small quantities of iced drinks, of which the best is barley water with a lemon squeezed into it, or, where diarrhœa is absent, lemonade made from fresh lemons or limes, great caution being exercised in straining off the pips and fibrous tissue of the fruit.

Distension of the abdomen is a common cause of discomfort, and often interferes seriously with the action of the heart and lungs. In the first instance, a turpentine fomentation may, by causing contraction of the muscular walls of the intestine, give relief. If

this fails, a rectal tube should be introduced for some inches up the gut, and allowed to remain for a few minutes.

The other complications and sequelæ which arise must be treated altogether on general principles.

We must bear in mind that typhoid fever is a disease which, when fatal, frequently kills by exhaustion and cardiac failure, and we should be prepared towards the termination of the disease to use alcohol and cardiac tonics so as to produce a real effect; of the latter, digitalis and strophanthus would appear the best.

It is hardly necessary to say much about the treatment of relapses, which are very common in this disease. The relapse must be treated exactly in accordance with the rules laid down for the treatment of the original disease. They are produced in precisely the same way as in temperate climates, and are due, in many instances, to indiscretions in diet. Nothing approaching solid food should be given, until a full ten days have elapsed after the last rise of either morning or evening temperature, and should then be of the lightest description for a few days. In spite, however, of all our care in this respect, relapses frequently occur.

Convalescence is, as we have said, very slow; the period is marked by months rather than by days, and health permanently damaged may be the result of over anxiety to shorten this time. The return to full mental and physical activity should be gradual. Fatigue should, as far as possible, be avoided, and no strain should be thrown on the heart, the muscular walls of which have suffered in the same way as the rest of the muscles of the body. Good food, proper exercise, an absence from worry, and a change of residence to a temperate climate, are among the best aids to a complete convalescence. Iron, quinine, strychnine, and phosphorus are the drugs which will most commend themselves to our notice as useful at this stage.

Diagnosis and Prognosis.

Diseases liable to be mistaken for Tropical Typhoid.—The diagnosis of tropical typhoid fever certainly presents some special difficulties. It is not always an easy matter to diagnose typhoid fever, even in temperate climates, but our hesitation in this instance usually proceeds from being called upon to make up our minds before the more characteristic symptoms of the disease have declared themselves.

When we consider the ease with which the febrile condition is induced in hot countries, and the great frequency of febrile attacks, evanescent and otherwise, we are not surprised that some con-

fusion is apt to arise as to the real nature of certain fevers of doubtful origin.

The effects of the sun, bowel complaints, the sudden check to perspiration caused by chill, malarial fevers, and many other conditions, give rise to fever more or less continuous, and often, for a time at least, closely resembling typhoid fever.

For many years in India, even after Bryden had conclusively proved that the disease existed, some authorities refused to admit its presence; and even now considerable doubts exist in the minds of some whether the so-called typhoid fever in India is really the disease known under that name in Europe. In a large number of cases the real nature of the fever is not made apparent till the post-mortem examination determines the existence of the specific lesions.

A few years ago, many of the cases of typhoid fever in India were returned under the headings of remittent, intermittent, and simple continued fever, and it is only quite recently that its great prevalence has been thoroughly realised.

The statistics of the army in India throw some light on this point. From 1870 to 1879 the deaths from typhoid fever were 2·03 per 1000; from other fevers (intermittent, remittent, and simple continued), 1·42 per 1000: giving a total of 3·45. From 1880 to 1889 the mortality from typhoid fever was 3·67; from other fevers, ·92: total, 4·59.

In 1888 the mortality from typhoid fever was 3·75; from other fevers, ·73: or a total of 4·48. In 1889 the mortality from typhoid fever was 6·11; other fevers, ·81: or a total of 6·92.

From this it will be seen that while the death-rate from all fevers combined has increased, probably, as has been pointed out, from the youth of the soldier on his arrival in a tropical climate, a condition due to the system of short service now in operation, there has been a decrease in the deaths from fevers other than typhoid. This would seem to suggest as a plausible inference, that typhoid fever is now better recognised, and that many fevers which previously would have been returned under the heading of malarial or of simple continued fever, are now placed in their proper category.

A great number of cases are still returned as simple continued fever; and although we quite admit that exposure to the sun and fatigue may produce a continued fever, yet we cannot help thinking that many of the cases returned under this heading are in reality mild cases of typhoid fever.

Attacks of simple continued fever do not, as a rule, last for more than two or three days, and are seldom prolonged beyond a week. A so-called simple continued fever which lasts for weeks, and is

independent of malarial poisoning, would seem to our minds to be highly suggestive of typhoid fever.

As we have said before, the symptoms are often exceedingly masked in the tropical variety, and the disease is not easy of recognition. It seldom presents all, and sometimes hardly any, of the symptoms we are accustomed to lay special stress on in the temperate climates. The absence, therefore, of those symptoms which we are accustomed to look for in typhoid fever in Europe should not prejudice our diagnosis.

Diagnosis between Typhoid and Malarial Fever.—The diagnosis between this disease and remittent fever often puzzles the best observers. In both typhoid and remittent we have a continued fever frequently lasting for weeks. In both, as a rule, we get a morning fall of temperature; in remittent fever this is usually more marked, often amounting to three or four degrees. Constipation is the rule in remittent fever; in typhoid fever diarrhœa is often absent, and constipation may be present; if the characteristic stools of typhoid fever are present, the diagnosis is rendered easy. Vomiting is common in remittent fever, but less so in typhoid fever. The spleen may be enlarged in both; but so far as our experience goes, it is not common to get great enlargement of this organ in tropical typhoid, and certainly not to the same extent as in malarial fevers.

Sweating, which is so pronounced in malarial fever, although often present in typhoid fever in hot climates, does not form such a prominent symptom. There is complete absence of tenderness and gurgling in the right iliac fossa in remittent fever. Relapses, which are so common in tropical typhoid, are not so frequent in remittent fever, the latter disease having a tendency to pass into the intermittent type of fever. The rash, though often absent in tropical typhoid, when observed is of great diagnostic value.

Among other points we must remember that typhoid fever is extremely common in the first two years of residence in a tropical country, and especially when youth is superadded. The absence of direct exposure to malarial poison, and the season of the year, must also be taken into account when forming a diagnosis.

The following table, compiled by Marston in the *Army Medical Reports*, 1879, emphasises the effects of seasonal influence:—

TABLE SHOWING THE DISTRIBUTION BY MONTHS OF 100 CASES OF TYPHOID FEVER AND 100 CASES OF MALARIAL FEVER IN THE ARMY OF INDIA. (1874–78.)

	Jan.	Feb.	Mar.	Apr.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
Typhoid Fever, .	5·13	3·75	4·33	9·39	14·73	10·54	9·17	9·89	13·86	8·74	4·69	5·78
Malarial Fever, .	4·29	3·6	4·03	5·56	7·50	8·20	8·88	9·93	12·60	15·06	12·01	8·34

It is easy to diagnose a simple classical case of either one or other of these two fevers, and it is not till we have to deal with cases in which many of the more characteristic symptoms are absent that the likelihood of error occurs. The previous occurrence of malarial poisoning distinctly modifies the symptoms and course of the disease; we have no reason, however, to believe that the malarial poison does more than this. Some writers have described a form of this disease to which they have given the name "typho-malarial fever"; but we think it is a mistake to invent any such compound title for the disease, and likely to lead to errors in treatment. If we have any suspicion that the disease may be typhoid fever, it is very much better to treat the patient as if he were undoubtedly suffering from this disease, and in accordance with this idea to be exceedingly cautious in the administration of drastic purgatives, and also in the matter of diet. This line of treatment can do no harm if the attack ultimately turns out to be one of remittent fever or some other disease; whereas, if the disease is really typhoid fever, the heroic treatment sometimes employed to cut short a supposed attack of malarial fever may do the greatest possible harm.

There is not much difficulty in distinguishing typhoid from intermittent fever. The paroxysms of ague, the profuse sweating, and the fall of the temperature to the normal between the paroxysms, will determine the diagnosis. We must remember, however, that when typhoid fever attacks people who have been subject to ague, the disease not unfrequently commences with what appear to be typical attacks of ague.

We do not propose to enter into the distinctions between typhoid fever, the specific fevers, and other general diseases, as these are fully treated of in the text-books on the subject; we may remark, however, that the diagnosis between this disease and acute miliary tuberculosis, or tubercular meningitis and enteritis, is often far from easy; and the same might be said about ulcerative endocarditis.

Prognosis.—The prognosis of typhoid fever in tropical countries is undoubtedly graver than in temperate climates. The case-mortality of the Asylum Board in London for the three years 1886–88 was 14·7 per cent. During the five years 1880–84 the admissions for typhoid fever in the European army in India numbered 2228, and of these 802 died,—a ratio of deaths to treated of 36 per cent., or more than double that of the home hospitals.

The prognosis in individual cases will be modified by special circumstances. The first consideration is whether the person can be kept quietly in bed in a hospital or private house, or whether he has to be constantly moved, as often happens during the progress of

a campaign. The risks are greatly increased if these movements be necessary. If the patient is seen early in the attack, and at once placed under favourable conditions as regards nursing and diet, a great point in his favour is gained. There is no disease in which the result depends more on careful nursing and unremitting care than typhoid. A sleepless night or a failure to administer nourishment at proper intervals often leads to a fatal issue. The difficulty of procuring suitable nourishment at some of the isolated and remote stations also tends to increase the danger of the disease. Milk may be hard to obtain, or of doubtful quality. With all the difficulties in the way of treating and feeding these cases, it is not surprising that typhoid fever in tropical countries is a specially fatal disease. In campaigns the mortality from this disease is enormous, and with all the difficulties inseparable from the movements and commissariat of an army on active service, it must, we fear, remain so.

In giving a prognosis, we must take all the above named circumstances into consideration. The same causes which prove fatal at home will do so abroad. High temperature, lung disease, hæmorrhage, excessive diarrhœa, cardiac debility, and the presence of other complications, must all receive our careful attention in forming an estimate of the chances of recovery. While we know that recovery often takes place in apparently the most hopeless cases, we must also remember that danger exists as long as the fever continues, and even for the first few days of convalescence. Any one of several complications may determine a fatal issue in a case which, to all intents and purposes, seemed doing well. Our prognosis should, therefore, always be most guarded.

Bacteriology.

Eberth's Bacillus.—It seems now definitely settled that there is present in all cases of typhoid fever a special micro-organism; how far this has to do with the actual causation of the disease remains still to be proved.

Eberth, in 1880, first drew attention to a bacillus which he believed was always present in the diseased organs of those suffering from typhoid fever. Eberth, Klebs, and Koch were able to demonstrate the presence of this bacillus in the spleen, mesenteric glands, and Peyer's patches of patients who had died from this disease.

Gaffky, four years after Eberth's discovery, described minutely the same bacillus, and called it after its discoverer, "Eberth's bacillus." Gaffky stated that this bacillus is about three times as long as it is broad, its length corresponding to about the third part of the

Fig 1

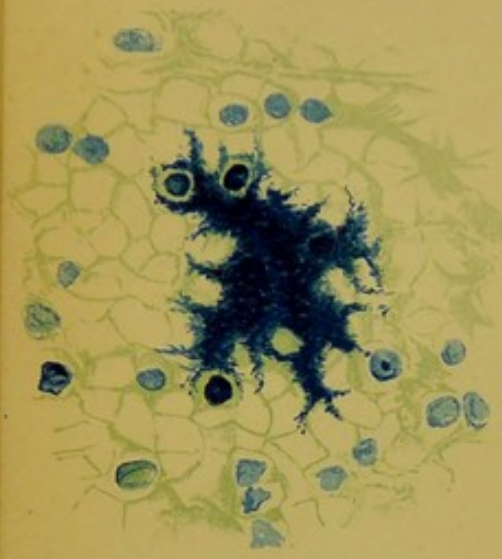

Fig. 1.—Section of spleen from a case of Typhoid Fever (× 500) and stained with methylene blue, showing the collections of masses of bacilli.

Fig. 2.

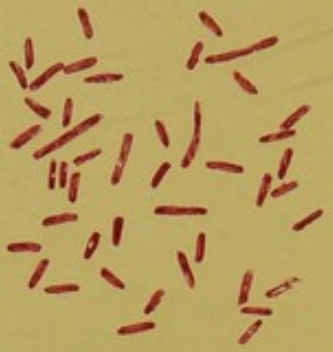

Fig. 2.—Typhoid bacilli (× 1500), and stained with fuchsin.

Fig 4

Fig. 4.—A plate cultivation of the Typhoid bacillus

Fig. 3.

Fig 3.—Growth of Typhoid bacilli in gelatine from a Stab cultivation.

Fig. 5.

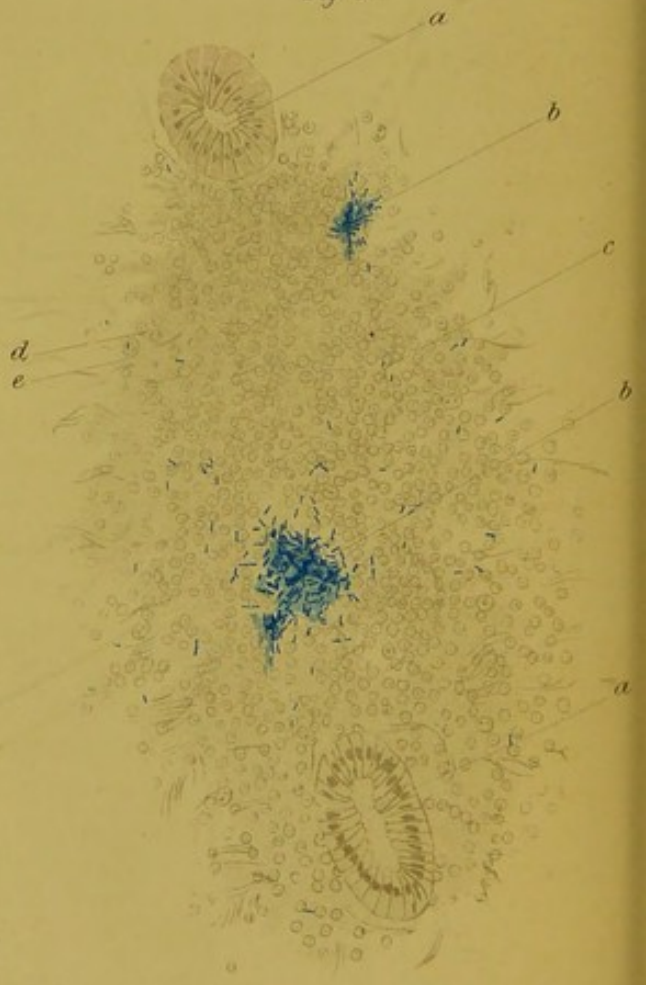

Fig. 5.—Section of typhoid ulcer of intestine—ileum, specially stained with gentian violet. (× 450.) (After Woodhead.)

a. Gland tubules. *b*. Groups of typhoid bacilli.

c. Single bacilli. (These are represented rather longer and not quite rounded at the ends as they appeared in the specimen.)

d. Small lymphoid cells in reticulum. *e*. Large endotheloid cells.

diameter of a red blood corpuscle, and that the ends are distinctly rounded off. It was usual to describe a spore formation in its centre, but it is now generally believed that this appearance was misinterpreted, and that it is in reality due to degenerative changes. Gaffky found this characteristic bacillus in twenty-six out of twenty-eight cases of typhoid fever, and his results have been fully corroborated.

The bacilli are present in the diseased part of the intestine, the spleen, the mesenteric glands, the liver and the kidney, though more rarely in the latter organ.

The bacilli are collected together in masses, and are not evenly distributed through the organs implicated. This will be seen on reference to Plate II. Fig. 1. As these masses are isolated, the presence of the characteristic bacilli may be absent in many of the microscopical sections.

The bacilli are easily stained in cover glass preparations by any of the ordinary aniline dyes, but sections require some care. The best results are obtained by staining them for twenty-four hours in Lœffler's methylene blue, or for a few minutes in warm carbol fuchsin.

Plate II. Fig. 2 shows the appearance of the typhoid bacilli magnified 1000 diameters, and stained with fuchsin.

Cultivation of the Bacillus.—The micro-organism can be cultivated outside the body. The easiest method to demonstrate the presence of these bacteria in the organs of fatal cases, is by planting small quantities of the tissue, as soon after death as possible, on the surface of any of the common nutrient media, as, for example, agar-agar.

For instance, in the case of the spleen, this organ should be removed from the body as soon as possible after death, an incision should be made through its centre by a sterilised knife, and particles of the spleen pulp should then be removed from the surface of the incision with a sterilised platinum needle and spread over the solid surface of the nutrient medium. At the end of twenty-four hours, if these tubes containing the nutrient medium are kept at the temperature of the blood, a copious growth of the bacilli will be found. Plate II. Fig. 3 shows the method of growth of the bacillus from a stab cultivation, and Fig. 4 a plate cultivation of the bacillus.[1]

If cultivated on nutrient gelatine, liquefaction of the jelly does not occur at any stage of the culture. Up to the present date, attempts to reproduce the disease in animals by inoculation from the pure cultures or from the typhoid stools have not been entirely successful. (See footnote, p. 225.)

[1] We are indebted to Mrs. Bruce, of Netley, for the drawings from which Figs. 1, 2, 3, and 4 of Plate II. are executed.

The presence of the bacilli has been demonstrated in the dejecta of patients suffering from the disease, especially when the case has advanced to ulceration and sloughing of Peyer's patches and the solitary glands. Although numerous papers have been written in which the authors claim to have detected the specific bacillus in suspected drinking water, yet the extreme difficulty experienced in separating the typhoid bacillus from other species, apart from the patient affected, is so great, that we should hesitate before accepting this as a fact until very conclusively proved.

Relation to the *Bacillus coli communis*.—It would seem right at this part of the article to refer to a recent review of the etiology of typhoid fever in the *British Medical Journal* of June 11, 1892. The great resemblance in character and habits of the *Bacillus coli communis* to Eberth's bacillus is here discussed. According to the writer, the recent discoveries in bacteriology seem to indicate that under certain circumstances all the characters, morphological and biological, of the organism regarded as peculiar to typhoid fever may be assumed by one of the common organisms present in healthy stools. Hueppe in 1887 came to the conclusion that under certain disturbing influences these organisms might acquire virulent properties. The question then comes to be, are the *Bacillus coli communis* and Eberth's bacillus simply varieties of the same species? in other words, is Eberth's bacillus the *Bacillus coli communis* modified in its passage through the body?

If it were proved that Eberth's bacillus, which is now almost universally admitted as the typhoid bacillus, and the *Bacillus coli communis* were of the same species, many questions as to the etiology of this disease would no doubt be simplified. But, on the other hand, such a theory is opposed to our present knowledge of these micro-organisms in general, and we do not believe that under any circumstances a harmless bacillus can be changed into a virulent one, and one capable of producing a specific disease.

Our knowledge, then, seems to lead us to a point where we can demonstrate that a definite micro-organism exists, practically speaking, in every case of typhoid fever, and that this organism has never been found in any other disease; but it remains to be proved that the bacillus in question actually produces the disease when introduced into the body.

To make the chain of evidence complete, we should be able to isolate the bacillus, cultivate it outside the body, and on introducing the pure culture into another body, produce the original disease. Until the bacillus is shown to fulfil these conditions we cannot with

absolute certainty attribute the incidence of typhoid fever solely to the presence of this micro-organism. At the same time, the fact of its constant presence, and its localisation in the diseased tissues, render it highly probable that this micro-organism is the cause of the disease.

Post-Mortem Appearances.

Identity of the Lesions in all Countries.—The post-mortem appearances are typical. It has only been by a careful study of these that the identity of this disease has been recognised in different parts of the world. It is most essential that we should be thoroughly acquainted with the exact post-mortem appearances, and avoid the mistake of returning as typhoid fever cases in which ulceration of the intestines has existed from other causes, combined during life with a high temperature. We have previously stated that there is no difference in the lesions found after death from tropical typhoid fever and those of this disease as it is met with in temperate climates.

Lesions in Ileum and Colon.—We find the characteristic evidences of this disease in the lower part of the ileum and the upper part of the colon.

The lymphatic glandular tissue of the intestines are the parts primarily affected. Peyer's patches and the solitary glands which are formed by this tissue, are therefore the seats of the morbid changes. The first change in connection with the inflammatory process is a general catarrhal condition of the mucous membrane of the parts affected. More especially is this noticed in the mucous membrane covering Peyer's patches. These agminated glands become swollen and raised above the surrounding surface. The swelling is sometimes so marked that the tissue bulges over the margin of the patch, and resembles a fungus growing on the mucous membrane of the intestine. This swelling is first of a rosy pink hue, but becomes later of a white or creamy white colour.

The inflammatory process is at its height about the eighth day, and either undergoes resolution when the fever aborts, or, what is more common, passes on to the second stage. This is one of ulceration. The swollen Peyer's patches become necrotic, sloughs form, which may either be shed *en masse* or piecemeal. The sloughs become ultimately stained by the bile, and appear of a dark yellow or brown colour; these are cast off in the third week, leaving a clean ulcerated surface, which in favourable cases goes on to repair and cicatrisation.

The three stages through which these patches run are represented diagramatically in Fig. 30 (after Thierfelder).

The process which goes on in the solitary glands is of the same nature. The glands become swollen and enlarged, and feel like shot under the finger. The mucous membrane covering the glands ulcerates, and the gland discharges its contents, leaving an ulcer, which goes on in favourable cases to repair.

The whole of the intestinal tract may be implicated by these morbid changes, but usually they are confined to the ileum and cæcum, especially the part of the intestine in the vicinity of the ileo-cæcal valve. The glands near this structure seem the first to be attacked, the more distant parts being involved later. When relapses occur it is from the invasion of fresh gland tissues.

Characters of the Typhoid Ulcer.—The ulcers met with in the intestine in typhoid fever are most characteristic. They are situated in the position of Peyer's patches and the solitary glands. Peyer's patches are from one to two inches long, and have their long axes coincident with the long axis of the gut. The edges of the mucous membrane are loose and undermined; this is well seen when the parts of the intestine in which these ulcers occur are floated in water.

The base of the ulcer is not, as a rule, thickened to any extent. The ulceration is found on that side of the intestine remote from the mesenteric attachment. The ulcers formed by the death of the solitary glands are circular in shape and of small size, and are dotted about the intestine. They are also met with in the large intestine, chiefly in the cæcum. The appearance of a section of the typhoid ulcer, from which the slough has separated, is well shown in the accompanying illustration (Fig. 30, *c*).

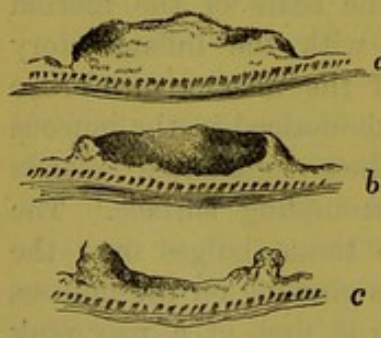

FIG. 30.—Diagram to represent the various stages of the typhoid lesion in Peyer's patches. (After Thierfelder.)

a, Section through the patch in the early or swollen stage.

b, Stage at which a line of demarcation is forming between the slough and the adjacent tissue.

c, Ulcer from which the slough has separated.

The typhoid ulcer heals by granulation from the floor of the ulcer. This process does not commence in most cases till after the end of the third week, and takes some days to accomplish. The covering epithelium is formed from the margins of the ulcer.

There is no contraction or puckering after the healing of a typhoid ulcer, and no thickening of the base. The cicatrisation of the ulcer does not lead to contraction of the calibre of the gut. The ulceration of

the gut accompanying typhoid fever must be distinguished from that caused by tubercular disease and dysentery.

The floor and edges of the tubercular ulcer are thickened, the ulcerations extend transversely round the gut, and the floor of the ulcer is nodular and irregular. No free margin of the mucous membrane exists. The tubercular ulcer rarely heals. There is no tendency to perforation, owing to the thickening of the base.

From dysenteric ulceration, the typhoid ulcer will be distinguished by the following points of difference. Dysenteric ulceration is usually found in the large intestine; it is irregular in shape, often affecting large patches of mucous membrane, and not limiting itself to the lymphatic tissues.

When death from typhoid fever results from hæmorrhage, it does so by extension of the ulcerative process to the deeper parts involving the walls of an artery, though it is often difficult to demonstrate this opening on post-mortem examination.

Perforation occurs by ulceration through the entire coats of the gut, thus allowing the passage of the contents of the intestine to escape into the peritoneal cavity, lighting up fatal peritonitis.

The natural process by which the surfaces of the gut are agglutinated, and perforation prevented, is not uncommon.

Lesions of other Organs.—The *mesenteric glands* are generally enlarged. Probably this is secondary to the lesions involving the lymphatic glandular tissue of the intestines. The mesenteric glands are soft and vascular. As a rule these glands undergo a process of resolution at the termination of the disease. They may, on the other hand, become caseous, or slough, and break down; the latter process is, however, rare.

The *spleen* is nearly always enlarged, of a dark colour, very soft, and friable.

The *kidneys* and *liver* are usually increased in size, and there is degeneration of the glandular epithelium.

The *heart* is pale, and the walls are thin and soft.

The *lungs* usually show signs of a certain amount of congestion and inflammation at the base, and the bronchial tubes show evidence of a general catarrhal condition.

Prophylaxis.

The objects to which our preventive measures must be directed are the following:—(*a*) The destruction or disinfection of typhoid discharges; (*b*) the prevention of the diffusion of the infective agent by air, water, and milk; (*c*) the lessening of the susceptibility of the individual to the infection; (*d*) the removal of unsanitary conditions

capable of preserving the vitality or fostering the multiplication of the infective agent.

Destruction or Disinfection of Typhoid Discharges.—Although we have been compelled to admit that the dependence of typhoid fever on Eberth's bacillus has not been conclusively demonstrated, the evidence in favour of the doctrine of a specific contagium is, nevertheless, so strong that we are bound to accept it as the basis of our prophylactic measures. That the infective agent is contained in the stools of the patient suffering from the disease, is no longer a matter of dispute. Outbreaks of the disease have been repeatedly traced to the contamination of drinking water by typhoid stools. Whether the excrements, when newly passed, are infective, is, perhaps, open to some doubt. There is good reason for believing that their infective properties become intensified outside the body, and that heat, moisture, and the exclusion of free access of air are conditions favouring this increase in the infective properties of the stools.

Bearing these facts in mind, our primary duty is to see that the dejecta, and all linen, etc., stained by the discharges, are rendered innocuous. We therefore strongly recommend that, when practicable, the typhoid stools should be burnt; and for this purpose it is best to mix them with sawdust. If this is impossible, the stools should be treated, as soon as they are passed, with the following solution, which has been recommended by the Local Government Board for the disinfection of cholera stools:—½ oz. corrosive sublimate; 1 oz. hydrochloric acid; 5 grains commercial aniline blue,—to 3 gallons of water. They should then be buried, at least, 2 feet below the earth's surface. The linen soiled by typhoid discharges should be soaked in the above solution, and afterwards placed for some hours in water. Articles of small value should be burnt. The value of the application of dry or moist heat by steam as a germicide must be remembered in this connection; and if suitable apparatus is at hand, advantage should be taken of it.

We have endeavoured to indicate how the dejecta, if imperfectly buried, can become a source of danger, and with what facility Eberth's bacillus can be carried by the wind; and we have questioned whether many of the disinfectants in common use are really germicides.

The stools should be buried far away from any water supply, as we know that the subsoil water can carry the micro-organisms.

The sites of the trenches in which, in the dry earth system, the excreta are buried, should be cultivated; and as in hot countries this usually entails irrigation, chemical changes in the soil are hastened. A very good example of the imperfect application of the dry earth system is given by Surgeon-Captain Nichols,

Army Medical Staff; it shows one method by which the specific germ may be imported into barracks. This officer found that the dry earth used for admixture with the fæces in the latrines in barracks was carried by the same carts which removed the "soil," and was dug in close proximity to the place where the filth was buried. While we fully believe that the dry earth system is admirably suited for tropical climates, we wish to emphasise that it has special dangers of its own, and requires careful supervision.

Measures for Preventing the Diffusion of the Infective Agent.—The channels by which the infection gains admission into the system in tropical countries are, as we have pointed out, somewhat obscure. This should only make us the more careful to guard every possible access by which the virus may effect an entrance. What we lose in precision from our defective knowledge of the etiology of the tropical disease must be compensated by the comprehensiveness of the prophylactic measures we adopt. Every possible source of infection should be sought out, and every channel of conveyance strictly supervised. In Europe one important medium of infection is recognised to be the impregnation of the air of habitations with sewer gas. The very factors which go to make up the comfort of the dwelling-house appear to increase the danger arising from this cause. A warm house, with perfectly fitting doors and windows which prevent the entrance of air from without, has an aspirating effect on the drains; and unless these are carefully trapped and disconnected, the gases which they contain will be drawn into the rooms. If the sewers happen to contain the excreta of a typhoid patient, the danger to those who breathe the air is obvious.

Sewers are not in common use in most tropical countries, the dry earth system of disposing of the excreta being that which is in use. Where sewers, however, do exist, it is obviously of the greatest importance to see that they are perfectly trapped, ventilated, and disconnected.

We have already alluded to the great probability that the specific organism of this disease present in the soil may be spread by the winds, and in this way become diffused through the breathing air, and contaminate water and articles of food, such as milk, which may serve as cultivating fluids for its multiplication, or, at least, as media for its introduction into the system.

Our only means of prevention in this case is to destroy or render the germ inert immediately after it leaves the body. This is to be effected by burning the excreta, or by resorting to the method of disinfection which we have already described.

The fouling of the water supply by the introduction of the specific bacillus is one of the principal means by which this disease propagates itself, and all sources of contamination must be carefully guarded against. Surface wells are a recognised danger; and every effort should be made to secure a pure water supply from a distant and uncontaminated source. Vague ideas often exist on the subject of the filtration of water, and too much reliance is placed on the mere passage of the water through a filter. Unless great care is taken, the small filters so commonly used become an actual danger. If these are used, the filtering medium must be frequently cleaned and aërated, otherwise it is better to discontinue their use entirely. The best safeguard is to boil suspicious water for a few minutes before filtration.

Milk, from its adulteration with water, or from the washing of the vessels which contain it with impure water, may readily be the means of infection. Suspicious milk should be rejected or treated in the same way as suspicious water, that is, boiled.

Measures for Lessening Susceptibility.—Among the causes which may exercise a predisposing influence, the diet of Europeans in hot countries may be reckoned, and this requires attention.

It would seem reasonable that young Europeans coming out to India and other hot countries, should not indulge in the same full meat diet that they are accustomed to in cold climates. It is not needful, and a much less stimulating diet would probably be more conducive to health. The intestinal irritation set up by an improper diet in hot climates has probably an important bearing on the liability to attack in new-comers. Diarrhœa and congestion of the bowels is set up, a state of things highly favourable to the reception and development of the specific contagium. On the same grounds the free use of alcohol is to be condemned. If required, alcohol should only be taken in small quantities with the meals, and very largely diluted.

Fatigue induces a state of the constitution which is favourable to the development of febrile conditions, and lessens the power of resistance to disease. Severe exercise in the sun should therefore be avoided, and the clothing worn should be suitable to the climate.

But of all the points in connection with the predisposition to this disease in tropical climates two stand out prominently, viz. age and length of residence in the country.

It may not be practicable to prevent individuals going to tropical countries at the susceptible age. With soldiers this would obviously be impossible, for, as a result of the short service system, our army is now largely composed of young men.

All we can attempt to accomplish is to place Europeans at first under climatic conditions resembling as near as possible the climates from which they have come, and carefully to preserve them by every means in our power from the chance of infection. The process of acclimatisation should be as gradual as possible.

The despatch of drafts and newly-arrived regiments to hill stations immediately on arrival in India is a step in the right direction. In addition to the careful sanitary supervision of these stations, the journey up country must be conducted with minute precautions. The great danger of infection from the water supply of railway stations and rest camps must be considered, with the view of insuring the purity of the drinking water. The liability of infection of the soil of rest camps must also be borne in mind, and the sites of these camps changed from time to time.

Measures of General Sanitation.—That the essential cause of typhoid is not a product of healthy excreta, or of ordinary fæcal decomposition, is proved by the fact that in tropical countries the natives, who show a total disregard of the most obvious sanitary considerations, appear to be less subject to the disease than Europeans who live under conditions immeasurably more favourable. The European stations most severely affected are not always those in which sanitary defects are most obvious. Nor can we always trace any strict relation between the prevalence of the disease in native communities and the hygienic circumstances in which they live.

In Europe, as well as in the tropics, it would seem no uncommon thing for people to live amidst the most insanitary surroundings for a considerable time without the appearance of typhoid fever, and that it is not till a case of the disease is imported that an epidemic breaks out.

Tyndall quotes[1] a letter from the *Times* of January 17, 1876, in which we have a remarkable illustration of this fact:—

> In one part of it (Edinburgh), congregated together, and inhabited by the lowest of the population, there are, according to the Corporation return for 1874, no less than 14,319 houses or dwellings—many under one roof—on the flat system, in which there are no house connections whatever with the street sewers, and, consequently, no water-closets. To this day, therefore, all the excrementitious and other refuse of the inhabitants is collected in pails or pans, and remains in their midst, generally in a partitioned-off corner of the living room, until the next day, when it is taken down to the streets and emptied into the Corporation carts. Drunken and vicious though the population be, herded together like sheep, and with the filth collected and kept for twenty-four hours in their very midst, it is a remark-

[1] *Floating Matters of the Air.*

able fact that typhoid fever and diphtheria are simply unknown in these wretched hovels.

While facts such as these prove that typhoid fever is not the necessary consequence of insanitary surroundings, experience shows that when the specific germ finds an entrance into a community living under such conditions, the disease spreads widely, and often persists for a long time. A soil surcharged with the products of animal and vegetable decomposition, and polluted with excreta, and accumulations of refuse and excrementitious matters in or near dwelling-houses, serve as lurking places, if not as breeding places, for the germ, from which it may become diffused by water, air, and milk, and thus give rise to the infection. Overcrowding and defective ventilation, with the habits of uncleanliness with which these conditions are too often associated, will further favour its spread and increase its fatality. It is therefore our obvious duty, in our efforts to prevent typhoid fever, to improve the general sanitary state of a community, especially as regards the disposal of excreta, the supply of pure water, the prevention of the pollution of the surrounding soil, and the proper construction and ventilation of dwelling-houses.

But while we thus insist upon the great need of attending to the sanitary surroundings of habitations, we wish, in conclusion, to say that the application of general measures of sanitation does not fulfil the primary indication of prophylaxis in this disease.

The mere tidiness of a town or station is not all that is required. The germ of the disease must itself be dealt with.

As Professor Tyndall graphically remarks: "As surely as the fig comes from the fig, the grape from the grape, the thorn from the thorn, so surely does the typhoid virus increase and multiply into typhoid fever;" and therefore, he continues, "it is not on bad air and foul drains that the attention of the physician will primarily be fixed, but upon disease germs, which no bad air or foul drains can create, and which may be pushed by foul air into virulent energy of reproduction."

We have to be on the alert to limit a disease that is very insidious in its transmission. This we firmly believe can only be accomplished by destroying the micro-organism upon which the disease depends and by which it is propagated.

The thorough destruction of the specific microbe in the stools immediately after their passage, and the disinfection, in its widest sense, of all articles soiled with typhoid discharges, is the key-note in the prevention of the disease.

CHAPTER VI.

MALTA FEVER.

BY SURGEON-CAPTAIN DAVID BRUCE, M.B., ARMY MEDICAL STAFF.

Synonyms.—Mediterranean fever (various writers); Gastric remittent and Bilious remittent fever (Marston, 1861); Mediterranean gastric remittent fever (Chatres, 1865; Boileau, 1866); La febbre gastro-biliosa (Gulia, 1871); Fæco-malarial fever (Donaldson, 1876); Intermittent typhoid (Borrelli, 1877); Adeno-typhoid (Cantani); Febris complicata (Veale, 1879); Febris sudoralis (Tomasselli, 1880); Pythogenic septicæmia (Moffet, 1889); Rock fever, Neapolitan fever, etc.

Definition.—A disease of long duration, characterised by fever, profuse perspiration, constipation, relapses almost invariable, often accompanied or followed by pains of a rheumatic or neuralgic character, sometimes swelling of joints or orchitis. On post-mortem examination enlargement and softening of spleen, congestion of the various organs, no enlargement or ulceration of Peyer's or other intestinal glands, and the constant occurrence in various tissues of a definite species of micro-organism.

Geographical Distribution.—This fever, which does not appear to occur in England, has a wide distribution along the shores and among the islands of the Mediterranean. That the fever as it occurs in Malta is identical with the so-called Rock fever of Gibraltar, there can be no doubt, ample opportunity having been afforded at the Royal Victoria Hospital, Netley, of comparing cases from both places. I have also had the opportunity of studying in the Naval Hospital, Malta, cases of fever contracted in many of the Mediterranean harbours visited by the British fleet, and have completely convinced myself of their identity with the disease under consideration.

In Italy it has been described as occurring at Naples and other towns along the coast. Cagliari in Sardinia, Catania in Sicily,

Candia in Crete, may also be mentioned as towns in which this fever appears to be endemic.

On further investigation, and when the characters and specificity of this form of fever are better recognised, I have no doubt the area of its distribution will be much extended, and many cases at present confounded with typhoid and malarial fevers found to be identical with it.

Etiology.—1. *Bacteriology.*—Like typhoid and other infectious diseases, Malta fever is due to the introduction into the system of a specific poison, which, until within the last few years, has been generally looked upon as arising directly from the decomposition of animal and vegetable matters in places where insanitary conditions prevail. An intermediate stage in the causation of such infectious diseases is now recognised in the presence of micro-organisms which are supposed to be evolved by the insanitary conditions, and in their turn to give rise to disease. The micro-organism associated with Malta fever was first described in 1887, and for want of a better name is called the micrococcus of Malta fever; and as its demonstration is very valuable as a means of diagnosis, I shall now proceed to describe how it may be obtained, and what are its morphological and cultural characters.

Unhappily for practical purposes it is difficult to demonstrate its presence during life, as it has not been found in the circulating blood.

The organ in which it occurs in greatest numbers is the spleen; and it is only by withdrawing a small quantity of splenic blood or pulp, by means of a sterilised hypodermic needle, that its presence during life has, up to the present, been proved.

In fatal cases, however, the micrococcus is readily obtained from the spleen by means of cultivation on suitable media.

This organ ought to be removed from the body as soon as possible after death, protected from contamination by being wrapped in a cloth saturated with a solution of corrosive sublimate (1 in 1000), and taken to a room set apart as a laboratory. In transferring traces of the splenic pulp to tubes containing the nutrient material, certain precautions should be taken. Three extensive cuts, the second in a plane at right angles to the first, and the third at right angles to the second and parallel to the first, are made by three knives previously thoroughly sterilised by heat. A platinum needle heated to redness before each inoculation is then used to convey a small portion of the pulp from the depths of the third cut to the solid nutrient jelly. The test tubes containing the inoculated medium should then be placed in an incubator, so as to keep them

at the temperature of the blood. This, of course, necessitates the use of a nutrient medium which remains solid at this temperature, and this is found in that known as agar-agar.

In this way, up to the present, thirteen fatal cases of Malta fever have been examined; and in twelve, one and the same micro-organism, which I shall shortly describe, was demonstrated. The failure to grow the micro-organism in the thirteenth case was found afterwards to be due to a nutrient medium of too alkaline reaction having been used for the cultivation experiments.

These cultivation experiments were made from the centre of organs removed in most cases within ten or fifteen minutes of death. I further made the attempt to grow the micro-organism from the tissues of the living subject, and succeeded in two cases.

The micrococcus of Malta fever is round or slightly oval in form, and measures in dried preparations about ·33 μ in diameter. It is therefore a very minute organism, and requires a magnifying power of 1000 or 1500 diameters to see it with any distinctness. Viewed in a drop of water, unstained, the microbes are seen as bright points in active molecular movement, the great majority of them single, a few in pairs, but never in chains. Like most micrococci, they possess no power of spontaneous movement. They can be readily stained in a watery solution of gentian violet; but Gram's method is not applicable, as they become decolourised in the process.

The best medium for the cultivation of this species is ordinary $1\frac{1}{2}$ per cent. peptonised agar-agar beef jelly. The inoculation into this medium may be made either as a stab culture, or by spreading the material over the surface of the agar-agar, which has been solidified in a sloping position so as to give a larger surface. In a stab cultivation made from the spleen of a fatal case or from a previous culture, no change takes place in the appearance of the jelly for several days. At length the growth appears as minute pearly white spots scattered round the point of puncture, and minute round white colonies are also seen along the course of the needle track. After some weeks the colonies on the surface grow larger and join to form a rosette-shaped growth, while the needle track becomes strongly marked, solid-looking, and yellowish-brown in colour, with serrated edges. After the lapse of some months the growth remains restricted in area, and its colour deepens to buff.

When growing on the sloping surface of agar-agar and examined by transmitted light, the appearance of the colonies is somewhat

different. At the end of nine or ten days, if kept at 37° C., some of the colonies are as large as No. 4 shot. They are round in shape, with an even contour, slightly raised above the surface of the agar-agar, and smooth and shining in appearance. On holding up the tube and examining such colonies by transmitted light, the centre of each is seen to be yellowish in colour, while the periphery appears bluish-white. On looking at the same colonies by reflected light no appearance of yellow can be seen, they then simply appear to be milky white in colour. The separate colonies on the surface of the agar-agar do not extend indefinitely, and after a couple of months are found to be no larger than hemp seeds.

The time which elapses before the colonies of this micrococcus can be seen by the naked eye on the surface of the agar-agar is fairly constant; kept at a temperature of 25° C., this period may be stated to be seven days; at 37° C., about half that time. When stab cultivations are made into 10 per cent. nutrient gelatine, and kept at 22° C., little or no growth takes place; after a month the needle track has become slightly developed, and on the surface can sometimes be seen a minute smooth white growth not larger than a pin-head. No liquefaction of the gelatine takes place.

I have not succeeded in making plate cultivations of this species on account of the extreme slowness of its growth at the temperature at which this medium remains solid.

Planted on boiled potato and kept at blood temperature, no apparent growth takes place.

I shall now state briefly the result of experiments on the transmission of Malta fever to animals by the inoculation of pure cultures of the micrococcus.

Up to the present the only animal found susceptible to the disease is the monkey, the animals commonly employed for such experiments, as mice, guinea-pigs, and rabbits, having proved insusceptible.

The species of monkey with which successful results have been obtained is that known as the Bonnet Monkey, and the material used for inoculation has been a portion of a colony removed from an agar-agar tube by means of a sterilised platinum needle, and rubbed up in a small quantity of distilled water or nutrient broth. This is injected into the subcutaneous tissue of the monkey by means of a sterilised hypodermic needle after the part has been shaved and thoroughly cleansed.

Monkeys inoculated in this way rapidly develop febrile symptoms with high temperature, and, as a rule, die in from thirteen to twenty days.

The pathological changes on post-mortem examination are similar to those which occur in man; and cultivations made from the various organs reveal the presence of the micrococcus of Malta fever in a state of pure culture.

In regard to the important question as to how this micro-organism gains access to the human subject, whether by the air, in the drinking water, or in the food, absolutely nothing is known up to the present; and on account of the high temperature required for its growth, the length of time which elapses before the colonies appear, and the absence of any well-marked morphological or cultural characteristics, the search for it outside the body will be very difficult, if not impossible.

2. *Age and Sex.*—As the great majority of the English soldiers stationed at Malta are young men, statistics in regard to age are of little value. I may, however, state that sex seems to have little or no influence, and that youth, as is natural, is a predisposing factor. If the disease occurs among the very young, it is difficult to be certain of the diagnosis, and it is probably often overlooked. The opinion of the native practitioners is that the greatest number of cases occur between the age of 6 to 30, less frequently from 2 to 6 and from 30 to 50, and very rarely above 50. This may be true for the native population who live all their lives in the infective area, without being true for strangers arriving there. In my opinion, given a susceptible constitution, any age is liable to this fever, but that from a variety of reasons the liability is exaggerated in those under 35.

3. *Mode of Prevalence.*—In Malta this fever is endemic, and never altogether absent; but now and then the cases become so numerous as to constitute epidemics. Some of these outbreaks have been attributed to the general immaturity and want of acclimatisation of the men of some regiments landing from England during the unhealthy season; in others, the cause has been assigned to the insanitary condition of certain of the barracks. Be this as it may, it is also certain that Malta fever attacks officers and their families living in large, well-ventilated houses probably in as large a proportion as it does soldiers in the more crowded barrack-rooms.

4. *Months and Seasons.*—In regard to the months and seasons in which Malta fever is most prevalent, the following chart (Fig. 31) represents the number of cases admitted during each month for eleven years from a garrison of some 3000 soldiers. For the sake of comparison, the admissions for enteric fever during the same period are represented by a dotted line. From these it will be seen that

Malta fever is a disease of summer, most cases occurring in July, whereas enteric fever is a disease of autumn and early winter.

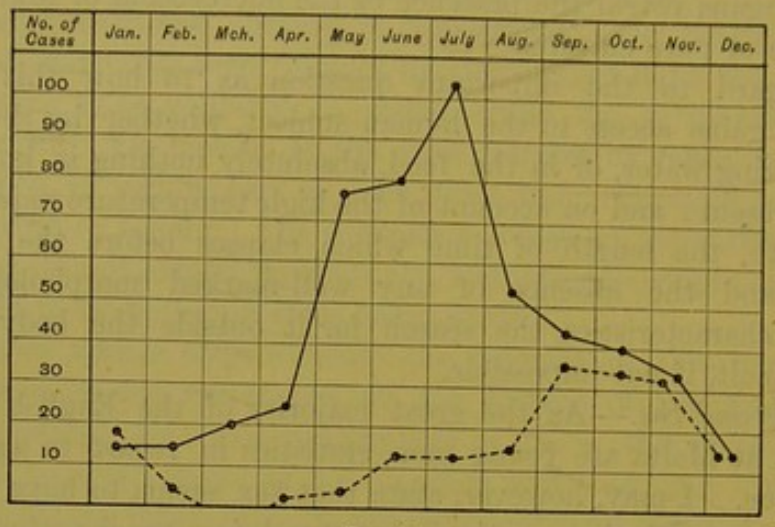

FIG. 31.

5. *Mode of Dissemination.*—Malta fever is never, as far as I know, transmitted directly from person to person. In the hospitals in Malta, although cases are to be found all the year round scattered through the various wards, there is no evidence that it has been communicated to any of the other patients in a single instance. As I have before stated, it is impossible with our present knowledge to say whether the poison gains entrance to the organism by the respiratory or alimentary organs. The tendency is to believe that it floats in the air, especially in the neighbourhood of foul drains or other insanitary conditions, and so reaches the body by being breathed in. A bad smell, *per se*, cannot give rise to a specific fever, although it may lower the general health and so predispose to disease.

Since this fever is caused by a definite vegetable organism, it is difficult to picture such a particle rising into the air from fluid or solid substances in which it may be supposed to find a suitable soil for growth. On this account it is probable that Malta fever, like typhoid, is chiefly carried by means of the drinking water or other fluid or solid foods to which the contagium has gained access.

6. *Incubation.*—It is impossible to state definitely how long the period of incubation is, but it ranges probably from a few days to twenty or thirty. In an epidemic which broke out in the Verdala barracks in Malta, the first cases occurred six days after the arrival of the regiment. Again, instances are on record in which the disease has broken out in individuals on their arrival in England, fourteen and seventeen days after leaving Malta. But the important

point to note in this regard is, that there is no evidence to show that this period of latency is much longer than above stated, so that individuals who remain free from the fever for, say two months after leaving the infective area, may be considered safe from attack.

7. *Immunity from Second Attack.*—Some writers state that this disease is often manifested more than once in the same individual; and in the navy this opinion is held so strongly that officers and men who have once had Malta fever are not sent back to this station until a long time has elapsed. So far from one attack granting immunity, it is believed to predispose very strongly to a second and third attack. In a fever of such indefinite duration, cases of which may last two years, there is always the danger of mistaking a relapse for a separate attack. My experience leads me to the general conclusion that, as in many other infective diseases, one attack of this fever does as a rule confer immunity.

8. *Duration of the Fever.*—Soldiers show an average stay in hospital of nearly ninety days; but the length of the fever varies between very wide limits in different individuals. While some return to duty after twenty or thirty days, others again remain in hospital for as many weeks or more. One case which came under my observation was admitted to hospital in July, and with the exception of February and March he was in hospital suffering from this fever and its sequelæ until the end of the following year.

9. *Rate of Mortality.*—Not the least extraordinary feature of this fever is its low death-rate. From the result of my observations, I would put it as low as 2 per cent., which is very different from that which obtains in typhoid fever,

Clinical Description.—*Early Symptoms.*—A patient suffering from the fever, on being admitted to the hospital is usually loth to give any information in regard to his symptoms. By dint of patience you draw out of him that he has been feeling out of sorts for a week or two; he has had no interest in life; his appetite has been fickle; vague feelings of discomfort, as shivering, sickness, headache, and pains in his bones, have often been present, and to escape his now irksome and wearisome duties he has sought admission to hospital. For the first week or ten days he often suffers from sleeplessness and headache, which may vary from the mildest form to the most intense, very often frontal, and more rarely shooting from the occiput through the eyes. In these severe cases the face is usually congested, the anterior temporal arteries are seen pulsating, the ears are ringing, and epistaxis may occur. The tongue is usually covered with a thin yellowish-white fur; it is large and flabby, the edges and tip are red, and it is usually marked

laterally by the teeth. Congestion of the pharynx is often present. The appetite is absent; there is nausea, sometimes causing vomiting, and a feeling of weight and tenderness in the epigastric region. The bowels are constipated, as a rule, but errors in diet or excessive use of medicines may bring on attacks of diarrhœa. The stools are often streaked with blood. The spleen and liver are enlarged, and both may be tender on pressure. Tympanitis is uncommon, but may occur, as also may gurgling in the iliac fossa. During this time almost invariably a slight cough, with scanty expectoration, is developed; and on examination the breathing at the bases is found to be unsatisfactory, harsh, and creaking in character, with now and then a moist crepitation. Morning after morning you look in vain for rose-coloured spots on the abdomen, but you find that the patient is bathed in a most profuse perspiration, and a more or less abundant crop of sudamina is developed. He may have had a little delirium at night during this time; but this is rare, and is so slight as scarcely to call for remark. Unless there is severe headache or pain in the lumbar region, the patient during the first week or two usually professes that he suffers very little. At the end of this period the headache and acute symptoms usually disappear, and the long and monotonous period of the fever begins, a period which seems interminable alike to medical officer and patient. The patient's aspect is natural, but listless; his tongue is clean; he has a wish for solid food, which must often be denied, and his bowels require the stimulus of an aperient or enema for evacuation.

Later Symptoms.—The profuse perspiration still continues, and day after day he becomes weaker and loses weight, until he has scarcely power to stagger a few yards. His red blood corpuscles diminish in number, and his complexion changes from pale to sallow, and from sallow to dull clay colour. During this period his temperature often ranges high, but he professes to be quite unconscious of any change in his condition. He sleeps moderately well, has no delirium nor restlessness, is uncomplaining, and takes without any ill effect a large supply of fluid food and stimulants. The only variety in his condition is afforded by a rheumatic affection of the joints; one day it is his knee which is red, swollen, and intensely painful on being touched; a few days after it is a swollen and deformed wrist which he holds up for commiseration. Sometimes almost every joint in his body is attacked in this manner, or he may have intercostal neuralgia, sciatica, or an inflamed and swollen testicle. In this way many weeks are long drawn out; but at last his temperature fairly comes down to the normal, and he begins very slowly to improve, his blood corpuscles gradually regain their

normal number, his weight increases, and his strength is slowly restored. This is a clinical picture of an ordinary well-marked case; but the fever may occur in such a mild degree that the rise in temperature is the only morbid phenomenon. On the other hand, it may be so severe as to be absolutely indistinguishable from the most rapidly fatal case of typhoid.

Principal Symptoms in Detail. — *Physiognomy.* — The expression of the face, in the great majority of cases, may be summed up as being dull, listless, apathetic, anæmic. During the first onset, when there is severe headache, the face and exposed mucous surfaces are often congested, and the patient has an excited, restless look; but this soon disappears, and is replaced by lethargy. In long and severe cases, the face may become of a dull clay colour; and as the patient in this condition has left all hope behind, his expression is naturally despondent in the extreme.

Alimentary System.—The tongue is at first more or less thickly covered with grey or yellowish-grey fur; afterwards, as a rule, it is large, flabby, indented by the teeth, and covered with a thin translucent fur, except the tip and edges, which are red. In about 10 per cent. of the cases, the tongue becomes dry and brown for some days during the progress of the disease, and in a few the dorsum becomes fissured, with a little blood oozing from the fissures. In many cases the gums, as the disease advances, are found to be soft and spongy, and in some cases bleeding from the gums is noted. The fauces are usually somewhat congested. Vomiting, except in severe attacks of this fever, is not a marked symptom; but when this occurs the vomited matter is frequently streaked with blood. Nausea is much more commonly complained of than vomiting; and, as is natural, anorexia is almost invariably present at the beginning of the fever. Constipation is one of the marked features of this disease. In 65 cases in which the condition of the bowels was carefully noted, in 48 there was constipation throughout, and in 17 only did diarrhœa occur at any time during the illness. Murchison states that he noted constipation in 4 and diarrhœa in 93 out of 100 cases of enteric fever. It is evident in Malta fever that constipation is the rule and diarrhœa the exception. The reason of this is not far to seek. In post-mortem examinations of cases of enteric fever, ulceration and an inflamed condition of the lower end of the small intestine are found, which must necessarily have tended to diarrhœa; while in Malta fever no such ulceration or inflammation occur. Tympanitis is rare, and ascites still more so. The liver is usually slightly enlarged, sometimes painful on pressure, and slight jaundice may be noted occasionally.

Respiratory System.—Epistaxis occurs in about one-sixth of the cases. Cough at one time or another almost always occurs, and is marked in one half the cases; in these the expectoration is sometimes profuse, and is often streaked with blood. Even when there is no cough the breathing is found to be unsatisfactory, and on auscultation the respiratory sounds are harsh and bronchitic. Not uncommonly slight touches of pleurisy are experienced in the severe and protracted cases. Dyspnœa may be noted, and often without any grave condition being found on physical examination to account for it. Pneumonic consolidation is rare, not occurring in more than 2 per cent. of cases.

Circulatory System.—Palpitation of the heart becomes developed in many. The pulse may be said to range between 70 and 120. During the first period of the disease, although the temperature may range high, the pulse is frequently found to remain low, 80 to 90. As the fever progresses, and the heart becomes weakened, it rapidly increases, so that about the fortieth or fiftieth day 110 or 120 beats per minute are often registered. The highest pulse rate I have noted in a non-fatal case was 132 on the seventy-eighth day of disease.

Blood and Blood Glands.—Only in rare cases are particles of pigment to be seen on examining the blood with the microscope. The red blood corpuscles, as a rule, fall from 5,000,000 per cubic millimetre to about 3,500,000. The white blood corpuscles in most cases are found to be normal in number. The spleen is always enlarged, frequently painful on pressure, and in a few cases severe pain is complained of in this region.

The Temperature.—On looking over a series of temperature charts of this fever, the first thing which strikes one is their extreme irregularity.

It is also seen that in the great majority of cases this fever belongs to the continued type, the difference between the morning and evening temperatures being only from one to two degrees; but in a few cases there is seen a tendency in the fever to assume a remittent or even intermittent type, the temperature being normal, or slightly above normal, in the morning, and rising to 104° or 105° F. in the afternoon.

Mild uncomplicated cases show a curve touching 103° or 104° F. during the first week or ten days, and gradually sinking down to normal limits from the fifteenth to the twentieth day, when convalescence begins and recovery is uninterrupted.

But in ordinary typical cases of this fever the course is much less satisfactory, as will be seen by the following chart (Fig. 32), which

shows the long course of the fever, and the relapses which occur so frequently in this disease.

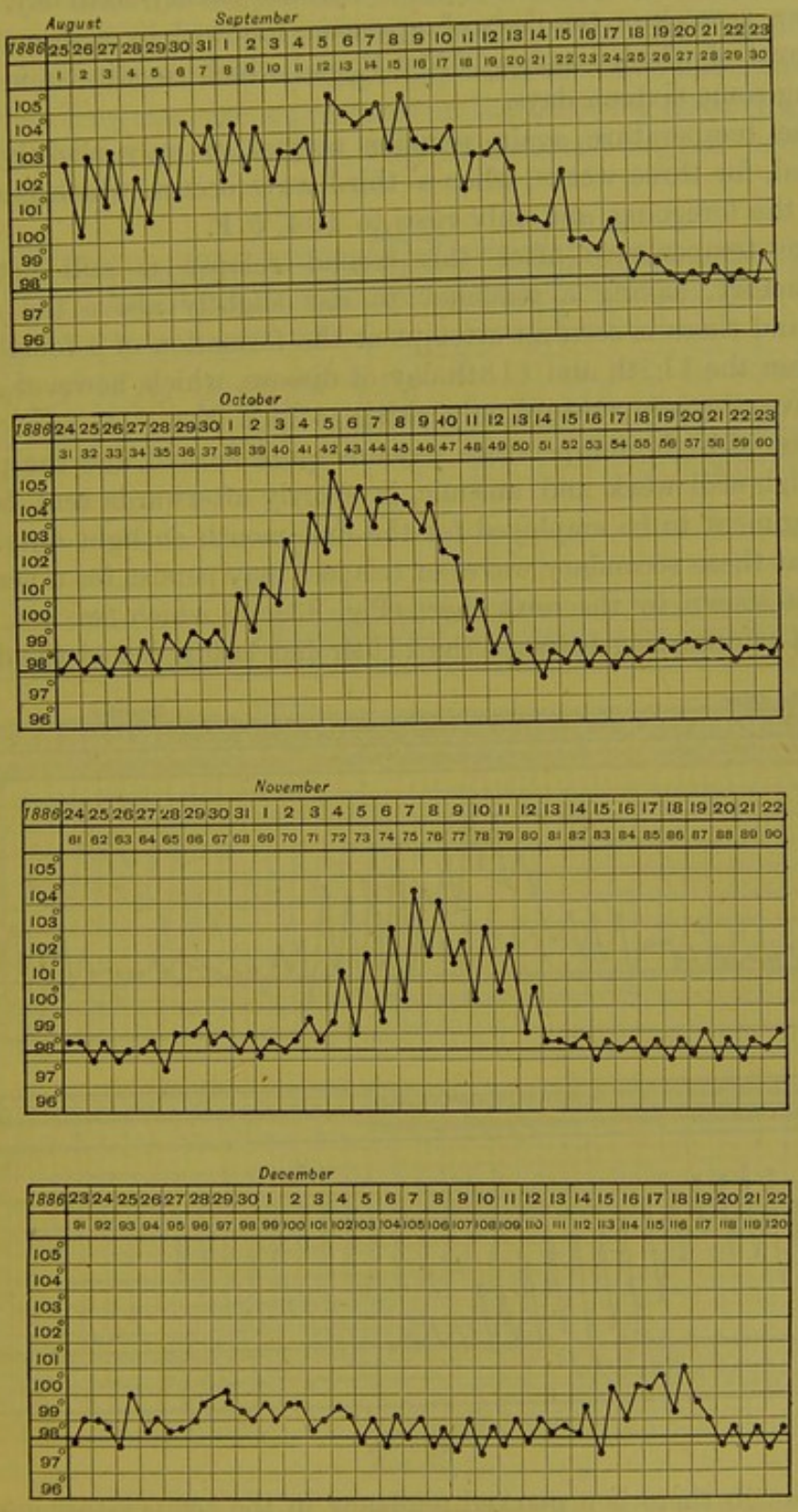

Fig. 32.

The first wave of temperature is seen in the foregoing chart (Fig. 32) to rise to 105°·4 F., and then to sink gradually down to the normal line, which is reached about the twenty-sixth day. Then follows a week or ten days of apyrexia, followed by a second wave, the crest of which touches 105°·6 F., and which constitutes a relapse lasting some sixteen days.

The temperature again remains normal, or only slightly above normal, for three weeks, when a third wave occurs, which is shorter than the foregoing, and only rises to 104°·5 F.

The temperature after this second relapse is still not quite satisfactory, having a tendency to rise slightly above the normal line, and there is seen an attempt at the formation of a fourth wave between the 112th and 118th day of disease, which, however, proves abortive, the temperature only rising to 101° F.

After this the temperature remains quite normal, and the patient, although still weak and anæmic, is shortly afterwards sent back to his regiment to be employed for the first month on light duty.

That these secondary waves or relapses may persist for a very long time is shown by the next chart (Fig. 33), on which two are represented as occurring between the 115th and 160th day of the disease.

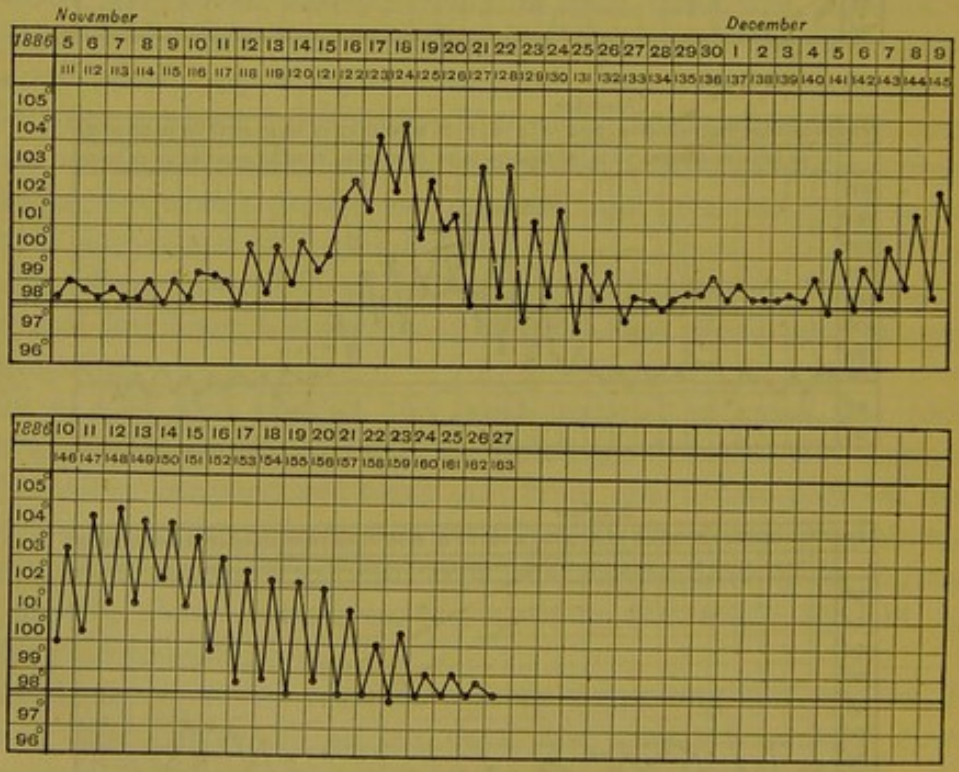

FIG. 33.

Although there is a marked tendency in the temperature curve of Malta fever to exhibit an undulatory course such as has been repre-

sented, nevertheless few cases occur in which the waves are separated by such regular periods of apyrexia.

In most cases the temperature remains a degree or two above the normal between the different recrudescences, and in many the fever is so irregular as to partially or completely mask the wave-like character.

Such an irregular case is presented in the following chart (Fig. 34), in which the temperature only fairly reaches the normal limits by the 100th day.

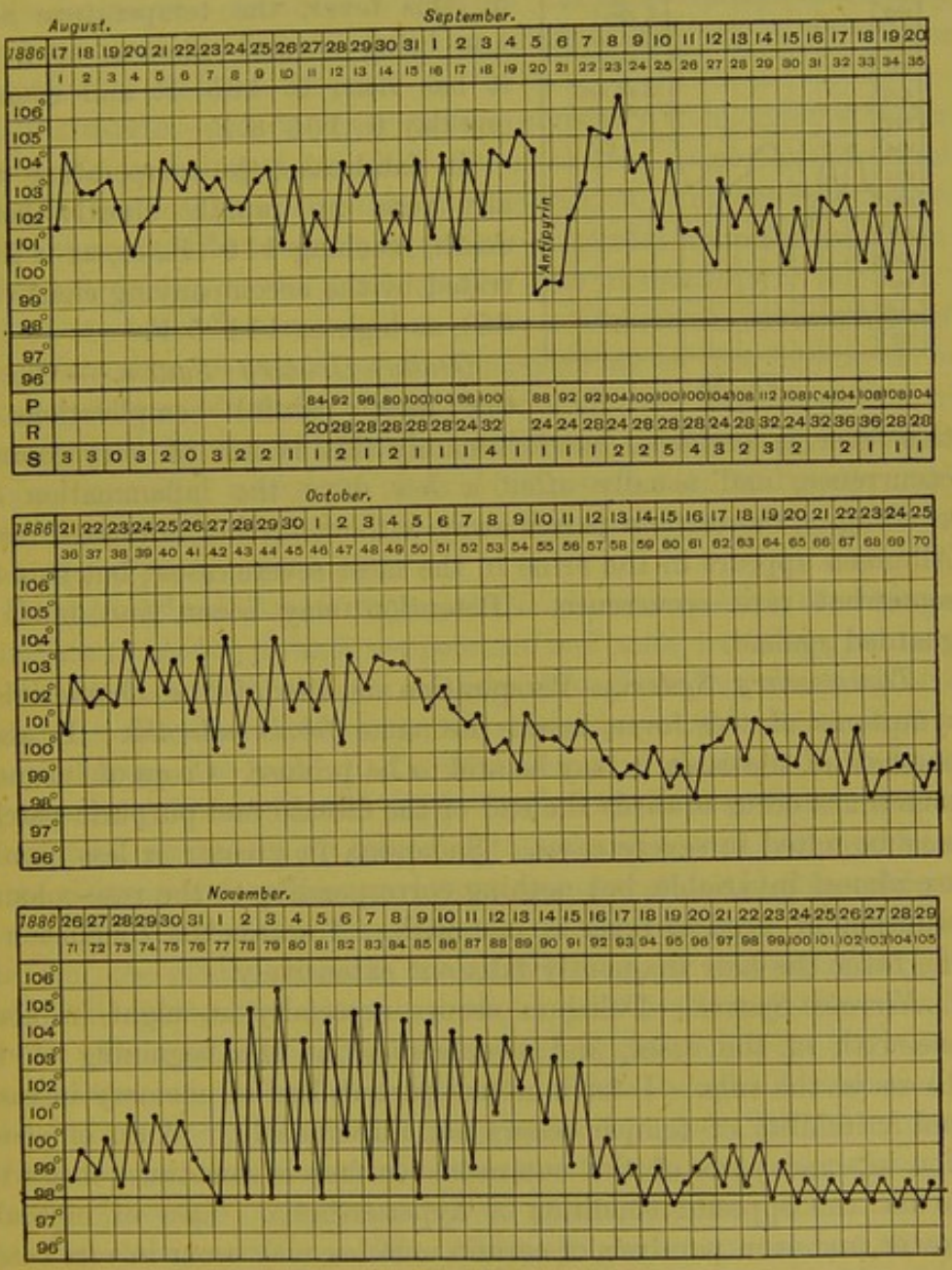

Fig. 34.

On examining this chart it will be seen that the first and second waves are separated by a period, not of apyrexia, but merely of

lowered temperature, and that between the second and third waves the temperature is also above the normal line except at a few points.

The third wave, from the seventy-seventh to the eighty-seventh day, is interesting, as being a well-marked example of the temperature assuming the intermittent type, which, as mentioned above, sometimes occurs in this fever, the temperature at 8 A.M. being normal, whereas at 2 P.M. it has risen to 105° F., and even on one day to 105°·8 F.

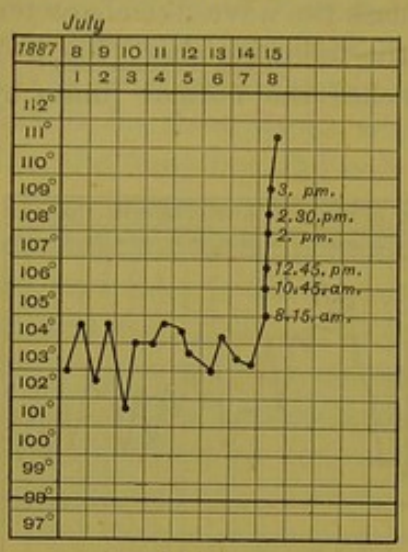

FIG. 35.

In fatal cases the temperature usually runs up rapidly shortly before death, reaching 110° F., or, as is illustrated by the accompanying chart (Fig. 35), as high as 111°·5 F.

Genito-Urinary System. — One of the most painful complications in this fever is orchitis; but fortunately this is not of very common occurrence, and usually after a few days the inflammation and swelling of the testicle passes away without further untoward result. Albumen appears in the urine in the most severe cases; but this is a somewhat rare occurrence. Retention may occur, and must be guarded against.

Tegumentary System.—Perspiration is one of the characteristic symptoms of this fever, hence the name *febris sudoralis.* In about one-half of the cases it may be said to be profuse. Crops of furunculi sometimes occur towards the end of the disease, and purpuric patches may be noted in severe cases. Sudamina in greater or less number are almost invariable, but nothing corresponding to the rose-coloured eruption of typhoid fever is met with. Towards the conclusion of the fever the hair frequently falls out extensively.

Nervous System.—Delirium occurs, especially at night, in about one-sixth of the cases, and sleeplessness at the beginning of the illness is the rule. I have noted partial loss of memory in cases, and irritability of temper occurs not infrequently. Want of sensation in some parts of the cutaneous surface is sometimes met with, as also the opposite condition of hyperæsthesia. Loss of muscular power, and even paralysis of certain muscles, may occur in rare cases; and this condition may persist for several months. Headache is severe in more than a third of the cases,—usually frontal, next in frequency occipital, and least commonly confined to the vertex.

Pain in the lumbar region is a frequent symptom; and sciatica in one or both nerves occurs in about one-twentieth of the cases.

Pain and Swelling of Joints.—This very important symptom is noted in nearly half the cases. The joints most commonly affected are the shoulders and knees, next in frequency the hips, then the elbows and wrists, and lastly the ankles. The amount of effusion, say into a knee-joint, may be very considerable, but in no case have I seen suppuration occur.

Special Senses.—In about a tenth of the cases dulness of hearing is complained of; and in very rare cases a patient is found who complains of dimness of vision.

Diagnosis.—Malta fever differs clinically from typhoid fever, principally in being of longer duration, in the absence of the characteristic rose-coloured eruption, in constipation being the rule instead of diarrhœa, in the frequent presence of painful articular complications, and, finally, in the much smaller rate of mortality.

But it cannot be denied that many severe cases occur which so closely simulate severe cases of typhoid, that for the first week or two it is impossible to be absolutely sure of their nature, except by post-mortem examination. After such cases have been under observation for several weeks, the clinical differences tend to become more manifest, and there is usually then no difficulty in separating the one from the other.

Pathological Anatomy.—As would be expected, the changes found are those due to high temperature and some irritating property in the blood. On making a section through a Peyer's patch, and examining it under a low power, the serous, muscular, submucous, and mucous layers are found to be unthickened, and almost normal in appearance. The epithelial layer is continuous over the surface of the gland. Under a high power, morbid changes are found restricted to the mucous and submucous layers, and consist in a slight proliferation of the cellular elements. On examining the large endothelioid plates of the glandular tissue they are seen to be somewhat swollen and proliferating, and there is a slight proliferation of the adenoid tissue. The mesenteric glands are only slightly enlarged; there is proliferation of the cellular elements of the lymphoid tissue; the reticulum is very delicate, and appears in places to be almost obliterated by the increase in the number of the cells; there is some proliferation of the endothelioid plates, and they are in a condition of cloudy swelling. The spleen weighs on an average 18 oz., and is soft and diffluent; the Malpighian bodies are enlarged from an apparent increase in the number of the round lymphoid cells; the endothelioid plates of the marginal sinuses are proliferating and

swollen; a condition of intense congestion is seen in the section, the sinuses being enormously distended with blood; there is a marked exudation of small round cells along the lines of most of the venules. The liver is congested, the cells in a condition of cloudy swelling, and there is an infiltration of small round cells in the interlobular fissures. The kidneys are also congested, and in a condition of glomerular nephritis.

Treatment.—*Prophylactic.*—Those who find it convenient to spend the hot and unhealthy season away from the Mediterranean, in more temperate climates, should do so. Failing this, a partial migration from the over-crowded cities to the neighbouring country may be recommended. The many, who can do neither, should carefully examine the sanitary state of their dwellings before the hot weather sets in. The house drains should be tested for leakage, seen that they are properly trapped, and kept thoroughly flushed. The water supply should also be looked to. Everything liable to decompose in the house or surroundings should be burnt or otherwise disposed of. Care should be taken to avoid over-crowding of sleeping apartments, and that nothing prevents their being flushed with fresh air and sunshine at suitable hours. Personal health should be attended to, since over-fatigue of mind or body, intemperance, exposure to chills, etc., strongly predispose to the disease.

Therapeutic.—It may be stated at the outset that there is no specific medicinal treatment known for combating this fever, and that the majority of the cases are best treated by leaving out drugs as much as possible.

On account of the name remittent fever being commonly associ- with this disease, most medical men at first treat it by giving large doses of quinine. As the result of many observations on the action of this drug in Malta fever, I can assert that quinine has absolutely no beneficial influence whatever, and if pushed, as is too often the case, is deleterious.

There is also a great temptation, on account of the frequency of joint complications, to use salicylic acid and the salicylates; but experience soon shows that these drugs are also quite useless.

In this disease, as in many others, the therapeutic treatment must still remain in the unsatisfactory position of treating symptoms as they arise. High temperature is one of the most important of these, and one of the most difficult to treat. Antipyrin, given to the extent of 60 or 90 grains in two or three doses, at intervals of an hour, causes a fall of temperature of many degrees in a few hours; but the return rise is just as remarkable for its rapidity, and I have never seen any real benefit accrue from its use.

The application of cold is probably the best method of combating this symptom; but, to be of any use, this therapeutic means must be carried out thoroughly. Cold sponging and the wet pack may be useful in moderate cases; but to save life when hyperpyrexia threatens to supervene, immersion of the body in the cold bath is necessary. This may require to be repeated for some days, every three or four hours, night and day, and is a method of treatment difficult to carry out thoroughly. The patient is placed at first in water at a temperature of 80° or 85° F., and this is then cooled down some 15° or 20° by placing ice in the water. At the same time, a quantity of cold water may be dashed over the patient's head from time to time. Ten minutes is about the usual time required for each immersion, during which time a little brandy should be given. The temperature in the rectum must be taken every hour, and the bath repeated as soon as the body heat tends to rise above 103°.

Sleeplessness is another symptom which may require to be treated at the beginning of the illness. As a rule, this soon passes off; but a full dose of antipyrin, or chloral with bromide of potassium, may sometimes be found useful. Headache also usually disappears for a time shortly after the administration of antipyrin; and the same drug may be tried if the lumbar pain presses for treatment.

The painful affection of the joints is best treated by local applications, such as painting with iodine, or gently rubbing with one of the ordinary liniments; and for the sake of protection the joint should be enveloped in cotton wool. The orchitic complication is relieved by hot fomentations, to which belladonna or opium may be added, and by the careful support of the inflamed testicle. Constipation is best relieved by enemata, or by some simple aperient, such as castor oil.

Dietetic.—As there is a great tendency to gastric irritability in this disease, every care should be taken regarding the diet. During the first days of the fever, milk alone will be found best, then beef or chicken tea may be added. Raw eggs beaten up in milk are also well borne. In many cases the addition of stimulants will be found necessary. I would specially mention at this point, that as this diet of milk, beef-tea, eggs, and brandy may require to be continued for several weeks, it is most essential that lemonade made from fresh lemons or lime juice be added to prevent the occurrence of scorbutic symptoms. After the acute stage of the fever has passed, the monotony of the diet should be varied as much as possible, by giving part of the milk in the form of rice pudding or custard. There can be no hard and fast rule as to when mild solid food, such

as fish or chicken, may be given, each case must be judged for itself; but the transition should be gradual, and not till the temperature has fairly come down to the normal, and remained there for a fortnight, should the patient be promoted to ordinary diet.

Change of Climate.—Finally, the question of the benefit of a change of climate to England may be touched upon. I do not hold, as many do, that it is as necessary to get patients out of the infective area of Malta fever as in the case of malaria. I rather look on Malta fever and typhoid as being similar in this respect, and that patients will throw off the disease and recover perfectly without leaving the island. By saying this, I do not wish in the least to disparage the benefit of change of climate which common experience proves to be so great; but to combat the exaggerated notion which prevails among many, that every effort must be made to hurry their fever cases out of the island.

On account of this mistaken idea, many invalids are sent to England while still suffering from the fever, and in an extremely debilitated condition, to bear the misery of a sea voyage, and to pass the long period of convalescence exposed to the vicissitudes of an English winter, when the climatic conditions of Malta are just becoming favourable.

CHAPTER VII.

YELLOW FEVER.

BY GEORGE M. STERNBERG, M.D., U.S.A.

Definition.—Yellow fever is a specific infectious disease which is characterised by a single febrile paroxysm of short duration (two to five days), followed by a period of great depression of the vital powers (so-called stage of calm), by the presence of albumen in the urine, by an icteric colour of the skin, and by a tendency to passive hæmorrhages from mucous surfaces—especially from the stomach—producing "black vomit."

Etiology.

A Miasmatic Contagious Disease.—Yellow fever is contracted by exposure in infected localities, and not directly by contact with the sick. This fact is established by abundant observations, although some physicians still look upon it as a strictly contagious disease. No doubt, as in cholera and in typhoid fever, a specific infectious agent is given off from the bodies of the sick, which when it finds a favourable nidus may establish a new centre of infection; and, as in the diseases named, it seems probable that this specific infectious agent is contained in the alvine discharges of the sick. That it is not given off from the general surface of the body, we infer from the well-established fact that the disease is not contracted by those intimately associated with the sick as a result of such association. During the epidemic prevalence of yellow fever, many physicians and nurses may fall victims to the disease, as they are necessarily exposed in infected localities. But when persons successively fall sick in the same house, or on board ship, they are not infected one from another by direct personal contact, but contract the disease from a common source—the infected locality.

It is well known to the people of the city of Mexico that a visit to the sea-coast city of Vera Cruz during the epidemic season is

likely to result in an attack of yellow fever. It is also well established that those who fall sick with the disease after their return to the city of Mexico never communicate it to others who are closely associated with them as attendants, etc. The same is true at the health-resort, Petropolis, located in the mountains within a few hours' ride of the city of Rio Janeiro. Frequently individuals fall sick in Petropolis who have visited the infected city of Rio. Never do they communicate the disease to others. This is also the experience of the physicians in charge of hospitals—*e.g.* the Charity Hospital of New Orleans; so long as the hospital and its vicinity remain uninfected, cases do not originate in the hospital, although yellow fever patients may be treated in the wards along with unacclimatised persons suffering from other diseases, and be cared for by susceptible attendants.

In his report upon the camps established near Memphis in the epidemics of 1878 and 1879, Colonel Cameron makes the following statement: "It was found necessary that the officer in authority should set an example of constant indifference to attack in order to appease, as far as possible, the constant anxiety of the population under his charge. Especially was this true in 1878, as depopulation went on slowly that year, and infected people poured daily into the camps from the more pestilential portions of the city. Very many reached camp with the fever on them, so that as many as seventeen persons fell victims in one night, not a few in their tents. In no instance, however, did they communicate the disease to their families or bed-fellows, as far as could be traced."

In the same epidemic (1878), Dr. Minor reports that over thirty cases were discovered among refugees in Cincinnati, O., and says: "No physician or nurse contracted the disease, and in no instance did it exhibit any tendency to spread." The same was true in Nashville the same year; twenty imported cases occurred in different parts of the city without any local cases resulting from them.[1] Evidence of this kind could be extended to fill a volume; but sufficient has been presented to establish the statement made, and the reader may be referred to the "proofs of non-contagion" in the second volume of the classical work of La Roche (pp. 236–566).

Epidemic Development.—The development of an epidemic of yellow fever, in places removed from the foci of the disease, depends upon—(*a*) the introduction of the specific cause by yellow fever patients, or through infected articles — fomites; (*b*) local conditions which favour the multiplication of the specific germ external to the body; (*c*) favourable meteorological conditions; (*d*) the presence of susceptible individuals in the infected locality.

Usually new infected centres are established by the arrival of individuals who fall sick of the disease as a result of exposure in previously infected localities.

[1] *Report of Nashville Board of Health.*

That infected centres may be established independently of the arrival of sick persons is, however, beyond question.

A striking instance of this is afforded by an outbreak which occurred at Madrid in 1878. A circumscribed epidemic was developed in this city about the 1st of September, which resulted in a mortality of thirty-five out of fifty cases taken sick. This outbreak was traced to importation, although all of the cases occurred among the permanent residents of the infected area. Associated with the young people who first fell sick, crowded in the same rooms with them to the number of ten or fifteen in a room, were a number of soldiers recently returned from Cuba with their baggage. These men had themselves suffered from yellow fever in Cuba, or were acclimatised by long residence there.

We have on record instances which appear to be authentic of the development of an epidemic as the result of the opening of a trunk containing infected clothing, sent from a locality where the disease was prevailing to one previously healthy. Epidemics have also been traced to the unloading of earth ballast from the shores of an infected port upon the wharves of a healthy place in the yellow fever zone.

The first cases of local origin in an epidemic do not, as a rule, occur until some time has elapsed after the arrival of the infected ship, or fomites, or sick person responsible for the introduction of the "germ." The interval may vary from a few days to several weeks, according as local conditions are favourable or otherwise for the development of the infectious agent.

In the great epidemic of 1878, which was traced by Dr. Choppin, President of the Louisiana State Board of Health, to importation by the steamship *Emily B. Souder*, which arrived from Havana on the 23rd of May, the first cases of local origin did not occur until after an interval of five or six weeks. But these first cases occurred, according to Dr. Choppin, in the immediate vicinity of the houses in which two of the officers of the *Souder* (Clarke the purser, and Elliott, one of the engineers) died soon after the arrival of that vessel.

Altitude.—The recorded facts do not justify the conclusion that the limitations as to altitude depend solely upon the lower temperature of elevated regions. As pointed out by Hirsch, "the disease stops short at many points in the West Indies where the climate is still in the highest degree tropical. On the other hand, there have been epidemics in cool weather at very considerable altitude, as, for example, at Newcastle, in Jamaica" (elevation about 4000 feet). In the Antilles the disease has rarely appeared at a height of more than 700 feet. In Mexico it has prevailed, however, at Cordova (2500), but is unknown in the cities of Orizaba, Jalapa, and Puebla, which have elevations of more than 3000 feet. In

Spain a single limited outbreak has occurred at Madrid, which is about 2000 feet above the sea-level; but with this exception the altitudinal range has rarely exceeded 1000 feet. In the United States the most elevated locality in which the disease has prevailed as an epidemic is Chattanooga, Tenn., which is 745 feet above the sea level.

Yellow fever is essentially a disease of the sea coast; and while in great epidemics it may become widely diffused in the interior, it follows, for the most part, the course of navigable rivers.

It is a disease of cities and towns of considerable size, and rarely extends to small country villages, or spreads among the scattered rural population.

In seaport towns it frequently makes its first appearance in the vicinity of the wharves, or in localities frequented by sailors.

Sanitary Conditions.—Above all, yellow fever is a disease which is influenced by local unsanitary conditions. In those places where it is endemic, it haunts the low-lying and filthy portions of the town; and in epidemics it exhibits a marked preference for towns which are in an unsanitary condition. It frequently happens when a town is invaded that the disease is limited, for a considerable time at least, to the filthy districts of the place, in which the degraded victims of poverty and vice congregate in ill ventilated apartments, surrounded by the filth which accumulates in such localities when not kept under a rigid sanitary supervision.

Decomposing organic matter of *animal* origin seems to furnish an especially favourable nidus for the germ. This is shown by its favourite haunts, and by the fact that in marshy places, in the vicinity of cities where it prevails, and where *vegetable* decomposition is active, it does not effect a lodgment. The influence of putrefying organic matter of animal origin in the causation of epidemics has several times been made apparent. Parkes, the famous English hygienist, maintained the fæcal origin of the disease; and there seems to be good reason for the belief that the accumulation of this kind of filth in exposed situations is favourable to the development of an epidemic.

Meteorological Conditions.—*Temperature.*—These control in a most decided manner the prevalence of the disease in localities where it is endemic, and its epidemic extension when new infected centres are established among a susceptible population. The influence of temperature is shown by the fact that it is a disease of the tropics and of hot seasons; that it prevails throughout the year in the cities of Rio Janeiro, Havana, and Vera Cruz, although to a much less extent during the cool season; while in

more temperate regions its prevalence is limited to the summer season. It does not prevail as an endemic disease in places which have a mean winter temperature much below 65° F.; and, as a rule, epidemics are not developed at a lower temperature than 75° to 80° F. The approach of cool weather checks the progress of an epidemic, and it is arrested completely when the temperature falls to the freezing point. There are, however, numerous facts which indicate that the infectious agent, although rendered inactive, is not destroyed by a freezing temperature. Epidemics which have been checked by frost have been revived by the recurrence of warm weather; and in certain instances in temperate regions the germ has survived the winter, and a second epidemic has occurred without a new importation (Memphis, 1878–79; Cadiz, 1800–1; Malaga, 1808–9). Epidemics which originate early in the season often terminate before there is frost, simply because the susceptible material is exhausted; but when strangers venture within the infected area they furnish evidence of the continued activity of the morbific poison, by falling victims to the disease.

The influence of season is shown by the following table:—

MORTALITY FROM YELLOW FEVER IN THE CITY OF HAVANA FOR TEN YEARS—1870 TO 1879 INCLUSIVE.[1]

Month.	1870.	1871.	1872.	1873.	1874.	1875.	1876.	1877.	1878.	1879.	Average of Ten Years.
January, .	6	18	20	32	7	16	24	8	26	11	17
February, .	4	23	13	23	4	16	24	9	13	13	14
March, .	4	12	4	27	18	32	29	11	5	6	15
April, .	6	54	4	37	22	34	33	8	28	13	24
May, . .	14	91	13	127	85	32	103	16	53	40	57
June, .	66	201	68	378	172	142	292	143	184	237	188
July, .	112	234	68	416	361	187	675	249	504	475	328
August, .	201	138	70	127	416	144	250	285	374	417	242
September,	91	72	59	35	186	102	97	234	179	148	120
October, .	77	55	38	28	91	109	42	185	106	44	78
November,	49	51	85	5	42	105	31	150	53	31	60
December,	35	42	73	9	21	82	19	76	34	9	40
Total, .	665	991	515	1244	1425	1001	1619	1374	1559	1444	1183

Although the development of an epidemic seems to require a comparatively high temperature (75° to 80° F.), experience shows that it may continue in full force at a much lower temperature (60° to 70° F.) when local conditions are favourable and a susceptible population is exposed in the infected area.

Atmospheric Humidity and Rainfall.—Another factor of import-

[1] Chaillé, *Report to National Board of Health.*

ance in the etiology of yellow fever epidemics is atmospheric moisture and precipitation. Yellow fever is a disease of the sea coast and of the margins of great rivers, and it does not prevail in arid, desert places in the interior, although the elevation may be but a little above the sea level, and the temperature extremely high. There is reason to think that this difference is largely due to the difference as to moisture in the atmosphere and in the soil. Some authors have insisted especially upon the presence of moisture in the atmosphere, almost to the point of saturation, as an essential condition for the development of an epidemic. But, on the other hand, epidemics have sometimes occurred in unusually dry seasons, as at New Orleans in 1841. In Martinique, according to Dutrouleau, yellow fever has sometimes committed its greatest ravages after and during the driest season.

Heavy rains, by purifying the air and cleansing the streets and sewers of an infected city, exercise a favourable effect upon its sanitary condition, and in the tropics the commencement of the rainy season often puts an end to the prevailing epidemic. It is probable that the statement made by some authors, that upon the coast of Guiana, and elsewhere in the tropics, dry weather is favourable to the spread of the disease, is but another way of stating the fact that the heavy rains of tropical regions are unfavourable, the dry weather being only dry by comparison.

Wind.—There is no evidence that the infectious agent of yellow fever can be conveyed by the wind, in an active condition, to any considerable distance; and the wind has but little to do with the dissemination of the disease. In cities, the extension of an epidemic from centres, resulting from the importation of cases, or fomites, is usually quite gradual, and independent of the prevailing currents of air.

In the comparatively small and land-locked harbour of Havana, vessels which anchor some distance from the shore, and which are kept in a good sanitary condition, do not suffer from yellow fever unless unacclimatised members of the crew are permitted to go on shore. Vessels lying at the wharves, on the contrary, are very likely to become infected.

Bacteriology.—The facts heretofore stated lead to the inference that the specific infectious agent in yellow fever is a living micro-organism which, under favourable meteorological conditions, multiplies external to the human body in unsanitary localities, where it finds a suitable pabulum for its growth.

But the correctness of this inference has not as yet been established by an experimental demonstration, and we are still

ignorant of the morphological and biological characters of the "yellow fever germ," notwithstanding the claims which have been made by several investigators to have discovered it. This assertion is based upon the personal investigations of the writer, made in Brazil, in Cuba, in Mexico, and in the United States, in accordance with an Act of Congress (1887) authorising such an investigation. The results of these investigations are given in detail in my *Report on the Etiology and Prevention of Yellow Fever* (Government Printing Office, Washington, 1890).

Briefly stated, these results were as follows:—

Forty-two autopsies were made in typical cases of yellow fever, and seventeen autopsies in other diseases for comparative researches.

Aërobic and anaërobic cultures were made from the blood, the liver, the kidney, the urine, the stomach, and the intestine.

The experimental data recorded show that—

The most approved bacteriological methods fail to demonstrate the constant presence of any micro-organism in the blood and tissues of yellow fever cadavers peculiar to the disease.

The micro-organisms which are sometimes obtained in cultures from the blood and tissues are present in comparatively small numbers; and the one most frequently found (*Bacterium coli commune*) is present in the intestine of healthy individuals, and consequently its occasional presence cannot have any etiological import.

A few scattered bacilli are present in the liver, and probably in other organs, at the moment of death. This is shown by preserving portions of the liver, obtained at a recent autopsy, in an antiseptic wrapping.

At the end of twenty-four to forty-eight hours the interior of a piece of liver so preserved contains a large number of bacilli of various species, the most abundant being those heretofore mentioned as occasionally found in fresh liver tissue, viz. *Bacterium coli commune* and *Bacillus cadaverus*.

Blood, urine, and crushed liver tissue, obtained from a recent autopsy, are not pathogenic in moderate amounts for rabbits or guinea-pigs.

Liver tissue preserved in an antiseptic wrapping at a temperature of 28° to 30° C., for forty-eight hours, is very pathogenic for guinea-pigs when injected subcutaneously.

This pathogenic power appears to be due to the micro-organisms present, and to the toxic products developed as a result of their growth. It is not peculiar to yellow fever, inasmuch as material preserved in the same way at comparative autopsies, in which death resulted from accident or other diseases, has given a similar result.

Having failed to demonstrate the presence of a specific "germ" in the blood and tissues, it seems probable that it is to be found in

the alimentary canal, as is the case in cholera. But the extended researches made, and recorded in my report, show that the contents of the intestines of yellow fever cases contain a great variety of bacilli, and not a nearly pure culture of a single species, as is the case in recent and typical cases of cholera.

Comparatively few liquefying bacilli are found in the fæces discharged during life, or in the intestinal contents collected soon after death from yellow fever cadavers. On the other hand, non-liquefying bacilli are very abundant.

The one most constantly and abundantly present is the *Bacterium coli commune* of Escherich.

This is associated with various other bacilli, some of which are strict anaërobics and some facultative anaërobics.

Most of these bacilli have been excluded by the fact that they are not constantly found, or that they have been obtained from the contents of the intestine of healthy persons, or of those dying from other diseases than yellow fever.

Some of the micro-organisms present in the dejecta of yellow fever patients, as shown by stained smear-preparations, have not developed in the cultures made, either aërobic or anaërobic. One extremely slender filiform bacillus, which can only be seen with high powers, and which is quite abundant in some of my preparations, has never been obtained in the cultures made; and no doubt there are others in the same category.

That the yellow fever germ is strictly anaërobic, or that it will only grow in a special nidus, may be inferred from certain facts relating to the extension of epidemics.

Media of Propagation.—There is no evidence that yellow fever is propagated by contamination of the supply of drinking water, as frequently, and probably usually, occurs in the case of typhoid fever and cholera. Moreover, epidemics extend in a more deliberate manner, and are restricted within a more definite area, than is the case with cholera and typhoid fever. It is usually at least ten days or two weeks after the arrival of an infected vessel, or of a person sick with the disease, before cases of local origin occur; and these cases occur in the immediate vicinity of the imported case or of the infected vessel. When the disease has effected a lodgment, the area of infection extends slowly, and usually has well-defined boundaries. In towns and cities having a common water supply one district remains perfectly healthy, while another, and usually the most filthy one, may be decimated by the scourge.

Micro-Organisms which have been described as the Specific Cause of Yellow Fever.—*Cryptococcus xanthogenicus*

of Dr. Domingos Freire, of Brazil.—In his principal work, published in 1885, Freire gives the following account of his cryptococcus :—

"When we follow with care and attention the march of the development which characterises the germs which produce yellow fever, we acquire a certainty that, commencing to present themselves under the form of little points almost imperceptible, they afterwards gradually increase in diameter until they attain considerable dimensions; so that the little beings, which at the outset had the appearance of little grains of sand, not measuring more than 0·001 millimetre to 0·002 millimetre in diameter, arrive little by little to such a development that they reach the dimensions of 0·005, 0·007, 0·008 millimetre, and sometimes even more in certain conditions. When they have attained the adult age, these cells are broken at divers points, and discharge their contents, composed of spores already formed, mixed with a viscous substance of a yellow colour, composed of a pigment and a protoplasmic substance, and of the liquids elaborated by the cells."

In an address delivered in Paris in 1887, Freire repeats this account of the mode of development of his cryptococcus. He says :—

"Each adult cell is ruptured at one or several points, and allows to escape its contents, composed of germs which are to perpetuate the species, and two pigments—one yellow, destined to infiltrate the tissues, and to produce the icteric colour which has given name to the malady; the other black, insoluble, and destined to be carried along the circulatory current, producing either capillary obstructions or blood stasis in the parenchyma of the organs."

This account is entirely fanciful, and is evidently based upon erroneous observation and misinterpretation of what has been seen under the microscope, and upon imperfect methods of research. No such micro-organism as Freire has described is known to bacteriologists, and certainly nothing of the kind is to be found in the blood and tissues of yellow fever cases, as he asserts.

At the time of my visit to Brazil (1887), Dr. Freire, who had just returned from France, presented to me, as his yellow fever germ, a liquefying staphylococcus. Like other micro-organisms of the same class, this multiplies only by binary division; and having cultivated it in various media, I can say with confidence it does not form a black or a yellow pigment, and consequently does not correspond with the *Cryptococcus xanthogenicus* as previously described by Dr. Freire. Nor is it found in the blood and tissues of yellow fever patients.

Tetragenus febris flava.—The space allotted to this article will not permit me to give an extended account of other micro-organ-

isms which have been supposed to be concerned in the etiology of yellow fever. The *Tetragenus febris flava* of Dr. Finlay, of Havana, is a micrococcus in tetrads which I have frequently obtained from the surface of the body of persons not suffering from yellow fever in the city of Havana, and which I have now had in continuous cultivation for several years. It is not pathogenic; and it is not found, unless by rare exception, in the blood or tissues of yellow fever cadavers. The bacillus found by Dr. Paul Gibier in a certain number of cases of yellow fever studied by him, is only exceptionally present in the alvine discharges of yellow fever patients or in the contents of the intestine obtained as soon as possible after death. There is no satisfactory evidence that it bears any relation to the etiology of the disease.

Susceptibility.—Individuals of every race and of all ages, who are exposed to the yellow fever poison for the first time, during the epidemic prevalence of the disease, are liable to be attacked. But there is a wide difference in the degree of this susceptibility among races, and among individuals of the same race.

It has been asserted that the negro race has a congenital immunity from yellow fever; but this is a mistake. The susceptibility of the negro is, however, much less than that of the white race; and among those attacked the mortality, as a rule, is small.

This is shown by the statistics relating to the white and black troops in the British service at West India stations. "While in Jamaica the annual loss among the former amounts to 102 per 1000 of the mean strength, the deaths among the blacks did not exceed 8 per 1000. In the Bahamas the mortality of the whites was 59 in 1000, that of the blacks 5·6 in 1000" (La Roche).

In the Report of the Board of Experts appointed by Congress to investigate the epidemic of 1878, we find the following remarks:—

> Berwick City, 40 cases among coloured, no deaths. Morgan City, 21 deaths among coloured persons. Brownsville (Tenn.), of 162 coloured cases, 21 died. Chattanooga, of 685 cases, 256 whites, 429 coloured; of 164 deaths, 118 whites, 46 coloured. Decatur (Ala.), of 64 white cases, 28 died; of 168 coloured cases, 21 died.

The indigenous races of the West Indies and of the continents of North and South America have no immunity, except such as is acquired by residence in an endemic focus of the disease; and the same is true of the Mongolian race; but, like the negro, although in a minor degree, its susceptibility is less than that of the white race, and the mortality among those attacked is not so great.

In general it may be stated that the natives of northern latitudes

are more susceptible than those born in tropical or subtropical climates.

Blair, who had an extended experience in Guiana, says:—

> The lower the winter temperature in the native country of those attacked, the more severe was their sickness; so that, while the mortality among West Indians amounted to only 6·9 per cent. of the sick, it rose to 17·1 among the Italians and French, 19·3 among the English, 20·2 among the Germans and Dutch, and 27·7 among the Scandinavians and Russians.

Immunity is acquired by suffering an attack of the disease, or by long residence in localities where it is endemic, or prevails frequently as an epidemic; this acquired immunity is not, however, absolute. Second attacks no doubt occasionally occur, although this has been denied by some authors. Blair, whose experience was very great, says that he does not believe there is an instance of a second attack after a month's perfect restoration to health. Other authors are equally positive in their statements. On the other hand, we have numerous authentic accounts of second attacks.

Acclimatisation.—It is a remarkable fact that the population of a large city like Havana or Rio Janeiro, in which yellow fever has been endemic for a series of years, enjoys such a degree of immunity from the effects of the deadly poison that there is no interruption of business or pleasure at a time when strangers in the city are falling sick on every side. The development of an epidemic in these cities depends upon the presence of susceptible strangers in sufficient number to furnish a series of cases considered large enough to justify the use of the word. The presence of but few strangers during the epidemic season leads to the announcement that the disease is not epidemic, but that sporadic cases occur from time to time. Under exceptional circumstances, however, epidemics are developed in these endemic foci of the disease in which those who, by birth and long residence, were supposed to be acclimatised furnish a certain quota to the general mortality. This has frequently occurred, for example, in the city of New Orleans, where yellow fever formerly prevailed almost annually, and where the Creole population was supposed to enjoy an hereditary immunity.

History and Geographical Distribution.

Restricted Range.—The geographical range of yellow fever is more restricted than that of any other acute infectious disease; and within the area of its prevalence it is, as we have seen, essentially a disease of the littoral, and especially of seaport cities.

While occasional epidemics have occurred upon the south-west coast of the Iberian peninsula, the disease, as an epidemic, is unknown elsewhere in Europe; and there is no evidence that it has ever invaded the great and populous continent of Asia. In Africa it is limited to the West Coast, and only to certain portions of that coast. In North America, although it has occasionally prevailed as an epidemic in every one of our seaport cities as far north as Boston, and in the Mississippi valley as far north as St. Louis, it has never established itself as an endemic disease within the limits of the United States. Vera Cruz, and probably other points on the Gulf Coast of Mexico, are, however, at the present time endemic foci of the disease. In South America it has prevailed as an epidemic at all of the seaports of the Gulf and Atlantic coasts, as far south as Monte Video and Buenos Ayres, and on the Pacific along the coast of Peru.

The region in which the disease has had the greatest and most frequent prevalence is bounded by the shores of the Gulf of Mexico, and includes the West India Islands. Within the past few years yellow fever has been carried to the west coast of North America, and has prevailed as an epidemic as far north as the Mexican port of Guaymas, on the Gulf of California.

Mode and Point of Origin.—The idea that yellow fever may originate *de novo* within the area of its occasional prevalence, was entertained by many medical authors during the first half of the present century, and still is held by a few. Thus Cornillac (1886) says: "In the zone which is habitual to it, yellow fever may develop at a given moment without apparent cause. It is born spontaneously at a point of this zone, or at several at a time; and neither the temperature, moisture, barometric pressure, electricity, or, finally, effluvia given off from the soil, can explain this sudden invasion." It is true that, in localities where the disease is endemic, cases occur which are not directly traceable to importation; but it is also true that in the principal endemic foci of the disease, such as Vera Cruz, Havana, and Rio Janeiro, yellow fever was at one time unknown; and we have reliable historical data fixing the date of its importation. In short, a careful consideration of the historical evidence relating to the disease gives no support to the idea of independent local origin, any more than in the case of smallpox, cholera, or other specific infectious diseases.

But the early history of the disease is involved in obscurity, and we are at present unable to determine whether, as maintained by some, it was endemic at certain points on the shores of the Gulf of Mexico at the time of the discovery of the "New World," or whether

it was imported to the West Indies from the African coast, as maintained by others.

Recent Extension to Brazil.—The highest medical authorities in Brazil agree that yellow fever was not endemic in the principal seaports of that republic prior to the year 1849, when it was introduced to the city of Bahia by the North American brig *Brazil*, which sailed from New Orleans, where yellow fever was prevailing, and touched at Havana. Two of the crew of this brig died of yellow fever during her voyage from the latter port to Bahia. Soon after her arrival the disease made its appearance among those who had communicated with the ship, and afterwards on other vessels in the harbour. The first case occurred a few days after the arrival of this brig (3rd November). A part of her cargo is said to have consisted of little barrels of beef which had become putrid. From Bahia the disease was carried to Rio Janeiro, where, during the epidemic season of 1850, it caused a mortality of 4160.

According to Professor Barata of the Faculty of Medicine of Rio Janeiro, yellow fever continued to prevail in Brazil until the year 1861, when it disappeared for eight years, to reappear in 1869–70, as the result of a fresh importation. The Italian ship *Creolla del Plata*, which had touched at St. Iago, where yellow fever was prevailing, is named as the vessel which introduced the disease on this occasion.

Epidemics in Rio de Janeiro.—The mortality from the disease under consideration in the city of Rio, from the time of its introduction in 1850 to a recent date, is shown by the following table :—

Year.	Mortality.	Year.	Mortality.	Year.	Mortality.	Year.	Mortality.
1850,	4160	1860,	1249	1869,	274	1878,	1174
1851,	475	1861,	247	1870,	1117	1879,	974
1852,	1943	1862,	12	1871,	8	1880,	1433
1853,	853	1863,	...	1872,	102	1881,	219
1854,	21	1864,	...	1873,	3659	1882,	95
1855,	...	1865,	...	1874,	829	1883,	1336
1856,	...	1866,	...	1875,	1292	1884,	618
1857,	1425	1867,	...	1876,	3317	1885,	278
1858,	800	1868,	...	1877,	282	1886,	1397
1859,	500						

Epidemics in the United States.—The mortality in the seaport cities of the United States which have suffered most severely from this exotic pestilential disease, as a result of favour-

able climatic conditions during the summer months, and of their proximity to permanently infected seaports in the West Indies, from the year 1800 to 1880, is shown by the following table:—

MORTALITY FROM YELLOW FEVER IN CHARLESTON (S. C.), PENSACOLA (FLA.), MOBILE (ALA.), NEW ORLEANS (LA.), AND GALVESTON (TEX.), DURING THE PRESENT CENTURY.

Year.	Charleston.	Pensacola.	Mobile.	New Orleans.	Galveston.	Year.	Charleston.	Pensacola.	Mobile.	New Orleans.	Galveston.
1800,	184	...	...	*	...	1841,	*	*	*	594	...
1801,	...	...	...	*	...	1842,	...	*	60	211	...
1802,	96	...	...	*	...	1843,	1	*	240	487	...
1803,	*	...	...	...	...	1844,	...	*	*	148	400
1804,	148	...	...	*	...	1845,	...	*	...	2	...
1805,	*	...	...	...	...	1846,	...	*	...	160	...
1806,	...	...	...	...	...	1847,	...	*	76	2259	200
1807,	162	...	...	...	...	1848,	...	*	75	850	...
1808,	...	...	...	...	...	1849,	125	...	50	737	...
1809,	...	...	...	*	...	1850,	...	...	...	102	...
1810,	...	...	...	...	...	1851,	...	...	*	16	...
1811,	...	*	...	*	...	1852,	310	...	...	415	...
1812,	*	...	...	*	...	1853,	...	*	115	7970	536
1813,	...	...	...	...	...	1854,	627	*	*	2423	404
1814,	...	...	...	...	...	1855,	...	...	...	2670	...
1815,	...	...	...	...	...	1856,	211	...	...	74	...
1816,	...	...	...	...	...	1857,	13	...	...	199	...
1817,	272	...	...	800	...	1858,	717	*	*	3889	344
1818,	...	...	...	115	...	1859,	...	...	...	...	182
1819,	177	...	274	2190	...	1860,	...	...	...	...	...
1820,	...	...	...	*	...	1861,	...	...	...	...	...
1821,	...	...	*	...	...	1862,	*	*	...	...	...
1822,	2	257	*	239	...	1863,	...	*	...	...	...
1823,	...	...	...	1	...	1864,	*	...	...	...	259
1824,	235	...	*	108	...	1865,	...	...	...	...	...
1825,	2	*	*	49	...	1866,	...	...	...	...	*
1826,	...	...	...	5	...	1867,	...	34	*	3093	1150
1827,	64	*	*	109	...	1868,	...	...	...	...	...
1828,	26	...	*	130	...	1869,	...	...	...	...	...
1829,	...	...	130	215	...	1870,	...	...	*	587	...
1830,	30	...	...	117	...	1871,	213	...	...	55	...
1831,	...	...	...	2	...	1872,	...	...	...	40	...
1832,	...	...	...	18	...	1873,	...	61	27	225	...
1833,	...	...	...	210	...	1874,	...	118	...	...	...
1834,	49	*	...	95	...	1875,	...	...	...	...	...
1835,	25	...	...	284	...	1876,	...	...	...	...	...
1836,	...	...	...	5	...	1877,	...	...	...	...	...
1837,	...	...	350	442	...	1878,	...	...	90	600	...
1838,	351	...	...	17	...	1879,	...	...	...	...	...
1839,	134	*	650	452	250	1880,	...	...	...	...	...
1840,	22	...	...	3	...						

* Number of deaths not stated.

Since the year 1880 there has been but one epidemic within the limits of the United States. This occurred in the States of Florida and Alabama in the years 1887 and 1888, as a result of importa-

tion through the town of Tampa, a seaport on the Gulf Coast of Florida, which is in constant communication with Havana.

The exemption enjoyed for this considerable period (twelve years) by the cities of New Orleans, Mobile, Charleston, and other seaports, in which, as shown by our table, the disease formerly prevailed almost annually, is no doubt largely due to improved methods of quarantine administration.

Epidemics in Europe.—In Europe the ravages of yellow fever have been restricted mainly to the Iberian peninsula. This is due, no doubt, to the frequent intercourse between Spain and Portugal and the West Indian ports, in which the disease is most prevalent, and to the fact that the summer temperature of these countries is favourable for the epidemic extension of the disease; whereas the northern portions of Europe are practically outside of the yellow fever zone.

The first epidemic in Spain occurred in the year 1700 at Cadiz. This city also suffered in 1730–31, 1733–34, 1764, 1780, 1800, 1804, 1810, 1819–21. The epidemics of 1800, 1810, and 1819 were not limited to the city of Cadiz, but the disease extended to the interior, and caused a considerable loss of life in the provinces of Granada and Andalusia, and also in some of the towns of Murcia and Catalonia—especially in Barcelona, from which city the disease was conveyed to the island of Majorca during the last epidemic. No widespread epidemic has occurred in Spain since 1821; but local outbreaks, as a result of importation from the West Indies, have occurred at Gibraltar (1828), Barcelona (1870), and Madrid (1878).

The first epidemic at Lisbon was in 1723; a second was inaugurated in 1856, and during the following year developed into a devastating scourge, which extended to the towns of Belem, Olivaes, and Almada.

In Italy yellow fever has only once effected a temporary lodgment—at Leghorn in 1804, where it was imported from Spain.

Ships with yellow fever on board have occasionally arrived at English and French ports; but local conditions have apparently not been favourable to an extension of the disease, except to a limited extent at Brest in 1856, at St. Nazaire in 1861, and at Swansea (Wales) in 1864.

Pathological Anatomy and Histology.

External Appearances of Body.—The exterior of the body of an individual who has recently succumbed to yellow fever presents an appearance which is quite characteristic. The

icteric colour of the skin, although often not noticeable during the last hours of life, is developed in a large majority of the cases very soon after death. The colour is that which is seen to follow a bruise in which there has been an effusion of blood, and the origin of the pigment is no doubt the same. The icterus from bile pigments, which occurs not infrequently during convalescence, and which may be seen in cases fatal from a relapse, or at a late period of the disease, gives a uniform saffron-yellow colour to the surface of the body and conjunctivæ. In the icteric discoloration of which we speak at present, the colour is not so intense and not so uniformly distributed. The depending portions of the body, and especially those subjected to pressure, have a deeper coloration, and are more or less livid and mottled from hypostatic congestion. Dutrouleau has shown that this ecchymotic appearance of the back is due to position and pressure by placing the body upon the abdomen or side. In this case it is still the most dependent part which shows the livid marbled appearance referred to. The face and hands frequently appear cyanosed, or the face may be livid and turgescent, like that of one recently drowned. A little stream of black vomit is frequently seen trickling down from the corners of the mouth, or a similar fluid may escape from the nostrils, and the lips and gums may be soiled with dark blood which has oozed from them.

Cadaveric rigidity is quickly established and well marked.

Internal Lesions and Pathological Changes.—*Brain.*—The appearances observed upon removing the calvarium are usually those of hyperæmia of the brain and its meninges. The pia mater is almost always congested, and the vessels of the brain are abnormally full of blood; the pons and medulla are especially in a condition of hyperæmia. There is more or less effusion into the sub-arachnoid space and in the ventricles; this is sometimes turbid, and has a yellow colour. The surface of the brain presents sometimes little hæmorrhagic points; and its substance, like the tissues of the body, generally has a more or less pronounced yellow tinge.

Blood Vessels and Capillaries.—Several of the most competent observers agree that there is a fatty degeneration of the walls of the small blood vessels and capillaries of the various organs, and account in this way for the hæmorrhagic tendency of the disease. This is certainly a more rational explanation than the one so frequently offered, viz. that the hæmorrhages are due to the disorganised and diffluent condition of the blood. A moment's reflection should show that this explanation is insufficient, and that the blood, however diffluent, cannot escape so long as the vessels are intact. Ecchymotic points, or hæmorrhagic infarctions, are

sometimes found upon the surface or in the substance of the heart, as well as in other muscles of the body and the organs generally. The cavities of the right side of the heart usually contain soft coagula or dark coloured fluid blood, and the right ventricle sometimes contains a more or less decolourised fibrinous clot.

Blood.—The blood in yellow fever has been described by many observers as "completely disorganised" as to its histological elements. This is a mistake, as is shown by the numerous photo-micrographs made by the writer while in Havana, in 1879, from the blood of cases near a fatal termination. Both the red and the white corpuscles retain their normal appearance, and I have frequently seen the leucocytes undergoing their characteristic movements, even after twenty-four hours, in blood which had been preserved in culture-cells. In the days of bleeding, numerous observers mentioned the fact that the blood either does not coagulate readily, or forms a soft, loose coagulum from which the serum does not separate. The same is true of the blood in the heart and large vessels after death; it is fluid and dark coloured. Although there is no general destruction of the red corpuscles, it is probable that a considerable number of these elements perish in severe cases, for the serum contains free hæmoglobin, which gives it a yellow colour even as early as the third or fourth day. This yellow colour is seen in the serum obtained from the application of blisters to the surface, and in blood drawn for microscopical examination. This passing of the hæmoglobin into the serum, which is no doubt the cause of the yellow discoloration of the tissues in yellow fever, has been ascribed to the presence of bile. This view is not sustained by the chemical researches of Cunisset, a recent French author, who has arrived at the following conclusions:—

"Yellow fever is not a poisoning by the bile; at the outset of the malady the biliary pigments are rarely found in the blood or in the urine. They appear generally only during the second period, and in a great number of cases they are not to be found at all.

"The biliary salts, of which the powerful action of 'deglobulation' admitted by certain authors might explain the disorders which the malady presents, do not exist in the matters vomited, or in the urine, or in the blood, except, in certain cases, in very feeble quantity. In view of the profound alterations of the liver, this absence of the biliary salts is easily understood, and the defective depuration of the blood is to be looked upon as a complication rather than a determining cause of the malady."

Stomach and Intestinal Canal.—The most important pathological changes in yellow fever are found in the organs contained in the

cavity of the abdomen. The mucous membrane of the stomach is always found to be more or less hyperæmic; the congestion is commonly not general, but is confined to smaller or larger spots or districts, in which it is observed to proceed from one or more centres. From these centres it extends or radiates in a lesser degree, either gradually to be lost, or to pass over to another congested district. It is owing to this peculiarity of the congestion that it presents no uniformity of character, but is observed to spread irregularly over larger or smaller portions of the membrane (Schmidt). In addition to these congested patches, there are often to be seen small red patches resembling ecchymoses, but which, according to Schmidt, consist of "an unbroken network of minute vessels congested with blood, and identical with that network of large capillaries which surrounds the aperture of the gastric glands." Crevaux believes that a fatty degeneration of the cells which line the gastric glands and the capillaries of the mucous membrane is the most important lesion here. Other authors speak of an acute gastric catarrh as the process indicated by the appearance and histological examination of the mucous membrane; others again deny that there is any inflammation. My own examination of thin sections stained in various ways, shows that in a certain proportion of the cases there is evidence of inflammation, as shown by the presence of an unusual number of leucocytes in the submucous coat. The dark fluid so often ejected during the last hours of life, known as black vomit, is almost always present in the stomach, in greater or less amount, after death. There is no question that the dark colour is due to the presence of blood pigment, more or less changed by the acid secretions of the stomach.

The present writer has repeatedly verified the fact that black vomit contains numerous decolourised blood corpuscles, in little agglomerations, surrounded by granules of pigment, which under the microscope is of a deep orange colour, rather than black.

The small intestine commonly contains more or less black matter, either fluid and resembling that found in the stomach, or mixed with mucous and smeared over the mucous coating, especially of the ileum. This, no doubt, comes partly from the stomach, but in other cases is due to passive hæmorrhage from the mucous membrane of the intestine itself. This membrane presents arborescent patches of congestion, or portions of the canal may be uniformly red from hyperæmia of the mucous coat; the colour varies from a pale red to a reddish-brown, and is usually more marked in the lower portion of the ileum than elsewhere. The large intestine occasionally

presents similar arborescent patches of congestion, but usually it has a normal appearance.

Liver.—The appearance of the liver in yellow fever is characteristic, at least so far as acute febrile diseases are concerned. Usually it contains less blood than in the normal condition, and is of a pale yellow or brownish-yellow colour, similar to that of new leather in its various shades; occasionally it is gorged with blood, and livid, deep blue, or dark purple in colour. In the victims of chronic alcoholism, it often presents the nutmeg appearance of cirrhosis. The dimensions do not differ materially from the normal, but the consistence is modified by the fatty change, which gives the characteristic colour, and the parenchyma is easily torn and more or less friable. On section it is found to be drier than in the normal state, except in the comparatively few cases in which it is hyperæmic; these are, as a rule, cases which have proved fatal at a very early period. A fact which indicates that there is a stage of congestion antedating that of anæmia and fatty degeneration. According to Crevaux, this congestion is located especially in the portal radicles surrounding the lobules, and is attended with œdema of the interlobular connective tissue. Whether this primary congestion is a constant phenomenon or not, it is certain that in a majority of the autopsies the liver is found to be anæmic, and to present more or less evidence of fatty change in the hepatic cells. This is not, however, a uniform process; but areas of greater or less extent are seen, in which the cells are infiltrated with fat globules, while in other places the cells appear normal. The fatty cells contain one or several fat globules of varying dimensions, and the protoplasm is reduced in quantity according to the extent of this fatty change. The nuclei very often remain intact in the cells infiltrated with fat; but, according to Schmidt, "a great number of the nuclei also undergo fatty degeneration." Often a collar of normal cells remains about the central vein, while those cells nearer the periphery of the lobule contain numerous fat globules.

Kidneys.—The kidneys are also the seat of important pathological changes in the disease under consideration. This consists essentially of a parenchymatous nephritis. Externally no material change is noted in the organs. They do not vary greatly from the normal size, and usually are normal in appearance. When the attack has been brief, however, they may be hyperæmic and of a deep red colour. Ecchymosed spots and hæmorrhagic foci are frequently seen beneath the capsule or in the cortical substance. In the latter situation little globular hæmorrhagic points, the size of a pin's head, have been observed, which, upon examination, proved to be the distended

capsules of the glomeruli. The change in the renal epithelium consists in a cloudy swelling, followed by fatty degeneration and desquamation. Every grade of change may be seen in the section, from a slight degree of cloudy swelling to complete disorganisation and desquamation of the cells. Whole bundles of tubes may often be seen which have been stripped of their epithelium and are entirely empty. In thin sections the lumen of the tubules is seen in places to be filled with infarctions of various appearance. Some are homogeneous and translucent, and it may be more or less coloured with blood pigment; these are composed of an albuminous material. Other infarctions are formed of the granular débris of the renal epithelium; or we may have a mixture of the granular and albuminous material, in which case the latter forms a matrix in which the granules are embedded. These infarctions correspond with the casts found in the urine; those of a granular character are most abundant, and in them the granules often have a fatty appearance. There are also accumulations which differ from these in the fact that they are deeply stained by the aniline colours. They seem to be made up of the nuclei of the cells, sometimes intact, although swollen and compressed; more commonly massed together, or broken into irregular fragments. At least this seems to me to be the most probable interpretation of those infarctions which are stained by nuclear staining agents.

Prophylaxis and Incubation.

General Prophylactic Measures.—What has been said as to the etiology of yellow fever indicates clearly enough the measures of prophylaxis to be taken in localities subject to invasion. These are—(*a*) exclusion of the exotic germ of the disease by the sanitary supervision, at the port of departure, of ships sailing from infected ports; and their thorough disinfection at the ports of arrival, when there is evidence justifying a reasonable suspicion that they are infected; (*b*) isolation of the sick on shipboard, at quarantine stations, and, so far as practicable, in recently infected places; (*c*) disinfection of excreta, and of the clothing and bedding used by the sick, and of localities into which cases have been introduced, or which have become infected in any way; (*d*) depopulation of infected places, *i.e.* the removal of all susceptible persons whose presence is not absolutely necessary for the care of the sick.

Of all measures of prophylaxis, those which relate to the sanitary improvement of cities and towns liable to become infected are perhaps the most important. Municipal hygiene has made great

strides since the early part of the present century, and it is probably to this fact, more than to any other, that certain northern cities, which formerly suffered severely from yellow fever epidemics, owe their long immunity from such visitations, *e.g.* New York and Philadelphia.

Individual Prophylaxis.—Individual prophylaxis requires the individual, first of all, to avoid infected localities. If it is absolutely necessary for a susceptible person to visit a place where yellow fever is prevailing, or to remain in one in which it has effected a lodgment, he should observe the following precautions: Keep away from low-lying and filthy portions of the city; avoid the vicinity of the wharves, and all localities known to be centres of infection, especially at night; sleep as far from the ground as possible; avoid excesses of all kinds, and especially in the use of alcoholic drinks; keep out of the sun during the hottest part of the day, and be careful not to become overheated by violent exercise; avoid constipation.

With reference to the method of prophylaxis by inoculation, practised in Brazil by Dr. Domingos Freire, and in Mexico by Dr. Carmona y Valle, the writer, after a careful examination, has reported officially that—

"There is no satisfactory evidence that the method of inoculation practised by Dr. Domingos Freire has any prophylactic value.

"The claims of Dr. Carmona y Valle, of Mexico, to have discovered the specific cause of yellow fever have, likewise, no scientific basis; and he has failed to demonstrate the protective value of his proposed method of prophylaxis."

Incubation.—The period of incubation in yellow fever does not usually exceed four or five days, and may be less than twenty-four hours. Instances of a much longer period of incubation have been given by various authors—even as long as six weeks or two months; but we are satisfied that these are due either to error in diagnosis, or to the fact that the cases resulted from the establishment of a new and unrecognised centre of infection. Those who believe that the disease is only communicated by personal contagion, naturally date the exposure to the latest date when such personal contact was possible. Thus, when a first case occurs on a ship at sea two or three weeks after leaving an infected port, the period of incubation is supposed to be at least this long. On the contrary, the attack is due, in all probability, to the fact that the ship is infected, and the first case will probably be quickly followed by others which have no direct connection with it, but result, as it did, from exposure on the infected vessel. Instances of an attack

occurring within twenty-four hours after arrival in a city where the disease was prevailing as an epidemic, are numerous and well authenticated.

It is proper to state that several authors, who have had great experience, believe that the period of incubation may be extended to fourteen, or even more days—Blair, Rush, Féraud.

Clinical History.

Onset and General Symptoms.—As a rule, an attack of yellow fever is not preceded by any well-marked premonitory symptoms. The attack may occur at night in one who went to bed in his usual state of health, or in the early morning after an uninterrupted sleep, or during the day while engaged in ordinary occupations. In other cases there is a feeling of lassitude and discomfort for two or three days prior to the attack, with loss of appetite, slight pain in the back and loins, a feeling of giddiness or slight headache, flatulent eructations, constipation, a tendency to perspire at night, or upon very slight exertion, and more or less muscular debility, together with a disinclination for any mental exertion.

The attack is commonly inaugurated with a more or less decided chill, which, by its violence and duration, affords some indication of the probable severity of the case. In certain grave forms of the disease the onset is insidious, and is not marked by any perceptible chill. In a considerable proportion of the mild cases also, especially in the tropics, there is no rigor, and the patient experiences at most only a slight sensation of coldness, which quickly gives place to that of heat. If a thermometer is placed in the axilla during the initial chill, it will be found that the temperature is already considerably above the normal, and very frequently it reaches the highest point attained during the entire attack within a few hours from its inception.

Accompanying the chill are other nervous phenomena, similar in kind to those attending the onset of other specific febrile diseases. There is cephalalgia, often very severe, and located by preference in the forehead and supraorbital region; the eyeballs also are painful, and there is intolerance of light in some instances. Pain in the loins is a very constant and early symptom, which occasions much distress, and sometimes extorts groans and cries from the patient. At the same time pain is usually experienced in the lower extremities, often of a very severe character, constituting the *coup de barre* of the French authors: it affects especially the calves of the legs, the

knees, and the ankles. These symptoms continue, and are even aggravated, after the rigor has passed and the febrile stage is fully developed; in the meantime the face becomes flushed, and sometimes deep red and swollen in appearance; the eyes are shining and suffused, the conjunctivæ more or less hyperæmic, and often deeply injected; in severe cases presenting a fiery, inflamed appearance, which is accompanied by photophobia; the skin becomes hot and dry, and there are apt to be, especially in patients of a nervous temperament, great restlessness and jactitation.

Course, Varieties, Special Symptoms.—*Temperature.*—Yellow fever is a disease of a single febrile paroxysm, lasting from forty-eight hours to seven or eight days—more commonly from three to five days. The acme of temperature is reached at the outset, and from this time the temperature line is a descending one, interrupted sometimes by a slight evening exacerbation, up to the termination of the first period of the disease—febrile stage. The second stage is characterised by great prostration of the vital powers, and lasts from a few hours to two or three days—stage of calm; the temperature during this stage sometimes remains a degree or more above the normal, but more commonly it is normal, or even subnormal for a time. This is followed in severe cases by a reactionary fever of irregular duration, which presents a more or less remittent character. These features are shown in the accompanying temperature charts (Fig. 36).

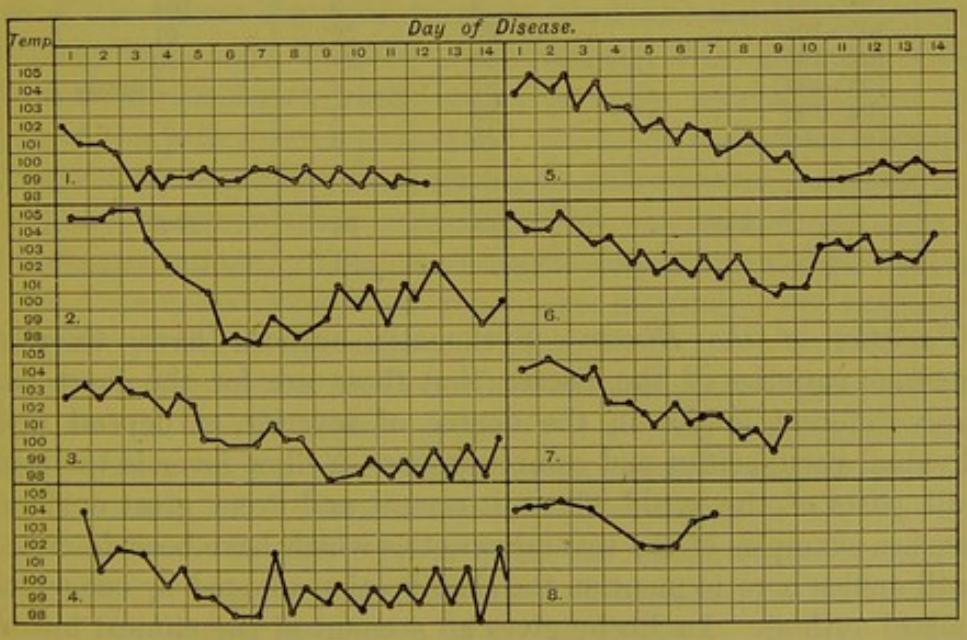

FIG. 36.

Naturally the typical temperature curve is disturbed by complications—visceral congestions, abscesses, parotitis, etc. It is also

disturbed by indiscretions in diet, over-active medication, and by moral causes, especially fright and grief. In mild cases the acme of temperature is reached during the first two or three hours of the attack. In more protracted and severe cases it is not reached until the second or third day, rarely later. In an analysis of 192 cases recorded by Faget, Jones, and myself, the acme was reached on the first day in 102, on the second in 54, on the third in 33, and on the fourth in 3. The highest temperature recorded by Faget was 107°·2 F. Thornton, in a total of 143 cases occurring at Memphis, noted a temperature of 108° F. in a single instance. With this exception, 105°·5 F. is the highest temperature recorded by him. In my own observations 106° F. has been the highest temperature noted. The temperature often rises rapidly just before death, and a very high post-mortem temperature (108° to 110° F.) is a common phenomenon. The temperature reached during the initial febrile paroxysm is a very good index of the severity of the attack, and furnishes a valuable basis for prognosis. This is shown by the following table, compiled by the writer some years since from a series of cases in which a complete and careful record had been made:—

Cases in which the Temperature was—	No. of Cases.	No. of Deaths.	Percentage of Deaths to Cases.
107° and above . .	13	13	100
106°–107° . . .	9	9	100
105°–106° . . .	36	22	61
104°–105° . . .	80	24	30
103°–104° . . .	87	6	7 nearly
102°–103° . . .	29	0	...
101°–102° . . .	15	0	...
Total, . .	269	74	27·5

Varieties of the Disease.—Systematic authors have described numerous varieties of the disease; but these are for the most part simply different grades in the degree of severity, or depend upon individual peculiarities and complications. Yellow fever is the same in all parts of the world where it occurs, and every great epidemic furnishes examples of the several varieties which have been described. It will suffice here to mention the classification adopted by one or two standard authors. La Roche gives an account of the clinical history of the disease under the following headings: *Inflammatory species*, including three grades, intense, mild, and ephemeral; *congestive species*, including four grades, aggravated, adynamic, walking, and apoplectic.

Béranger-Féraud, in his elaborate account of the disease as it prevails at Martinique, classifies the cases as follows:—

1*st Degree.*—Mild yellow fever.

2*nd Degree.*—Yellow fever of moderate intensity: (*a*) cases in which the onset is frank; (*b*) cases in which it is insidious.

3*rd Degree.*—Grave yellow fever: (*a*) ordinary forms, including the gastric, adynamic, ataxic, congestive, and typhoid forms; (*b*) rare forms, including the hyperæsthetic, gangrenous, algid or choleraic, and hydrophobic.

4*th Degree.*—Yellow fever *sidérante.*

Different epidemics are sometimes characterised by the predominance of one or the other of the forms described, and the character of the disease often varies greatly during the same epidemic. The earlier cases are sometimes mild and the mortality small, while later the greater intensity or malignancy of the poison is shown by the occurrence of a large number of cases of the severest grade, and even of those rapidly fatal attacks denominated by the French *sidérante.*

Pulse.—The pulse in sthenic cases is full, strong, and hard at the outset of the attack, and may reach 120 or more pulsations in the minute, more commonly not more than 100 to 110. It diminishes in rapidity and force as the disease progresses; and this occurs even when the febrile heat is not reduced for two or three days, and is considered by Faget a valuable diagnostic sign. During the second stage of the disease the pulse, however hard and accelerated it may previously have been, becomes preternaturally slow and soft. The feebleness of the heart, which seems to suffer especially from the action of the yellow fever poison, and which has undergone a certain degree of fatty degeneration, is shown by this very compressible and slow pulse, which is often reduced to 40, and sometimes even to 30 beats per minute. This constitutes a very characteristic feature of the disease, and affords an important indication for treatment during the period of depression or "calm."

Tongue.—The tongue is sometimes but slightly coated at the outset, and is usually moist; generally it quickly becomes covered with a white coating, which may be in streaks; the margins, as a rule, remain red. Very commonly the tongue is narrow and pointed, differing in this respect from the broad, flabby tongue of the malarial fevers. In the progress of the disease it often becomes dry, and the coating assumes a brownish colour, or it may become very foul, and loaded with sordes.

Face and Conjunctivæ.—The face is at first flushed or bright red and swollen, or it may be of a dusky violet hue; this, with the

deep red suffusion of the eyes in severe cases, is quite characteristic. The countenance often has an expression of anxiety or pain, or of dejection; again it may appear dull and indifferent.

In the last period of the disease, in fatal cases, the features become shrunken,—sometimes bloated and flabby,—the brows are often contracted, and the eyes sunken, with ecchymosed lids.

The hyperæmia of the conjunctivæ in mild cases may be temporary, in the more severe ones it is apt to last through the first period, and in quickly fatal cases the eyes may be deeply injected throughout. Usually by the third day a careful inspection will show that the conjunctivæ have a yellowish tinge, which becomes more intense as the disease progresses.

Skin.—The skin is hot and dry in some cases throughout the first period; in others it soon becomes moist, and there is a tendency to free perspiration, which is readily induced by covering with blankets, warm drinks, etc. Even in those cases in which the skin is hot and dry, the termination of the first period is marked by a soft, cool, and usually moist surface. In exceptional cases, the skin remains hot and dry up to the fatal termination. When death occurs in the stage of depression, the surface becomes cold, and is often covered with a clammy sweat. Many authors have spoken of a peculiar odour given off from the surface of yellow fever patients, and various attempts have been made to define its character by comparison with other known odours. Dr. Rush said that it resembled that of "the washings of a gun." Dr. Jackson describes it as "sickly and faint, and not unlike the smell of a fish-market."

The colour of the skin, which has given its name to the disease, is not always seen, but usually a yellow discoloration begins to make its appearance towards the end of the first period, and later becomes more intense, lasting for some time after convalescence is established. It varies much in intensity, from a slightly yellow tinge to a deep orange or saffron colour. In certain cases the skin presents a mahogany colour, or that of bronze. In fatal cases the yellow colour is developed immediately after death, even if it had not been very noticeable before dissolution took place.

The proportion of cases in which this yellow colour of the skin is observed varies greatly in different epidemics.

Various skin eruptions have been described as occurring occasionally, but there is nothing characteristic about any one of them unless it is the erythematous eruption about the scrotum, which Béranger-Féraud believes to be pathognomonic of the disease. Other eruptions mentioned as occurring occasionally are petechiæ, vesicular and pustular eruptions, livid spots and vibices, erythematous patches

about the knees and elbows, or a general erythematous eruption, papular eruptions, pustules about the mouth, furuncles, etc.

Urine.—The urine in yellow fever, even during the first period, is reduced in amount below the normal standard. This marked reduction, and in fatal cases very commonly complete suppression, is a notable feature of the disease.

The presence of albumen in greater or less amount is a symptom which is so constant that it has come to be generally accepted as one of the pathognomonic features of the disease.

The diminution in the amount of the urinary secretion in connection with the amount of albumen present is of importance in a prognostic sense, as it is to a certain extent an index of the gravity of the attack.

The most marked diminution occurs during the stage of depression, and the few ounces secreted in the twenty-four hours, in severe cases, are loaded with albumen to such an extent as to form coagula which occupy one-half to two-thirds of the contents of the test tube.

In mild cases only a slight trace of albumen may be found in the urine for a day or two, but usually the deposit is sufficiently abundant after the second day to leave no doubt as to its character. It becomes more abundant at the termination of the first stage, and in severe cases throughout the stage of calm, at which time suppression is very liable to occur, especially if, as a result of exposure, the cutaneous transpiration is checked. The early appearance and abundant presence of albumen is generally recognised as an evil prognostic. The reappearance of albumen, after it has once disappeared, indicates a relapse.

The amount of urea eliminated by the kidneys is less than normal, and in inverse proportion to the severity of the attack. According to Cunisset, the quantity is generally in proportion to the amount of urine secreted, and when the amount increases it is a favourable symptom. Uric acid is also said by this author to be present in diminished quantity, but in much less proportion than the urea. He says: "We have seen urine, containing only 7 grammes of urea per litre, give a relatively abundant deposit of uric acid." Bile pigments usually appear in the last days of sickness, and their presence is generally considered a favourable prognostic sign. In exceptional cases the urine may contain blood, from renal or vesical hæmorrhage. The urine in yellow fever almost invariably presents a decided acid reaction.

Nervous Symptoms.—The symptoms connected with the nervous system are varied. In mild cases the intellect remains undisturbed,

and only a moderate amount of transient pain in the head and back marks the onset of the attack. In severe cases the frontal headache and rachialgia are most distressing, and may last throughout the first period of the disease. This stage is also one of great restlessness and jactitation; the patient sleeps but little, and his sleep is apt to be disturbed by distressing dreams; the mind often seems to be in a state of tension; the patient is watchful, excited, and anxious. In other cases the mind is calm; and in others, again, there is a condition of apparent apathy or indifference. Delirium is not a very common symptom in yellow fever, and the intellect often remains unclouded throughout, even in fatal cases. Some cases, however, are attended with incoherency of ideas or hallucinations, and in some there is active delirium. More frequently the mind falls into a torpid condition, the patient is somnolent, and, when awakened, is disposed to be taciturn. A certain number of fatal cases are characterised by active delirium, and a greater number by coma gradually developed. In a limited number of cases death is preceded by convulsions, and occasionally tetanic symptoms of more or less general character have been noted. Among the nervous symptoms may be mentioned deep sighing respiration, sometimes of a spasmodic character. Naturally the respiration is increased in frequency during the febrile stage; in the stage of calm it again becomes normal in mild cases, or sighing and spasmodic in those of a graver character.

Symptoms referable to the Digestive System.—There is complete anorexia during the first stage of the disease; but when the remission occurs patients are very likely to desire food, and even to insist upon having it. Thirst is a constant symptom during the febrile stage, and also in the second period, especially when there is frequent and copious vomiting—black vomit. The bowels are commonly somewhat constipated at the outset. In the second period of the disease there is apt to be more or less diarrhœa, and in fatal cases a dark fluid is often discharged from the bowels, resembling precisely that from the stomach. Occasionally there is a discharge of pure blood, the result of intestinal hæmorrhage. A choleraic form of the disease is sometimes met with, in which there is profuse diarrhœa and collapse, similar to that in the algid stage of Asiatic cholera.

Symptoms connected with the stomach form a prominent part of the history of yellow fever. The characteristic black vomit which has so much occupied the attention of medical authors is for the vulgar the most striking feature of the disease; and even many physicians reserve their diagnosis in early cases during an epidemic

until they have had ocular evidence that the vomited matter is black. The common name of the disease in Spanish countries, "vomito," refers to this symptom. Yet in a majority of the cases there is no vomiting of black matter. Indeed, this is recognised as a very grave symptom; and although a considerable number of recoveries occur after the characteristic black vomit has been ejected, this may be considered an exception to the general rule.

Vomiting is a common symptom during the first period of the disease. Sometimes the fluid ejected has a yellow colour, from the presence of bile, but more frequently it is colourless, and consists only of the fluids ingested, containing, in suspension, a little mucus from the stomach; it is almost always acid. In favourable cases vomiting ceases with the first period of the disease; in those of a graver character, after an interval, perhaps, of twenty-four hours or more, during which there is more or less gastric distress, or a feeling of weight and discomfort, vomiting again occurs, either of a clear acid fluid, or of one of the varieties of black vomit. At first the black material may be in the form of little flocculi suspended in a transparent fluid, "coffee-ground vomit"; and as the case progresses the amount of this material increases, until the whole fluid appears to be uniformly black. Upon allowing it to stand, however, it commonly separates into two portions, and it will be seen that it still consists of a clear liquid with the black matter in suspension. Upon microscopic examination it will be found that the coloured material is made up of little masses which are not black, but have a yellowish-brown colour. A careful examination of recent specimens shows that these little masses contain decolourised and more or less deformed red blood corpuscles and granular leucocytes, and that the brown matter is diffused about these agglomerated cellular elements, and the matrix of mucus in which they often seem to be included. There is no doubt that the black vomit is due to passive hæmorrhage from the gastric mucous membrane, although this has been disputed by some recent authors—Freire, Carmona, Gibier. In certain cases there is vomiting of pure blood, resulting from an active gastric hæmorrhage. The black vomit is not always discharged during life, but it is extremely rare not to find it in the stomach after death.

The fact that the gastric mucous membrane is seriously involved in this disease, is shown not alone by the vomiting, the passive hæmorrhage, and the post-mortem appearances, but also by the constant feeling of discomfort or pain in severe cases, and by a marked tenderness upon pressure over the epigastrium.

Hæmorrhages.—Hæmorrhage may occur, not only from the mucous membrane of the stomach and intestine, but also from that of the

mouth, nose, bladder, and uterus, and even from the eyes or ears. In some epidemics epistaxis is quite common; it may occur during the first period of the disease, but it is more frequent during the second, at which time the general tendency to hæmorrhage is developed. Next to epistaxis, hæmorrhage from the buccal mucous membrane, and especially from the tongue, the gums, and the lips, is the most frequent.

Convalescence and Relapses.—In mild cases of yellow fever, convalescence is quickly established. Instead of the usual depression which marks the second stage of the disease, the patient, after a fever of three to five days' duration, may enter almost immediately into convalescence, and within a week may be ready to resume his usual avocations. But in severe cases, convalescence is often slow, and may be interrupted by various complications—parotitis, buboes, furuncles, abscesses, hepatitis, diarrhœa. In the army, experience shows that but few of those attacked are able to resume their duties in less than ten days, and a considerable number remain in hospital from thirty to fifty days. Blair found the average number of days during which patients were retained in hospital after convalescence was established to be 6·55 for mild cases, and 7·91 for grave cases.

Relapses occur for the most part during the early period of convalescence, or before it is fairly established; they are generally regarded as even more dangerous than the first attack. Occasionally a relapse, or second attack, occurs as late as from two to four weeks after the termination of the febrile period of the first attack.

Prognosis and Mortality.

Prognosis.—The prognosis in yellow fever should always be guarded; for not only is it a very fatal disease in its severer forms, but cases which appear mild at the outset may suddenly assume a grave character. It is more favourable in the case of women and children than with men, and is especially unfavourable in those of intemperate habits, and in plethoric persons who have recently arrived in the infected locality. The principal guide in forming a prognosis at the outset of a case is furnished by the temperature observations. When the body heat does not go above 103° to 103½° F. during the first two days, a favourable result may be anticipated. Later, the amount and character of the urinary secretion is the most important prognostic indication. When this is scanty and heavily loaded with albumen, the case is very grave, although the general appearance of the patient may be favourable,

and he may insist that he is "all right," and wants something to eat. On the other hand, hopes of a favourable issue may be entertained, even in cases characterised by great prostration, and in which the hæmorrhagic tendency is pronounced, if the urinary secretion is tolerably abundant, and the quantity of albumen small. The throwing up of black vomit is by no means a fatal sign; but, as a rule, the passive hæmorrhages which occur during the second stage of the disease are of grave import. Epistaxis occurring during the first stage has been considered by some authors a favourable event (?).

The early appearance of jaundice is an unfavourable sign; as are also frequent vomiting, and great distress and tenderness in the epigastric region. Intense and long continued injection of the conjunctivæ, giving the eyes a fiery red colour, is unfavourable. Delirium, great restlessness and jactitation, and sighing respiration, are all symptoms which give reason for anxiety as to the result.

Mortality.—The mortality from yellow fever varies greatly in different epidemics, and among different classes of the community.

Among the natives of the cities in which the disease is endemic, or in which it has frequently prevailed as an epidemic, it may be as low as from 7 to 10 per cent. One reason for this comparatively small mortality is found in the fact that a considerable proportion of the cases in such a community are among children. Among unacclimated adults the mortality ranges from 20 to 50 per cent., and under certain circumstances even exceeds the latter figure. In the great Philadelphia epidemics of the last and the beginning of the present century, the mortality was from 20 to 80 per cent. In the French Antilles, according to Dutrouleau, the mortality during the years 1851 to 1857, inclusive, ranged from 12·9 per cent. to 50 per cent. Blair gives the mortality at the Seamen's Hospital at Georgetown (Demerara) in 1838 at 19·5 per cent.

At Vera Cruz the mortality for seven years (1875–1881), according to Dr. Molina's statistics, was 41·78 per cent. in the hospital for men, and 41 per cent. in the hospital for women. In Rio Janeiro the mortality for the year 1870 was 17·4 per cent. In the epidemic of 1873 it was 23·3 per cent.

The mortality among the Spanish troops in Cuba, according to the statistics of Dr. Bastarreche, was, in the year 1855, 24·31 per cent., and in the royal navy 17·8 per cent.

Dr. Bemiss gives the mortality rate for the city of New Orleans, during the great epidemic of 1878, as 16·66 per cent.

Treatment.

Remedies that have been recommended.—The unsatisfactory results obtained from the various methods of treatment proposed, are shown by the fact that a majority of the physicians, in those parts of the world where yellow fever prevails, who have had an extended experience, agree that active medication is injurious, and have settled upon an expectant or symptomatic treatment, with careful nursing, as giving the most favourable results. Yet the records of the past show that a certain proportion of the cases recover under all modes of treatment, even the most active and opposed to our present views.

Emetics were formerly considered an essential part of the antiphlogistic treatment. We speak of them only to condemn them, except in those cases in which at the outset of an attack there is evidence that the stomach contains undigested and fermenting material which is the cause of nausea and distress. In this case a simple emetic may be administered, with the sole object of unloading the stomach.

Purgatives have at all times been considered useful at the outset of an attack of yellow fever; and it is the standard treatment, wherever the disease prevails, to administer a cathartic of some kind as soon as the patient comes under observation. Opinions differ as to the best form of cathartic medicine: some prefer a mercurial, some a saline, and others a full dose of oleum ricini. On account of its certain, prompt, and thorough action, and the absence of irritating properties, the last mentioned medicine is a favourite in our own southern cities, in the West Indies, and in Brazil, especially in domestic practice among the creole population. The dose usually given might seem excessive to those who have not seen its favourable action; half a tumblerful, or even more, is the common dose for an adult—2 to 4 fluid oz. Many physicians still prefer a mercurial purge, followed if necessary by a saline cathartic; and calomel is the mercurial usually selected.

It must be remembered that the gastro-intestinal mucous membrane is in a hyperæmic condition early in the attack; and experience has demonstrated that this condition is aggravated by active medication, or by the presence of food,—even the simplest,—and that the gravest danger may result from the administration of anything which may add to the irritation of the stomach or intestine, the normal functions of which seem to be arrested as the result of the action of the toxic agent which gives rise to the morbid phenomena. For this reason it is necessary to administer cathartics with great

caution, if they seem called for after the second day of the disease. As a rule, it will be best to spare the stomach, and to move the bowels with purgative enemata.

The prevailing opinion that yellow fever is closely allied to the malarial fevers, led many physicians, during the first half of the present century, to prescribe quinine in the expectation that it might prove a specific in this disease. The expectation was not fulfilled; but there are still a certain number of experienced physicians who think a full dose at the outset of an attack to be beneficial; others consider it injurious, and, as a rule, its use has been abandoned.

Blair, whose experience was very extensive, and whose treatment seems to have been unusually successful, gave 20 grains of calomel and 25 grains of quinine at the outset of an attack, and under certain circumstances repeated the dose several times.

Dr. Porcher, of Charleston, follows Blair's treatment so far as the first dose is concerned, but protests against a repetition of it.

Symptomatic Treatment.—*To reduce the Temperature.*—In the symptomatic treatment of the disease, sedative doses of aconite or of digitalis, or of one of the modern antipyretics—such as antipyrin—may be cautiously administered during the first stage; but it must be remembered that as the temperature falls and the patient passes into the second stage of the disease, the action of the heart is very feeble, and there is a tendency to death by syncope.

Baths of all kinds—hot, cold, tepid, medicated—have been given in yellow fever, and the evidence is varied as to their utility. The most judicious and experienced practitioners are, however, in accord as to the danger of disturbing the patient to the extent demanded by frequent baths, and, as a rule, content themselves with hot pediluvia at the outset of the attack, and with sponging the surface with cold water or evaporating lotions to reduce the temperature. In the southern part of our own country, and in the West Indies, a hot mustard foot-bath, administered at the outset of the attack, is a standard method of treatment. The patient is wrapped in a blanket, and sits with his feet and legs immersed in a bucket of water as hot as he can bear, to which a liberal quantity of mustard has been added. This has a tendency to relieve cerebral congestion and headache, and often produces a free perspiration. It may be repeated several times during the first twenty-four hours. Cold applications to the head, and repeated sponging of the hands, arms, and chest with cold water, give comfort to the patient, and are of decided benefit on account of their antipyretic effect. Some practitioners recommend the use of tepid water for sponging the surface;

Féraud prefers a mixture of one part of aromatic alcohol with three parts of water; Dr. Peyre Porcher recommends "the assiduous and protracted application of ice-cold water to the head, hands, and arms as long as they are abnormally hot."

Béranger-Féraud speaks highly of the use of large enemata of cold water, frequently repeated, as a means of reducing the temperature. He has made extensive use of this mode of treatment, and has never seen any evil results from it.

To relieve Visceral Congestion.—The use of sinapisms and vesicants to relieve visceral congestion by revulsion to the surface, is approved by experienced physicians everywhere. We prefer sinapisms, and believe that, as a rule, they will accomplish all that can be expected from a blister. The feeling of weight, distress, or absolute pain in the epigastric region, attended with nausea, which is largely due to hyperæmia of the gastric mucous membrane, is often notably relieved by the application of a sinapism to the epigastrium. The patient, who has been restless and uncomfortable, will sometimes quickly fall asleep after such an application. In the same way, cerebral congestion and headache may be relieved by revulsives to the extremities, and lumbar pain by the application of a large sinapism to the loins.

The scanty urinary secretion, and often complete suppression, in yellow fever, seems to call for the use of diuretics; but unfortunately experience teaches that the most reliable medicines of this class do but little good. The kidneys are best relieved by those means which promote perspiration, and by revulsants applied to the loins. Complete suppression is rarely, if ever, relieved by any plan of treatment. In cases with a hot, dry skin, and scanty urinary secretion, with lumbar pain, indicating congestion of the kidneys, we should expect benefit from the hypodermic administration of the muriate of pilocarpine.

To stimulate the Heart.—Stimulants are rarely required before the fourth or fifth day, and must be given at first cautiously and in small doses, so as not to disturb the stomach; later, however, they are often absolutely demanded to sustain the feeble heart. During the second stage of the disease, when the pulsations of the heart often do not exceed 40 or 50 to the minute, there is always a tendency to syncope. This is especially manifested during the night, or toward morning; and many a patient, whose condition seemed satisfactory at the evening visit, has been found by his physician dead the next morning. This dangerous period may often be tided over by the use of stimulants. When the stomach is very irritable, a little iced champagne will often answer better

than anything else; but perhaps the best form of stimulant is good brandy, given in teaspoonful doses at intervals of half an hour, more or less, according to circumstances. It should be given ice cold. Later, milk punch, English ale or porter, may be given in liberal quantities, especially to those who are in the habit of using spirits.

Blair, whose opinion in all that relates to this disease is worthy of attention, prefers hock to any other form of stimulant. He says: "Of all cordials the best is Rhenish wine. When of good quality, it is retained when everything else is rejected, and it is universally liked by the patients. I have seen the most excellent effects from its use, and have often given it to the extent of two bottles in twenty-four hours. I believe it has saved many lives, and know of no substitute for it."

To control Vomiting.—Medicines administered by the stomach to control vomiting, as a rule, are worse than useless. Minute doses of morphine, administered hypodermically, are sometimes useful in checking gastric irritability; but this is a dangerous remedy in yellow fever, and only very small doses are tolerated. Blair has seen stupor, prostration, and complete narcotism follow the administration of one-tenth of a grain. On account of the danger attending its administration, he thinks that it will be more judicious to place it in the *index expurgatorius* of yellow fever.

The Sternberg Treatment.—Recent experience during the last epidemic in the United States (1888) indicates that a mode of treatment suggested by the writer while pursuing his investigations in Cuba in 1887, has a very favourable influence in preventing or checking gastric irritability, and also in increasing the amount of the urinary secretion. The intensely acid condition of the urine and of the vomited matters, and the fact that I have usually found the contents of the intestine more or less acid, led me to think that a very decidedly alkaline treatment might be beneficial; and in view of the probability that the specific infectious agent is located in the alimentary canal, I combined with the antacid selected an antiseptic agent which is known to restrict the development of micro-organisms when present in very minute quantity.

The formula suggested was as follows:—

℞ Bicarbonate of soda, . Grams x (150 grains).
Bichloride of mercury, . Centigrs. ii (⅓rd grain).
Pure water, . . Litre i (1 quart).
℔ Sig., 50 grams (3 tablespoonfuls) every hour; to be given ice cold.

Bichloride of mercury, in a comparatively small amount, was added to the formula, not with the idea that it would to any extent destroy micro-organisms in the intestine, but as an antiseptic, which might be useful in preventing fermentative changes in the stomach, which would perhaps be favoured by the free administration of an alkali. To what extent it is concerned in the favourable results which have been reported by several physicians who have employed this treatment, I am unable to say.

The mercuric chloride, which remains in solution in presence of sodium bicarbonate in the proportions prescribed, would be precipitated by potassium carbonate, which is also contra-indicated in a disease in which there is a great tendency to suppression of urine, and uræmic poisoning. The experiments of Zelz and Ritter, and of Bouchard, show that in uræmia the toxic symptoms are largely due to the retention of potassium salts rather than to urea.

The treatment referred to has been tested by a number of physicians in the United States, in Cuba, and in Brazil, and I have had reports of 374 cases treated by ten physicians. Of these, 301 were whites, with a mortality of 7·3 per cent.; and 73 cases blacks, with no mortality.

During the last epidemic in the United States, at Jacksonville, Florida, Dr. Sollace Mitchell treated 106 cases in the "Sand Hills Hospital" with 5 deaths, a mortality of 4·7 per cent. Of the 106 cases, 79 were whites, and of this number 73 were adult males. All of the deaths occurred among these; and the mortality among this class, considered separately, was 6·8 per cent.

The mortality among cases treated at the Mercedes Hospital, Havana, during a period of seven years (1882 to 1888), as shown by the official statistics, was 43·5 per cent. (total cases, 712). In 1889, Drs. La Guardia and Martinez treated 44 cases in this hospital by the method under consideration, with a mortality of 15·9 per cent. They say in reporting their results: "It appears that the Sternberg treatment has diminished the mortality to less than one-half the mean mortality of this hospital. We have observed the following facts: The patients have offered a notable gastric tolerance during the medication; when treated from the first day, vomiting has rarely occurred. The secretion of urine has always been considerable; even in grave cases, when deaths occurred, they did not die anuric."

Alimentation.—The alimentation in this disease, as in the specific fevers generally, is a matter of prime importance. During the first part of the febrile stage no food is desired, nor is it required. It is best to give nothing in the way of food for the

first three or four days of sickness; after this, if the stomach tolerates it, an ounce or two of iced milk, or of chicken broth, may be given every two or three hours. If the stomach is very irritable, a smaller quantity at shorter intervals may be given; and it will be best to give the milk in combination with lime water. If the stomach will not retain this, or if it gives distress, do not push it further, but support the strength by nutritive enemata.

Even when the stomach is quiet and the patient craves food, it will be necessary to give only liquid nourishment for two or three days, and then to allow nothing but the simplest and most digestible forms of solid food; for there is danger of a relapse from imprudence in diet, even in the mildest cases.

Illustrative Cases.

The following cases which occurred during the epidemic at Fort Barrancas, Florida, 1875, illustrate the clinical history and treatment of the disease:—

Case 1.

Private Rosenberg, aged 32, admitted to hospital at 12.30 A.M., July 21st. —Temperature, 105°; headache; pain in back and loins. To have ol. ricini ℥iss, and a hot mustard foot-bath. 7 A.M.—Temperature, 103°; tongue heavily coated; eyes natural. To have warm tea or cold water *ad lib.* 6 P.M.—Temperature, 104°; pulse, 70; respiration normal; skin perspiring; face flushed; eyes glistening; bowels moved twice. To have small pieces of ice, or iced water.

July 22nd.—Temperature, 104°; pulse, 64; eyes glistening; tongue coated in centre with red margins; in a profuse perspiration; bowels moved once. 6 P.M.—Temperature, 104°·5; urine during the day ℥ix, s. g. 1025, acid, non-albuminous.

23rd.—Morning temperature, 104°·5; feels better; no headache; pain still in back; urine ℥ix, s. g. 1020, acid, albuminous. 6 P.M.—Temperature, 103°; urine ℥ix, acid, s. g. 1020, albuminous; complains of pain in his stomach. Apply mustard poultice at 7.30 P.M. At 8. P.M. feels much relieved.

24th.—Temperature, 101°; slept some, and feels better; urine ℥viij, s. g. 1024, acid, albuminous. Continue iced water or pounded ice *ad lib.* 6 P.M. —Temperature, 102°; urine ℥xij, s. g. 1024, albuminous.

25th, 7 A.M.—Temperature, 100°; urine ℥xv, s. g. 1023, acid, non-albuminous. To have chicken broth, ℥i, every hour, alternately with whisky toddy, ℥ss. Evening temperature, 102°; pulse, 50.

26th.—Morning temperature, 99°·8; pulse, 46; urine ℥viij, s. g. 1020, non-albuminous. To have iced milk and brandy toddy. Evening temperature, 100°; pulse, 52; perspiring freely all day.

27th.—Morning temperature, 98°·4; pulse, 46. To have iced milk and milk toast; continue brandy toddy. Evening temperature, 99°.

28th.—Morning temperature, 98°·4. Observations upon the temperature

and urine were taken until August 2nd; but as convalescence progressed favourably, it is unnecessary to detail them.

Note.—Case reported by Surgeon Sternberg, U.S.A.

Case 2.

Corporal Berger, aged 24 years. Taken sick at 9 A.M., July 21st. Temperature, 105°. To have ol. ricini ℥iss, and a hot mustard foot-bath; warm tea or iced water, as preferred, *ad lib.* 6 P.M.—Temperature, 105°; pulse, 110; tongue heavily coated; is perspiring freely; face flushed; eyes congested. ℞—Hyd. chlor. mite et quinæ sulph. aa grs. x, at once.

July 22nd, 7 A.M.—Temperature, 104°; tongue slightly coated; bowels moved twice; pulse, 80; eyes congested; urine ℥iv, s. g. 1020, acid, albuminous; feels but little better. 6 P.M.—Temperature, 105°. 9 P.M.—℞, Quinæ sulph. grs. xv; temperature at 10 P.M., 105°·5. 11 P.M.—Feels a little better.

23*rd.*—Temperature at 7 A.M., 105°·5; urine ℥viij, s. g. 1020, acid, albuminous; feels no better. To have iced water or pounded ice *ad lib.* 6 P.M.—Temperature, 105°·5; headache, and is very restless.

24*th.*—Morning temperature, 103°; urine ℥xiv, acid, s. g. 1020, albuminous (highly). Feels better; slept some; vomited two or three times. To have pounded ice *ad lib.* 6 P.M.—Temperature, 103°.

25*th.*—Morning temperature, 100°; urine ℥xx, s. g. 1020, acid, albuminous; slept pretty well, and feels better. To have chicken broth ℥i, alternately with whisky toddy ℥ss, every two hours. Evening temperature, 101°·8; skin moist; pulse, 70.

26*th.*—Morning temperature, 100°; pulse, 64. Continue chicken broth and brandy toddy. Evening temperature, 101°.

27*th.*—Temperature, 99; pulse, 60. To have iced milk or chicken broth and brandy toddy. Evening temperature, 100°·6.

28*th.*—Diet: Milk, broth, and toast. Convalescence progressed favourably, and notes were taken upon the temperature and urine until August 3rd, on which day boiled chicken was allowed for dinner.

Note.—Case reported by Surgeon Sternberg, U.S.A.

Case 3.

Private Spaulding, aged 26 years. Taken sick during the night of July 26th. No chill.

July 27th.—Morning temperature, 104°·5; pulse, 118; headache; eyes injected. ℞—Ol. ricini ℥iss, and hot mustard foot-bath. Evening temperature, 104°·4; urine ℥iv, s. g. 1035, acid, non-albuminous; tongue clean; perspiring freely; feels easy except slight headache.

28*th.*—Morning temperature, 103°·8; pulse, 94; bilious vomiting last night, but feels better this morning; no nausea; urine ℥iv, s. g. 1031, slightly albuminous. Evening temperature, 104°·8; pulse, 96.

29*th.*—Morning temperature, 104°; pulse, 96; urine ℥viij, s. g. 1025, slightly albuminous; perspiring freely; vomited a little last night. To have champagne. Evening temperature, 104°; pulse, 85; vomiting ceased; hæmorrhage from nose; urine ℥viij, s. g. 1025, albuminous.

30*th.*—Morning temperature, 102°; pulse, 90; urine ℥vj, s. g. 1025, slightly albuminous; bowels acting freely; slight hæmorrhage from the nose. To have chicken broth. Evening temperature, 103°·5; has been vomiting. To have iced champagne. Urine ℥vj, s. g. 1031.

31*st.*—Morning temperature, 102°; pulse, 84; urine ℥viij, s. g. 1020, albuminous; hæmorrhage from the nose last night. ℞—Ext. ergot fl. ℥i; beef-tea ℥i, every hour. Evening temperature, 101°·4; pulse, 78; urine ℥v, s. g. 1020, albuminous; nose bled to-day; feels very weak. To have champagne.

August 1*st.*—Morning temperature, 101°; pulse, 98; urine ℥viij, s. g. 1018, albuminous; hæmorrhage from the nose continues. Apply tr. ferri perchlor. Evening temperature, 102°; pulse, 80; gums bleeding. Chlorate of potass and iron (tr. ferri chlor.) mouth wash. Urine ℥xiv, s. g. 1015, albuminous.

2*nd.*—Morning temperature, 100°·5; urine ℥xviij, s. g. 1019, albuminous; bleeding from gums ceased. To have milk punch. Evening temperature, 101°·5; violent epistaxis. ℞—Ext. ergotæ fl. ℥i, every hour until relieved. Urine not collected.

3*rd.*—Morning temperature, 101°; urine ℥xviij, s. g. 1039, albuminous; bleeding from nose ceased for a while during night; commenced again on picking his nose. ℞—Acidi gallici ℈i, aquæ ℥i. ℔ A tablespoonful. Evening temperature, 100°·5; urine ℥xiv, s. g. 1015, albuminous. Gallic acid controlled epistaxis, but it was renewed on his picking his nose. Gallic acid to be repeated.

4*th.*—Morning temperature, 100°·5; urine ℥xxiv, s. g. 1013, albuminous; nose bled all night; very weak. Hypodermic injection of ergotine, grs. iii. To have milk punch. Evening temperature, 101°; urine ℥v, s. g. 1011, albuminous; pulse feeble; epistaxis ceased after hypodermic injection; very weak and pallid. Hiccough during the day; stopped by champagne.

5*th.*—Morning temperature, 102°·5; feels stronger this morning; pulse fuller; breathing laboured; no more hæmorrhage; urine ℥xiv, s. g. 1010, non-albuminous. To have milk punch and beef-tea. Evening temperature 100°; urine ℥vij, s. g. 1010, albuminous. Is very weak; sinking. Died at 1 A.M.

Case reported by A. A. Surgeon Salomon.

Remark.—A good example of a hæmorrhagic case in which death finally resulted from exhaustion. It is worthy of note that the epistaxis ceased after the hypodermic injection of 3 grs. of ergotine after all other means had failed to arrest it.

Case 4.

Hospital Steward W. E. Hill, aged 26 years. Taken sick during the morning of July 28th; no chill; general feeling of malaise; took mass. hyd. grs. x, last night; had a hot mustard foot-bath this morning. Temperature, 100°·5; pulse, 86; face flushed.

July 29*th.*—Morning temperature, 100°·8; pulse, 72; urine ℥viij, s. g. 1028, non-albuminous; perspiring freely; feels cheerful; don't care for any nourishment. To have orange-leaf tea *ad lib.* Evening temperature, 100°; complained of pain in abdomen, relieved by application of flaxseed poultice; urine ℥vj, s. g. 1030, non-albuminous.

30*th.*—Morning temperature, 101°·5; pulse, 91; urine ℥viij, s. g. 1025, non-albuminous.

31*st.*—Morning temperature, 102°; pulse, 82; urine ℥vi, s. g. 1027, non-albuminous; slept well, but feels weak. To have chicken broth and arrow-root. Evening: Feels stronger; temperature, 101°·8; pulse, 90; urine ℥iv, s. g. 1025, non-albuminous.

August 1*st.*—Morning temperature, 101°·5; pulse, 90; urine ℥v, s. g.

1027, non-albuminous. Evening temperature, 101°; pulse, 78; urine ℥v, s. g. 1030.

2nd.—Morning temperature 101°; urine ℥iv, s. g. 1030, non-albuminous. Continue diet. Evening temperature, 99°·5; urine ℥vi, s. g. 1027, non albuminous.

3rd.—Morning temperature, 106°. Relapse, produced by exposure to cold during the night; the night was chilly, the thermometer having fallen to 65°, and the patient, having thrown off his bedclothes, was chilled. To have a hot mustard foot-bath, and orange-leaf tea *ad lib.* Urine ℥vij, s. g. 1028, non-albuminous. Evening temperature, 104°; feels better; perspiring freely; urine ℥v, s. g. 1025, non-albuminous.

4th.—Morning temperature, 104°·5; feels very well; skin moist; urine ℥vij, s. g. 1026, non-albuminous. Evening temperature, 106°. To have a hot foot-bath. No action of bowels. To have ol. ricini ℥i, ol. terebinth, ℥ss, per enema. Urine ℥vi, s. g. 1023, non-albuminous.

5th.—Morning temperature, 104°·2; urine ℥xij, s. g. 1025, non-albuminous; feels better; is perspiring freely. Evening temperature, 105°; urine ℥vj, s. g. 1020, non-albuminous.

6th.—Morning temperature, 103°. ℞—Potass. bromidi ʒss, every hour. Urine ℥x, s. g. 1017, albuminous. Evening temperature, 102°·6; urine ℥viij, s. g. 1018, albuminous. Has taken six doses of bromide; very drowsy.

7th.—Morning temperature, 101°·5; urine ℥xviij, s. g. 1014, albuminous. Very weak. To have champagne freely and beef tea. Evening temperature, 102°·5; urine ℥xj, s. g. 1015, highly albuminous. Is a little stronger; pulse fallen. Champagne does not agree, has vomited twice; ale to be substituted. Continue chicken broth.

8th.—Very listless, and inclined to sleep all the time. Morning temperature, 101°·8; urine ℥v, s. g. 1010, albuminous. Evening temperature, 104°; still inclined to sleep, and mind wanders. ℞—Liq. ammon. acetat. ℥ss, every hour. 9 P.M.—Took a sudden change for the worse; pulse hardly perceptible; sinking fast; died at 9.45 P.M.

Case reported by A. A. Surgeon Salomon, under direction of Assistant Surgeon Harvey E. Brown, U.S.A.

Remarks.—This is a characteristic case of fatal relapse, occurring as a result of improper exposure in a case which at the outset was of the mildest character.

CHAPTER VIII.

DENGUE.

BY ANDREW DAVIDSON, M.D., F.R.C.P.Ed.

Definition. — Dengue is a specific febrile disease peculiar to warm climates, characterised by severe articular and muscular pains, and, in most cases, by a primary and a terminal cutaneous eruption.

Synonyms.—Exanthesis rosalia; dandy fever, from the stiff dandified carriage of the sufferer; break-bone fever; broken-wing fever; bouquet fever; giraffe, from the stiffness of the neck; colorado; fièvre éruptive rhumatismale, etc. etc.

Etiology.—*Epidemic History.*—The earliest accounts which we have of this disease do not go farther back than 1779–80. The records of this outbreak are necessarily imperfect; for not only were the distinctive characters of the disease unknown to the physicians of that period, but the medical records from those tropical regions in which it must have prevailed were then extremely scanty. In the former of these years dengue is known to have been prevalent in Batavia and in Egypt, while in 1780 it was epidemic in Spain, Zanzibar, India (the Coromandel Coast), and probably also in Philadelphia, where a disease, which presented many of the characters of dengue, was observed by Rush, and described under the name of bilious remittent fever. If we remember that, although dengue had not at this time been recognised, the incidental descriptions of it which have come down to us indicate its existence in both hemispheres and in four continents, we may justly surmise that it had, at that period, assumed something like a pandemic extension.

Excepting a few limited outbreaks, notably one at Lima in 1818, little more is heard of dengue until the period 1824–28, when the next pandemy, which afforded an opportunity of studying its clinical and epidemic character, occurred. On this occasion it spread over a great part of the delta of the Ganges, extending inland as far as Berhampore; it also appeared at various points on the Coromandel Coast, and was observed at Gujerat in the west and Rangoon in the

east. It was widely prevalent from 1826–28 in the Southern States of the Union, in the West India Islands, and at some points, at least, of the South American continent (Carthagena and Bogota).

Dengue was now distinguished from other fevers, and, as a consequence, after this pandemy had subsided, we meet with numerous accounts of limited outbreaks in various regions. It was observed in Arabia in 1835; in India in 1836, 1844, 1847, and 1853–54; in Egypt in 1845; in Senegambia in 1845–48, 1856, and 1865–67; in Tahiti repeatedly from 1847–56; in Rio de Janeiro in 1845–49; in Callao and Lima in 1851; in Cayenne in 1864; in the Southern States of the Union in 1844, 1850, and 1854; in Havana in 1854, and in Martinique in 1860; in Bermuda in 1837, 1860, and 1863; in Spain and Teneriffe in 1865–67; Port Said in 1868; in Mauritius and Réunion in 1851.

The third pandemic extension of dengue began with its appearance in Zanzibar in 1870, where it seems, according to the recollections of the older inhabitants, to have raged during the former pandemic period (1824–28). From this point it spread to Aden, with which Zanzibar is commercially connected, and thence westwards along the Arabian coast to Port Said, and eastwards to India. From Bombay, Calcutta, and Madras, as centres, it spread along the lines of communication to most parts of the east and west coasts of the peninsula, and inland to the foot of the Himalayas. The epidemy did not rest confined to India, but extended to Burma, Siam, Cochin-China, Java, Celebes, Sumatra, and other islands of this region. It further appeared in 1873 in Mauritius and Réunion, where it was introduced by coolie immigrants from India, and the same year it broke out in some of the Southern States of the Union.

After the subsidence of this outbreak, dengue has only appeared in limited epidemies, such as that at Benghazi in Tripoli in 1878 (where it had already been observed in 1856); on the Gulf Coast of North America in 1880; in New Caledonia in 1884–85; in Fiji, for the first time, in 1885; at Port Said in 1885, and again in wide extension throughout Egypt in 1887. The summer of 1889 witnessed an extension of dengue to both shores of the Ægean and to most of the islands of the Archipelago excepting Crete. Beyrout and Jaffa were invaded in June, and somewhat later it broke out successively in Smyrna, Constantinople, and Salonika, reaching Athens towards the end of summer. It is said to have spread over the whole of Asia Minor and the Lebanon, visiting localities situated at elevations of 4000 and 5000 feet. At Smyrna it is calculated that 150,000 out of a population of 200,000 were attacked; at Jaffa very few escaped the malady.

Dengue is known to be endemic in certain countries. Dr. Sandwith, of Cairo, confirms, in a private communication, the statement of other observers, that dengue is met with in a sporadic form in Egypt every year during the damp months of autumn, and dies out in December. He remarks that in Egypt dengue is usually milder, and the rheumatic element less marked than elsewhere. Pruen has observed the disease in an endemic form in East Central Africa,[1] and ascribes it to the use of foul drinking-water. It is also noticed to occur sporadically from time to time in Senegambia, in Arabia, in some parts of India, in the Hawaiian Islands, and in Bermuda and Honduras.

A Disease of Warm Climates.—From this review of the principal epidemic outbreaks, and of the regions in which it appears to be endemic, it will be seen that dengue is eminently a disease of warm climates. Spain, Turkey, and Greece are the only European countries which have been visited by the disease. In America it has only once extended so far north as Philadelphia, and then it is stated to have died out in October when the cold weather set in. It is not only a disease of warm climates, but even in tropical countries epidemics of dengue usually attain their maximum during the warmest months of the year and decline or die out in the cold season. In the same way it is observed that the elevated and cooler regions of tropical countries are often spared, or at most suffer in a slight degree when the disease is prevalent on the warm coast plains. Thus, when dengue was raging at St. Denis in the island of Réunion in 1872, it did not propagate itself among the inhabitants of Salazie, a Spa situated in the hills at an elevation of 900 metres, although this place was in constant communication with St. Denis, and although the disease was actually introduced into it by visitors from the coast.

Dengue has often remained restricted to coast districts, and has shown but little tendency to invade the interior. This partial limitation to the coast line has been observed in Jamaica, Cuba, Brazil, and Peru, nor has the disease ever extended widely in the interior regions of the United States. This tendency of the disease to hug the shore may, in some cases, find its explanation in the lower temperature of elevated inlands being unfavourable to its spread, as in the instance of Salazie just referred to. That it is not necessarily restricted to coast regions, is evident from the fact that in India it spread in 1872–73 to the foot of the Himalayas, in lat. 34° N. Indeed, we have somewhat uncertain accounts of its having, towards the end of the last century, penetrated into Persia and Tibet. During the warm months of 1889 it broke out, as we have seen, in very high altitudes in Asia Minor.

[1] Davidson's *Geographical Pathology*, vol. ii. p. 692.

Relation to Weather.—The geographical distribution and seasonal prevalence of dengue indicate very clearly the influence of a high temperature in favouring its diffusion. Other meteorological conditions do not appear to be of much importance in relation to its prevalence. It has spread alike during the rainy and the dry seasons, and during calm and boisterous weather. The comparatively cold weather met with at certain seasons in tropical countries is insufficient to stop its progress, for it was during unusually damp and cool weather that it broke out in the West India Islands in 1827–28; but temperatures approaching the freezing point arrest its epidemic prevalence.

Soil and Locality.—The physical and geological characters of the soil appear to be without the slightest significance in respect to its sporadic or epidemic manifestations. Witness its outbreaks on the arid sands and rocks of India and in the flooded delta of the Ganges, on the volcanic soil of Mauritius and the alluvial plains of Senegambia. Low-lying and densely-crowded districts of cities are first and most severely affected when an epidemy occurs.

Mode of Spread.—Having once been introduced into a community, dengue usually spreads among the population with great rapidity. Dumarescq, referring to the outbreak of dengue in New Orleans in 1828, says: "Four persons were attacked by it at the same time, and its spread was so rapid among the inhabitants that, in eight or ten days, at least one-third of the population was labouring under its influence."[1] We would do well, however, not to take too literally such vague statements as that of Moreau de Jonnés, to the effect that one-half of the inhabitants of Havana were attacked by it *almost simultaneously.* It must, at the same time, be admitted that, in respect to rapidity of diffusion, dengue is exceeded by no disease except, perhaps, influenza.

Equally remarkable is the large proportion of the population attacked during an outbreak. In the epidemy which visited Mauritius in January and February 1873, it was estimated that two-thirds of the population was attacked. Perhaps this estimate was under the mark. It would appear that three-fourths of the inhabitants of Smyrna suffered in 1889, and the proportion was still higher at Jaffa. Stedman assures us that scarcely one out of the 12,000 inhabitants of the principal town of St. Thomas escaped.

Its period of epidemic prevalence in a particular locality is limited, seldom exceeding three or four months; but this depends a good deal upon the season of the year. Sporadic cases may occur for months or years after the epidemy has subsided.

[1] Hirsch, *Handbook of Geo. and Hist. Path.* vol. i. p. 77.

The manner in which dengue is propagated has given rise to much discussion. Some have ascribed it to obscure meteorological conditions, or to earthquakes and volcanic agencies—a view which Corre appears to regard with some favour.[1] Others, and we think with better reason, ascribe it to contagion, or infection, or to both.

The rapidity with which it spreads among the inhabitants of a locality in which it has appeared has been urged as a conclusive argument against its diffusion by contagion; and it is certainly an argument not without weight. Hirsch justly remarks that "nothing tells so much against contagiousness as the sudden and simultaneous appearance of the disease over a great part of the population." But the question is: does dengue ever spread in this altogether sudden and simultaneous manner? We have good evidence to prove that the rapidity with which dengue spreads in a community is very great; but the facts in individual instances, when critically examined, do not appear to justify a strict interpretation of the term "simultaneous." If the disease, as we have reason to believe is the case, has a short incubatory period, it will not appear at all impossible on the theory of contagion that it should affect one-third of the population of a large city in the space of eight or ten days, as is stated to have been the case in New Orleans. As soon as it is clearly shown that one-half of the population of a city, such as Havana, can be simultaneously seized with dengue, all question of its spread in a particular community by contagion will be at an end. But the mode of its diffusion in a city, and that of its propagation from one place to another, are entirely distinct questions. It is perfectly conceivable that the germ of the disease may be transportable from place to place by means of human intercourse, and that, having once been introduced into a particular locality, it should grow and multiply in the soil or other surroundings, when these and the climatic conditions are favourable, and that it should thus be spread by infection as well as by contagion.

Whatever may be the manner in which it is diffused in localities into which it has been introduced, the evidence for its transportability from one region to another by human intercourse appears strong, if not altogether irresistible. A general survey of the progress of the epidemy of 1870—of its successive appearance in different places along the lines of communication—is strongly presumptive of its dissemination by human intercourse. But definite facts of its sudden outbreak in communities after the arrival of infected persons go far to convert this presumption into something like

[1] *Maladies des Pays Chauds.* Paris, 1887.

certainty. A few examples only of this kind can be given here. Dickson affirms, and he had the means of knowing, that dengue was introduced into Charleston in 1828 by a vessel from Havana. The captain of the ship communicated the disease to his family, and its further transmission was traced from one person to another.[1]

The steam-ship *Dalhousie*, which arrived at Bombay in December 1871 with dengue on board, embarked European artillery for Cananore. The disease appeared among the Europeans while on board, and on their landing it broke out at Cananore and spread as far south as Cochin. Its outbreak at Trichinopoly, again, in 1872 followed upon the arrival of troops from Madras, where the disease was then prevalent.[2] It suddenly appeared at Mauritius and Réunion after the arrival of immigrants from infected districts in India.

Equally convincing are such instances as the following, which are recorded by Corre.[3] A government official had occasion to visit a locality 14 kilomètres from Tay-Ninh in Cochin-China where dengue was prevalent. Here he contracted the disease, and after his return home a colleague, with whom he lodged, became affected *within three days*, and he, in turn, communicated it to a European boy who attended him in his sickness.

The first case of dengue that occurred on board the *Andromaque*, in the harbour of Port Said, was in the person of the doctor who had visited patients suffering from it on shore. The disease afterwards spread among the men on board. The frequency with which medical men and nurses contract the disease is a matter of common observation.

Race, Age, Sex, etc. — No race enjoys an immunity from dengue, although some differences have been observed as regards their liability to contract the infection. The negro has been observed in some epidemics to escape more frequently than Europeans. This was the case, according to Stedman,[4] in St. Thomas; yet the three deaths ascribed to the disease in that island occurred in negroes. In Java in 1872, and at Benghazi in 1878, the negro race is said to have enjoyed a remarkable immunity; but it appears from the statement of Pasqua that in Egypt and Senegal negroes were as liable as others to contract the infection. In some outbreaks it has been observed that the natives of India have suffered to a larger extent than the Europeans. The Chinese at Amoy are stated to have suffered more frequently and severely than the European residents, while in Cochin-China the disease was more common among the Anamese than the

[1] *Amer. Journ. Med. Sciences*, vol. iii. p. 3.
[2] Smart, "Epidem. Soc.," Feb. 13, 1877.
[3] *Loc. cit.*
[4] *Edin. Med. and Surg. Journ.* vol. xxx.

Chinese (Hirsch). How far these differences are to be explained by circumstances which involve a greater or lesser exposure to the infection, or to a greater power of resistance in certain races, we have no means of determining. The fact that the same race exhibits a varying degree of liability to the infection in different countries, seems to show that the liability is mainly determined by other circumstances than racial peculiarities.

The youngest ages are not exempt. Children a few days old have been attacked; and it has been stated that children have been born suffering from it. The disease seems in many instances to be specially severe in the aged, who often suffer from rheumatic pains and debility for months after the attack. In some epidemies pregnant women attacked with dengue have been little liable to abort, in other outbreaks abortions and miscarriages have been very frequent.

One attack of dengue usually confers an immunity from a second infection. Dickson, however, makes the interesting observation, that if the eruption proper to the second stage of the disease is absent, the patient is liable to have relapses.

Epizootic disorders are recorded to have coincided with epidemies of dengue at Cadiz in 1784, and at Baroda and Rangoon in 1872. Whether in these instances the animals suffered from dengue has not been determined.

Analogy points to the parasitic nature of dengue; but although several observers have found what they believed to be parasitic bodies in the blood of patients suffering from the disease, the specific agent of the infection has not hitherto been satisfactorily demonstrated.

Symptomatology.—The prominent symptoms of dengue are fever, pain, and cutaneous eruptions. Its clinical features, although remarkably variable, are generally distinctive. After a period of incubation, which may vary from a few hours to several days, the patient is suddenly seized with severe arthritic and muscular pains, headache and fever, accompanied by a cutaneous eruption, which is as varied in its type and seat as it is fugacious in its character. This may be described as the period of invasion or of primary fever. After the fever has lasted for one or two days a copious perspiration breaks out, which is followed by a remission of the fever, and an amelioration of the rheumatic symptoms, constituting what is called the period of remission, which generally lasts for two or three days. A second febrile movement now ensues, with more or less aggravation of the joint-pains, and a scarlet efflorescence, which generally appears on the palms of the hands and rapidly spreads over the body. This, which is the terminal, the most constant, and, indeed, the only

characteristic eruption of dengue, may disappear within the space of a few hours, or may persist for two or even three days. This is the eruptive stage of the disease. A fourth, or rheumatic, stage is frequently observed. A few days, or it may be one, two, or three weeks, or even longer, after the subsidence of the eruption, excruciating pains, not accompanied by fever, may recur in the fingers, toes, wrists, ankles, or knees. The fingers are not only painful, but are swollen, stiff, itchy, and extremely tender and sensitive. The pains in the joints are most severe in the morning, and become alleviated towards evening. Several joints may be thus affected simultaneously or in succession, and the attack may pass off in a few days or persist for one or two weeks. Relapses of these rheumatic symptoms are not uncommon, and we have known instances in which recurrences of joint-pains have been experienced for years at irregular intervals in persons who have suffered from dengue, and which appeared, indeed, to be in some sense a result of the disease.

A few points respecting the leading symptoms require brief notice.

Fever.—Premonitory symptoms are rare. Occasionally it happens that for a time, varying from three to twenty-four hours before the attack, the patient complains of lassitude, vertigo, a feeling of cold down the back, or of numbness of the extremities. Suddenness of accession is, however, a marked feature of the disease. In one instance, which illustrates the unexpected manner of attack, a gentleman went on board a vessel in the harbour of Port Louis, in perfect health, to pay a short visit, and had to be carried on shore suffering from fever and racked with pain. Rarely is the disease ushered in by distinct rigors; more frequently its onset is announced by slight chills; but these are often absent. In children, it is not rare for the disease to commence with convulsions.

The temperature in dengue fever rises rapidly, and attains its acme within twenty-four hours. Its usual course may be seen from the following observations by Vorderman:—

	First Case.		Second Case.	
	Morning.	Evening.	Morning.	Evening.
1st Day, . .	40°·8	41°·2	38°·0	40°·4
2nd ,, . .	38°·5	39°·0	39°·0	39°·8
3rd ,, . .	37°·5	36°·7	39°·4	38°·0
4th ,, . .	37°·2	37°·3	37°·8	37°·2

Dengue has occasionally been observed to present the febrile characters of ague, each accession being marked by a cold, hot, and sweating stage. During the fever, the patient sometimes complains of a feeling of intense cold. Dr. Stedman, who observed and

suffered from the disease in St. Thomas, informs us that he had to cover himself with three blankets, although the weather was at that time sultry. There is always great restlessness, and sometimes nausea and vomiting, with intense temporal and frontal headache, pain in the eyeballs and congestion of the conjunctivæ. The pulse is always fast and weak, and the respirations correspondingly accelerated.

When the fever falls the pains abate, but the patient remains languid, depressed, and restless.

The secondary fever may be absent, and when it is present it is very irregular in severity and duration.

Pain.—Pain, at some stage, is a constant symptom of dengue, and frequently it is one of the earliest, as it is one of the most persistent, symptoms. Its severity, however, differs greatly in particular outbreaks and individuals, as well as in the different stages of the malady. In the epidemy described by Stedman in St. Thomas,[1] the disease, we are told, generally announced itself by a sudden pain in one of the fingers—usually in one of the little fingers. The pains are generally at first located in the small joints, which may be swollen and tender as well as painful; but after a time the large articulations become affected, and the neck, back, and often, indeed, the whole body, is racked with pain. The pains, which, as we have said, abate with the remission of the fever, become accentuated during the eruptive stage, and may recur once or oftener after the disease in its febrile manifestations has long disappeared. This post-febrile rheumatic stage is often wanting. It should be noted that in the epidemic fever with scarlet eruption observed by Goodeve in Calcutta in 1853,[2] severe pain in the joints was seldom observed.

Eruptions. — An eruption during the primary stage is only met with in about two-thirds of the cases. It may present the appearance of an erythematous rash, of a miliary or papular eruption, or of a scarlet or rubeolar efflorescence. It usually lasts only for a few hours; sometimes it has been seen to disappear in half an hour, at other times it has been more persistent. The characteristic eruption, which follows the remission, appears about the fifth or sixth day of the disease; but in some cases even this eruption is absent, or is so fugitive as to escape detection. It generally takes the form of a scarlet rash, commencing on the palms and then spreading to the rest of the body. Occasionally it assumes the character of urticaria, the wheals being of a red colour on a scarlet ground. In the severer cases observed by Stedman, there were "swelling of the feet, hands, and eyelids, attended with distressing tingling, which, as the eruption disappeared, became an intense itching." This eruption is always

[1] *Loc. cit.* [2] *Indian Annals of Medical Science*, October 1853.

followed by desquamation, and occasionally by boils, carbuncles, abscesses, or ulcers.

The mucous membrane of the mouth seldom fails to become involved. In most cases it is reddened and aphthous. In the Calcutta epidemy described by Goodeve, to which we have already referred, the gums, the inside of the cheeks, the uvula, and the posterior third of the palate were of a bright red or vermilion colour. But in dengue the throat complications proper to malignant scarlet fever are never present.

When the eruption subsides, the patient is left in a state of great nervous prostration, and is more or less anæmic.

The Urine.—The following is Poggio's analysis of the urine in dengue, which is not without interest:[1]—

Constituent Elements. Proportion per 100.	Normal Urine.	Urine in Dengue.	
		During the Fever.	During the Eruption.
Urea, ...	3·00	2·920	2·925
Uric Acid, ...	0·10	0·104	0·100
Salts and Organic Matters, .	3·60	12·074	6·045
Water, ...	93·30	84·902	90·930

Digestion.—The tongue is coated; the patient often complains of nausea, anorexia, and colic. The bowels are constipated. In a few cases the skin becomes jaundiced.

Glandular Swellings.—During the period of remission, the glands of the groin, axilla, and neck frequently enlarge. This enlargement may pass off in a few days, or the glands may continue swollen and painful for a considerable time after the other symptoms have disappeared. The salivary glands are often enlarged and tender, and in some cases the patient suffers from troublesome salivation. Enlargement of the parotid frequently occurs; but suppuration, although it has been occasionally observed, is very rare.

Prognosis and Morbid Anatomy. — Dengue is never directly fatal, except in the case of old or otherwise worn-out subjects, in whom fatal syncope has in a few instances been observed. The few deaths that occur in patients suffering from dengue are the result of some anterior or intercurrent affection rather than of the disease itself. The prognosis is thus entirely favourable in every

[1] Poggio, *La calentura roja observada en sus apariciones epid. de los años* 1865 y 1867. Madrid, 1871.

uncomplicated case of dengue, however distressing or even alarming may be the symptoms. The scanty post-mortem records of deaths from dengue throw no light whatever on the morbid anatomy of the disease.

Diagnosis.—The presence of the eruption and the course of the fever are sufficient to prevent dengue from being mistaken for rheumatic fever, while the rheumatic symptoms will prevent it from being confounded with any of the exanthemata, such as scarlet fever, measles, or roseola. Scarlet fever, the eruption of which is often closely simulated by that of dengue, is seldom seen, and never in extensive epidemies, in many of the tropical countries which have been most frequently and extensively visited by the latter.

In its wide distribution, its rapid diffusion, and in the numbers attacked, dengue is closely allied to influenza. But the clinical characters of the two diseases are entirely different, although in every epidemy of influenza cases occur in which rheumatic pains and a scarlet eruption so closely simulate dengue, that it will only be possible to avoid error by interpreting the symptoms in the light of the prevailing epidemy. Although influenza in exceptional instances may thus be mistaken for dengue, the latter seldom presents the characters of influenza.

Treatment.—As dengue is so little dangerous, treatment should be directed solely to the relief of urgent symptoms. Fever scarcely requires to be combated, for, although it runs high, it subsides rapidly and spontaneously. Some advantage may, however, be derived from antipyrin or salicin, not only in reducing the temperature, but in relieving the pain. Constipation should be obviated by mild laxatives; but it is well to remember that the articular pain is often so severe that frequent calls to stool occasion great distress, so that free purgation will only increase the sufferings of the patient. The pain, if not relieved by the use of the remedies suggested, may call for the administration of full doses of opium, which have proved exceedingly useful. The nervous depression which marks the eruptive stage demands the use of stimulants and restoratives, while the resulting debility and anæmia indicate the exhibition of quinine and iron, which are also the remedies most effective in preventing the relapses of arthritic pains.

CHAPTER IX.

THE PLAGUE.

BY MONTAGU LUBBOCK, M.D., F.R.C.P.

Synonyms.—*Λοιμός* (Greek). Pestis, pestilentia, lues, morbus contagiosus, febris pestilentialis, pestis (or clades) inguinaria or glandularia, vera pestis, typhus pestis, pestis bubonicus (Latin). Pestilence, black death, bubonic, Levantine or Oriental plague (English). La peste (French). Die peste (German). La peste or pestilenza (Italian). La peste or plaga (Spanish). Indian plague, Bombay plague, Pali plague, Mahamurree (names given to the plague in India).

Definition.—A specific, contagious, and epidemic disease, usually acute and febrile, which specially affects the lymphatic system, producing inflammatory enlargement of the glands (buboes). Hæmorrhage in the form of epistaxis, hæmoptysis, hæmatemesis, melæna, or hæmaturia may occur, and carbuncles, petechiæ, or other exanthems are occasionally present. The glandular swellings and cutaneous manifestations may be absent in acute forms of the disease.

History.—The name of "plague" or "pestilence" has been given to any epidemic disease which is fatal to a large number of persons, and the existence of many severe pests is therefore historically known of which the nature is uncertain. Such are the plagues of Egypt,[1] and in the Wilderness;[2] the plague of Œgeria,[3] and that in the Grecian camp at the siege of Troy;[4] the plague in Canaan;[5] the plagues which occurred at Rome in 738 B.C.,[6] 461 B.C.,[7] 451 B.C.,[8] and 433 B.C.;[9] the plague of Athens in 430 B.C., recorded by Thucydides;[10] and those at Rome in 363 B.C.,[11] 295 B.C.,[12] and 175 B.C.[13]

The first undoubted historical allusion to true plague was made

[1] Ex. xi. 1.
[2] Num. xi. 33.
[3] Ovid, *Metam.* lib. vii. 523.
[4] Homer's *Iliad*, Book i. 10, etc.
[5] 2 Sam. xxiv. 15.
[6] Plutarch's *Life of Romulus.*
[7] Livy, iii. 6.
[8] Livy, iii. 32.
[9] Livy, iv. 21, 25.
[10] Book ii. 49–51.
[11] Livy, vii. 1.
[12] Livy x. 31.
[13] Livy, xli. 21.

by Rufus the physician, who is supposed to have lived in the reign of Trajan (A.D. 98–117). He states that pestilential buboes (λοιμώδεις καλούμενοι βουβῶνες) are mentioned by the contemporaries of Dionysius, who lived at the beginning of the third century B.C., or at an earlier date, and adverts to the disease as described by Dioscorides and Poseidonius in the second century of the Christian era, and which existed in Libya (Egypt) at their time.[1]

In the sixth century A.D., the bubonic plague, called the plague of Justinian, from occurring in his reign (A.D. 565–74), spread over the whole Roman Empire. Originating, as supposed, in Egypt in the year 542 A.D., it extended in an easterly direction to Syria, and in a westerly to Constantinople, where a thousand persons died daily. The disease then overran the whole of Europe, spreading devastation wherever it appeared, and receiving the name of "pestis inguinaria" or "glandularia," which it retained until the seventeenth century.

Severe pests occurred frequently in the Middle Ages, some of which were undoubtedly examples of true plague. Since, however, the description of the disease is in most cases limited to an announcement of the date of its appearance and the number of victims, while such epidemics as those of typhus, smallpox, etc., were looked upon as outbreaks of plague, the true nature of the disease is usually uncertain. It is only from the fact that in some cases it was called by the name of pestis inguinaria or glandularia, that the occurrence of true plague can be at times determined.

In 1347 A.D. the disastrous pestilence known as "black death" (probably on account of the petechiæ present upon the surface of the body) appeared in Europe. Supposed to have originated in Cathay (China) or Tartary, and to have spread thence into the Crimea, it was imported from that place into Constantinople. The disease then invaded the whole of Europe. Turkey, Greece, Italy, Spain, France, England, and the Scandinavian countries were overrun by it, while in all Europe, Hecker[2] believes 25 million persons, or one-fourth of the whole population, to have perished.

The plague occurred frequently during the fifteenth and sixteenth centuries in different parts of Europe. It appeared in London in 1400, 1406, and 1428, and though probably endemic in England during the fifteenth century, is specially mentioned as having occurred in this country in 1472 and the succeeding years, whilst London was severely attacked in 1499–1500.

[1] *Œuvres de Oribase*, by Bussemaker and Daremberg, 1858, vol. iii. p. 607; Works of Oribasius, lib. 44.

[2] *Epidemics of the Middle Ages*, by J. F. C. Hecker, p. 30, Syd. Soc. 1844.

At the beginning of the sixteenth century the plague is said to have been most destructive in China, which it almost depopulated. It occurred in London in 1537–39, 1547–48, 1563–64, 1592, 1599.

In 1603 there was a severe epidemic of plague in Egypt, where one million persons are said to have died from the disease, and though the plague had now begun to decrease in Europe, the Continent was visited by many severe epidemics during the seventeenth century. London suffered again in 1609, 1625, 1636, and 1647, after which year, although sporadic cases still occurred in the country, England was almost free from plague until 1664.

In 1656 the plague appeared in Europe in its most aggravated form. After being very destructive at Naples, where 300,000 deaths are said to have occurred in five months, it spread to the rest of Italy, and invaded the other countries of Europe. So fatal and malignant was the disease, that many places were almost depopulated by it. Thus, while 14,000 persons died at Rome, Geneva lost 60,000, Amsterdam 50,000, and London 70,000 lives. This, "the great plague of London," began in that town in 1664, and became more virulent during the spring and summer of 1665, the number of deaths gradually increasing until September, during which month more than 30,000 deaths occurred. It then abated, although in 1666 nearly 2000 (1998) deaths were due to this cause. The total number of deaths from plague in London during 1665–66 was 70,594, the total population of the town being 460,000, of whom two-thirds are supposed to have departed in order to avoid the disease. The plague then spread over the rest of England, and did not disappear from the country until 1679, since when no case of the disease has occurred in it. During the remainder of the century there were occasional outbreaks of plague in some parts of Europe (Spain, Italy, Germany, Austria, Poland, and Turkey), but the area of plague in Europe was now becoming narrower; and whilst the British Isles, the North of France, the Netherlands, Belgium, and Switzerland have been totally free from the disease since this period, the South of France has suffered from but one epidemic (1720), the western limit of plague now occupying a more easterly position.

In the eighteenth century occasional outbreaks of plague occurred in Europe, being confined with few exceptions to the eastern portion of the Continent. In 1708 and 1711, Granada, Seville, and the South of Spain suffered from the disease, which is the last record of plague in that country, whilst in 1720 it overran the South of France, having been apparently introduced into Marseilles by a

vessel arriving from Syria, in which country plague then existed. Cases of plague had also occurred in the ship. Since that epidemic France has been free from the disease.

In 1710 plague prevailed in Scandinavia, in 1743 at Messina, whilst epidemics occurred from time to time in Eastern Europe, Egypt, Northern Africa, Turkey in Asia, Egypt, Palestine, and Mesopotamia. During this century plague was still retreating in an easterly direction from the soil of Europe.

In the nineteenth century the same easterly recession of the plague has continued, and no considerable epidemics have occurred in Europe except at its eastern part. The following list represents the places in which plague has existed during the present century:—

1801, Egypt, Mesopotamia (Bagdad). 1802–03, Constantinople. 1803, Turkey (Salonica). 1804, Egypt. 1805, Armenia Bayazid), Georgia and the Caucasus (cases having probably occurred in the region of the Caucasus from 1798 until 1819 or longer), 1806–7, Southern Russia (Astrakhan and the north side of the Caucasus). 1808, Constantinople. 1809, Turkey in Asia (Smyrna). 1810, Egypt. 1812, Constantinople, Turkey, Egypt, Malta, Turkey in Asia (Smyrna, Cyprus, Trebizonde), Russia, Armenia, Mesopotamia (Bagdad). 1814–15, Egypt, Turkey, Italy (Noja), Austria (Dalmatia), Corfu, Arabia (west coast). 1816, Constantinople, Greece (Morea, Corfu, Cephalonia), Turkey in Asia (Smyrna), Candia, Cyprus. 1817, Morocco. 1818–19, the north coast of Africa from Egypt to Tangiers. 1823, Egypt. 1824, Egypt, Armenia. 1826, Constantinople, Greece (Morea). 1827, Greece, (Morea), Turkey in Asia (Aleppo, Erzeroum). 1828, Greece (Morea), Turkey (Moldavia, Wallachia), Turkey in Asia (Erzeroum). 1827–29, Tripoli, Russia (Odessa), Syria. 1830–31, Constantinople, Cyprus, Syria (Beyrout), Arabia (Mecca), Mesopotamia (Bagdad). 1832, Arabia (west coast). 1833–34, Turkey in Asia (Smyrna), Egypt (Alexandria). 1834, Constantinople, Egypt, Syria, Mesopotamia (Bagdad). 1836, Algiers. 1837, Greece, Turkey (Roumelia), Russia (Odessa), Turkey in Asia (Smyrna), Egypt. 1840–41, Turkey in Asia (Erzeroum), Constantinople, this being the last appearance of the plague in Turkey. 1841, Egypt (Damietta, Cairo, etc.), Palestine (Jaffa, Jerusalem, etc.). 1842, 1844, 1845, Egypt, since when this country has been free from the epidemic. 1852, San Christobal de la Laguna, in the Canary Islands. 1853, Arabia (west coast). 1856, Tripoli (Benghazi), where the disease was called typhus with glandular swellings; thence it was imported into the island of Chios (where it was termed petechial typhus analogous to that of Benghazi), and into the Lazaretto at

Alexandria. 1858–59, Tripoli (Benghazi) and Fezzan (Murzuk). 1856–85, and probably since, Mesopotamia (Irak, Dagarra, Bagdad, etc.), where the disease has been regarded as adynamic typhoid fever, or intermittent fever with glandular swellings. 1859, Syria (Beyrout). 1863–64, 1870–72, 1876–78, 1881–83, 1885–86, and since, Persia (chiefly the Province of Azerbijan), the hæmorrhagic form occurring frequently in certain epidemics. 1877–79, Russia (Province of Astrakhan), where it was at first regarded as intermittent fever with buboes, or by others as croupous pneumonia, or typhus complicated by pneumonia. 1879, Arabia (west coast). 1889, Arabia (west coast).

India.

1815, Island of Cutch, extending in 1816 to Gujerat, and in the following year to Sind, Hyderabad, Ahmadabad, and Dollera, and not disappearing from this region until 1821. 1823, Provinces of Kumaon and Gharwal (south-western slopes of the Himalayas), occurring again in 1834, and extending along the Pindar, and again in 1847, remaining in this territory until 1853. 1836, Pali in Rajputana, spreading to Jhodpore, the capital of Marwar, and to the state of Mewar; hæmorrhagic form frequent. 1876, Gharwal. 1884, Kumaon.

China.

1850–1878 and since, South of China (Province of Hunan, Kiang-si, Kwei-chau, and at Pakhoi and Lien-chau, to the north of the Gulf of Tong-king).

Plague has very rarely occurred within the Tropic of Cancer, the exceptions being when it occurred upon the western coast of Arabia as far south as 19° lat., in India upon the island of Cutch, in Gujerat and Rajputana, and in Southern China. It has never appeared in the Southern Hemisphere or the New World.

Symptoms.—Rapid death within a few hours of the onset of the disease only occurs during the most fatal period (usually at the beginning or at the height) of severe epidemics. In most cases the duration of the plague is from one to three weeks, and the symptoms occur at different periods of the disease.

1. *Period of Incubation.*—The incubation varies from a few hours (in malignant epidemics) to two, or exceptionally, three or four days. It has been supposed in some cases to be as long as eight or even ten days.

2. *Period of Invasion.*—There is often a feeling of discomfort and

fear which cannot be described, the face assuming a dejected, resigned, gloomy, or wild and defiant expression. Rarely it appears vacant or bewildered, but may indicate a feeling of anxiety, and the expression is said at times to be so peculiar as to indicate the disease, although its special character cannot be described. The face has often a yellow hue. Headache is apt to occur, which is usually frontal, and causes a darting or stabbing pain, which may be almost intolerable. Usually ceasing after the first few days, it may persist until the end of the disease, and is often accompanied by such giddiness that the patient can scarcely turn his head upon the pillow without its occurrence. Pain, at times severe, may be also felt in the epigastrium, loins, back, or extremities, at times stabbing in character, and giving rise to the belief that an arrow has been shot by an invisible demon,—an idea which is said still to exist in Mohammedan countries. A sensation of burning heat is also liable to be felt in the throat, or in the epigastric or precordial region, or severe pain may occur in the region of the glands, which are to be subsequently enlarged. The general sensibility is usually diminished, but hyperæsthesia may exist.

The mental faculties are nearly always affected, although in some epidemics the intellect has been little, if at all, troubled, and the mind has remained clear until the time of death. More often, however, some form of mental aberration has existed. There may be a feeling of drowsiness, or uncontrollable restlessness and insomnia may occur. Alarming objects may be seen, and great terror felt. The patient may be troubled by distressing dreams, and an impulse to suicide may exist. Delirium, usually quiet, but sometimes noisy, or a typhoid condition, lethargic sleep, stupor, or coma at times occur before the onset of fever. The gait is often uncertain, resembling that of an intoxicated person, but although the agitation and disorder of the movements may be carried to an extreme degree, paralysis rarely occurs. Nervous trembling, lasting from a few hours to three days, may precede the onset of fever, being unaccompanied by any sensation of cold or depression of temperature, and usually followed by profound coma ending in death. Convulsions may occur. The voice is usually feeble, and interrupted or trembling, or the power of speaking may be entirely lost. The eyes are often red and injected from inflammation of the conjunctiva, but at times are dull and lustreless. The sight may be impaired, or strabismus exist. The hearing is often obtuse, deafness having been considered a favourable indication in some epidemics.

In severe cases the respiration is apt to be hurried (40 to 50 in the minute) and laboured, and a dry, frequent, paroxysmal cough may

supervene. In rare cases pneumonia occurs. Epistaxis and hæmoptysis have been frequent in some epidemics, constituting the most striking symptom of the disease, and being supposed to indicate a special form of plague.

The tongue becomes rapidly covered by a white coating, compared to chalk or mother-of-pearl. In other cases it is said to be of red, green, or black colour, or it may be white at the centre and red at the margins. It is often dry, but may be moist, and at times is so large that it only passes through the mouth with difficulty. The salivary secretion may be much increased, but is more often diminished. The appetite is usually lost, but may be insatiable. Nausea and vomiting are at times persistent symptoms, the vomited matters being at times partly composed of blood. Diarrhœa, which usually follows the vomiting, may exist, the motions having often an offensive odour, and possibly containing blood. Tumefaction of the abdomen, and increased size of the liver and spleen, may be observed.

The urine has often been noted to be of normal character, being usually pale and clear, but hæmaturia may occur, or the urine be secreted in large quantity.

These symptoms may constitute the whole disease; and in severe epidemics death may occur without the occurrence of pyrexia or glandular enlargement.

3. *Period of Fever.*—After from a few hours to one or two days, pyrexia usually occurs. Often accompanied by one or more rigors at its commencement, which may last from one to ten hours, and resemble those of intermittent fever, the pulse becomes more rapid (130 or more in the minute), and the temperature rises to 102°, 104°, or even 106° F. or higher. In exceptional cases the fever is slight or absent; but more often pyrexia exists, the skin being hot and dry, and the patients becoming at the same time more weak and prostrate. The decubitus is dorsal, and the delirium, typhoid condition, or coma are more pronounced. The tongue is often covered with a brown or black crust, and sordes collect upon the teeth and lips. There is subsultus tendinum, and collapse may exist, the pulse becoming very rapid, weak, small, and scarcely perceptible; or carbonic acid poisoning may occur, as indicated by lividity and cyanosis, which especially affect the face. The urine may become scanty and contain blood, or be suppressed, and death occur. Hiccough is apt to occur at the end of serious and fatal cases.

4. *Period of Glandular Swelling.*—In most cases, after two or three days of fever, perhaps less, buboes appear, this being the most characteristic symptom of plague. The fever usually becomes less

severe when this happens, and abundant sweating may occur. The pulse is often slower (90 to 100), the temperature falls, and the mental condition is better. The enlargement of the glands often occurs suddenly, but may have been preceded by severe pain in the region which they occupy. The inguinal, axillary, cervical, and other external glands may be involved, or glands situated internally, such as the external or internal iliac, sacral or lumbar glands in the abdomen, and the mediastinal, bronchial, or cardiac glands in the chest. The affected glands belong to the lymphatic system, the inguinal glands (usually the inferior vertical before the superior oblique set) being most often affected, after which, in order of priority, the axillary, the cervical, the popliteal and other glands of the extremities and the internal glands are involved. The cervical glands are said to be most often affected in children, the enlargement in some cases impeding the movement of the jaw, or interfering with respiration or deglutition. The swelling of the axillary glands usually ends in resolution, and this is the best position for the buboes to occupy. Their size varies from that of a hazel-nut to that of a goose's egg, and their surface may be smooth or nodular. The number varies. Usually there is but a single bubo, one gland or set of glands being alone involved; and next in order of frequency two buboes occur (most often in both groins), but three, four, or more may exist. The right side is more frequently affected than the left. Buboes occur in more than two-thirds of the cases of plague, the inguinal glands being enlarged nearly twice as often as any others. The enlargement, though at times the first indication of the disease, usually occurs on the second or third, but in some cases not until the fourth or fifth day, or even quite at the termination of the malady. Finally, resolution or suppuration occurs, the latter being the most favourable termination. The swelling then becomes more prominent, the skin covering it more thin, and an opening forms through which pus, sloughing cellular tissue, and blood escape, the discharge having at times the most offensive odour. Gangrene may occur in this and the adjoining parts, perhaps with serious consequences, such as erosion of a blood vessel and fatal hæmorrhage. When the opening has formed the part heals in most (two-thirds) of the cases, but may become indurated, specially in weak patients, this being an unfavourable indication. The suppuration may again continue, and produce a fatal result. The cicatrix which forms has large indurated margins, and presents a characteristic appearance.

Carbuncles occur far more rarely than buboes, viz. in from 3 to 5 per cent. of the cases. They have been termed "gangrenous

pustules," as they are formed by one or more pustules, surrounded by a red areola, and containing serous fluid of brown or black colour. After enlarging to about the size of a hazel-nut, they burst through the skin, leaving a gangrenous surface in their place, and the slough separates, leaving an ulcer with sharply cut margins. They are specially apt to occur upon the trunk and extremities, but may appear upon the genital organs, scalp, face, or, in fact, any part except the palm of the hand or sole of the foot. They have been found upon the buboes. There are rarely less than three, and may be as many as twelve or even more, being seen from the first to the third, or even as late as the seventh day, and existing in some cases when buboes are absent. They may occur only in fatal cases, or appear, as in some epidemics, towards the end of the outbreak, when it has lost its severity. Not only the skin, but the subcutaneous tissue, and even the subjacent muscles and bones, may be involved, and a slough one or two inches in diameter be formed. They usually appear before, but at times after the buboes, and as the buboes increase they are apt to diminish in size, and *vice versâ*. When the adjoining skin becomes gangrenous, the gangrene may spread in all directions, involving the adjacent structures for some distance, and possibly the whole of a limb. The part heals as when gangrene occurs in other circumstances, well marked and irregularly formed cicatrices remaining in the place of the carbuncles.

Petechiæ usually appear in the most unpromising cases, and indicate the approach of death, although recovery after their existence is not impossible. These constituted the "plague spots" or "tokens of the plague" mentioned in the popular accounts of the disease which have been written from time to time, and gave the name of "Black death," as already observed, to the epidemic which occurred in the fourteenth century. They vary in size from that of a scarcely perceptible point to that of an almond, and may occupy any part, occurring specially upon the neck, chest, and extremities, and but rarely upon the face, but appearing in some cases upon the mucous membrane of the tongue, gums, or eyelids. Their colour may be purple or black, and they are the more serious in proportion as they have a darker hue. They vary in number, almost the whole skin being in some cases covered by them. Usually seen at the height of the disease, they appear in the most severe cases from the third to the seventh or eighth day, being in the latter case the most certain indication of approaching death.

Patches of erythematous redness, bullæ, pustules, ulceration with diminished sensibility of the skin, and large extravasations of blood

in the subcutaneous cellular tissue immediately before death have been noted.

5. *Period of Convalescence.*—Convalescence usually begins from the sixth to the tenth day, being often announced by suppuration of the lymphatic glands. It may be of short duration, but is often lengthened by prolonged suppuration of the buboes, the occurrence of parotitis, or of abscesses in the skin or more deeply situated. Carbuncles, pneumonia, a persistent typhoid condition, mental disturbance, some form of paralysis, or an actual relapse of the disease, may also occur. Irregular attacks of fever, due perhaps to blood-poisoning, suppuration of the middle ear, and deafness are occasional sequelæ to the disease.

Plague may occur twice, thrice, or even more often in the same person.

Forms of Plague.—Different forms of plague have been recognised according to the severity of the disease (pestis major and minor), its existence as a sporadic or epidemic malady, or the prevalence of certain symptoms (bubonic or hæmorrhagic plague, etc.). The mild form of plague which often accompanies or follows the severer disease is often attended by such slight symptoms that the patient continues his ordinary habits of life, and is not even confined to the house (ambulatory form of plague). The prostration, pyrexia, glandular swelling, etc., may be absent, or but little pronounced, and the infection in these cases is so feeble that it is not generally admitted to exist, the disease being apparently never conveyed to another country from such cases. Weakness and exhaustion may again be the only recognised symptom of the disease. The existence of pulmonary hæmorrhage has in some cases given such a special character to the plague that it has been looked upon as a different disease. Thus in India, this symptom was so frequent (at Ahmedabad in 1820, and at Pali in 1836) that the name of Indian or Pali plague was given to the complaint. Such hæmorrhage is, however, not infrequent in epidemics of plague (Black death, 1348; Tripoli, 1874; Assyria, 1874; Irak-Arabi, 1875–77, etc.), and although cases in which hæmoptysis occurs are usually fatal before the appearance of buboes, viz. on the second day of the attack, the bubonic and hæmorrhagic forms may be mixed or merge into each other.[1]

Duration.—From a few hours to thirty days or more.

Morbid Anatomy.—In this disease the cutaneous surface may be so discoloured by petechiæ as to have a dark livid hue, giving a

[1] *Vide* F. Forbes, *Trans. of Bombay Med. and Phys. Soc.* vol. ii. p. 15; Maclean, *India Journ. of Med. and Phys. Science*, vol. i. p. 619.

blackened appearance to the corpse, which, as already observed, must have caused the name of "Black death" to be given to the plague. The enlargement of the glands may be noticeable after death, and putrefaction rapidly occurs when the disease has been soon fatal. Brown sordes may be seen upon the lips and teeth.

The cerebral membranes show increased vascularity, the venous sinuses being engorged with blood, the dura mater ecchymosed, while extravasation of blood or effusion of serous fluid may have occurred into the cavity of the arachnoid, or beneath that membrane. Similar changes take place in the membranes of the spinal cord. The vascularity of the brain is also increased, an unusual number of red points being seen upon section of the white matter, while the lateral ventricles may contain an increased quantity of serous fluid. The brain has been observed to be softened in some cases, but its consistence is usually normal. No change has been noted in the consistence of the spinal cord.

An effusion of serum or blood may be found in the pleural cavity, while the mucous membrane of the bronchial tubes has often a bright red hue, although no evidence of bronchitis existed during life. Hypostatic congestion is not unfrequently found to exist in the posterior part of the lungs.

A similar effusion may be found in the pericardial cavity, in which from half a pint to a pint of fluid may be found. The heart, distended by blood or coagula, is said to be much increased in size, principally on account of dilatation of the cavities upon the right side, which are distended by dark blood in which yellow or red coagula float. The heart may attain a very large size, and rupture of the right auricle is said to have occurred. The muscular tissue of the heart is at times softened. The cavities upon the left side of the heart are usually vacant. There is general venous enlargement, and the veins opening into the right side of the heart (the venæ cavæ) and jugular veins are said to attain at times the size of the small intestine. The arteries are unaltered in size, and usually empty. Not only may effusion of blood take place into the serous cavities, but also into the viscera, mucous membranes, and subcutaneous cellular tissue.

The mucous membrane of the pharynx and œsophagus is injected, and that of the stomach is apt to be softened when the disease has been of long duration, or may be ulcerated. The mucous membrane of the small intestine is also injected, and may present ulcerations. Peyer's patches are unaltered. Ulcerations may occur upon the ileo-cæcal valve, extending to the appendix vermiformis. The colon

is usually narrowed, the large intestine presenting otherwise no appreciable change.

The liver and spleen are enlarged, at times it is said to more than twice their normal size. The kidneys may be surrounded by extravasated blood, which may be also effused into the tissue of the organ; while coagula may be found in the pelvis, ureter, or bladder. The lymphatic glands may be much swollen, chains of enlarged glands being often formed by the cervical, mediastinal, axillary, and bronchial glands in the neck and chest, and by the inguinal, external iliac, sacral, and lumbar glands in the thigh and abdomen. The mesenteric glands may be also enlarged. The glands may be found to be engorged with blood, indurated, softened, or perhaps decomposed. Nothing abnormal has been found in the thoracic duct.

Small abscesses may be found in any of the viscera.

Etiology.—*Predisposing Causes.*—1. *Soil.*—The geological character of the soil has no influence upon the occurrence of plague. The disease may appear upon a dry soil, one which is saturated with moisture, or upon ground which is frozen and covered by snow.

2. *Elevation of the Ground.*—Plague may occur in valleys situated little above the sea level, or at an altitude of 5000, 7000, or even 10,000 feet.

3. *Climate. Season of the Year.*—There is little doubt that the climate and season have a special influence upon the onset of plague. Thus in Egypt the disease was almost invariably most severe during the spring (February to June), in Turkey in Asia at Aleppo during the summer (July, August), at Smyrna and Trebizonde in the spring and summer (February to August), in Turkey in Europe in the summer (June to October), etc. These and other similar facts, and the rarity of its occurrence within the tropic of Cancer (lat. 23° 30′), indicate that a moderate amount of heat (60° to 85° F.) is favourable to its occurrence, while a very high or low temperature usually prevent its appearance. At the same time it may prevail during the severest cold of winter, as on the Volga (1878–79) and in Moscow (1771); as also in extreme heat, as in Smyrna (1735), Malta (1812), and in India (Kumaon, 1850).

4. *Social Position.*—Poverty is the principal predisposing cause of the disease, being associated as it is but too frequently with want and filth. From its prevalence among the poorer part of the population the great plague of London in 1665 was termed the "Poor's Plague," and the disease has always been found to be most destructive to those who suffer from the miseries of poverty. It would seem that dirt, specially organic dirt, such as fæcal matter, decomposing urine, and other forms of decaying animal matter, supply

whatever is necessary for the development of the poisonous element to which plague is due. The disease is rare among the better classes of society, and its gradual disappearance from Europe is in all probability mainly due to increase of cleanliness, and the improved habits which result from attention to public and private hygiene.

5. *Sex.*—The female is slightly more liable to suffer than the male sex.

6. *Age.*—Plague is said to occur most frequently between the ages of 10 and 20, then between 20 and 30, then from 30 to 40, and, lastly, between 40 and 50. It rarely occurs after the age of 50. At the same time, the age which principally suffers varies in different epidemics, so that this statement is not always true. The fœtus may be affected, and swelling of the glands may occur before birth.

Exciting Causes.—1. *Contagion.* — That plague is a contagious disease is so certain, that it is unnecessary to discuss this point, although until quite recent years many competent observers have denied this to be the case. The virus derived from the body or secretions of the patient exists in the air, which, however, readily destroys it. It attaches itself to the house in which the patient lives, where it remains after his departure, and may be conveyed by healthy persons, clothes,[1] merchandise, etc., to other parts, although such transference of the disease has never been undoubtedly known to occur over long distances measured by miles. Infection from the dead bodies of those dying from the plague is possible, but rarely occurs.

Origin and Foci of Plague.—The fact that plague occurs repeatedly in some districts of Africa and Asia, has led to the supposition that it is endemic in these localities. Such are the part of Tripoli adjoining the town of Benghazi, the district of Assir on the western coast of Arabia, and in Asia the countries of Mesopotamia, Persia,

[1] The plague which occurred at Eyam in Derbyshire in 1665 is supposed to have been conveyed to a *tailor* in that village from London, where the plague was then raging, through the medium of materials relating to his trade. This rests upon the authority of Dr. Mead, who states (R. Mead's *Medical Works*, vol. ii. p. 89) that the servant who opened the box containing these materials, while drying them at the fire, was "seized with the plague and died," and that the same misfortune extended itself to the rest of the family except the *tailor's* wife, who alone survived. The house in which this occurred, known afterwards as the "Plague House," belonged at this time to a *miner* named Edward Cooper, whilst the first death which occurred from the plague was that of George Vicars. It is probable therefore that the *tailor* was merely lodging in the house, and that Dr. Mead should have said that all the family died except the *miner's* wife, which the register shows to have been the case, Mrs. Cooper alone surviving. As Dr. Mead derived his information solely from the son of the clergyman who was at Eyam at that time, errors may possibly exist in his account. *Vide Hist. and Antiq. of Eyam*, by W. Wood, 6th ed. pp. 71, 72.

Hindustan (specially the southern slopes of the Himalayas), and Southern China (province of Yunnan and Pakhoi).

More than one explanation has been given of the recurrence of plague from time to time in these places from which it has to all appearance been completely absent during the intervening years. The first explanation, and probably the correct one, is that sporadic cases of little or no severity, and unobserved, continue to occur in these districts, and that the contagium thus passes from one patient to another without producing any serious outbreak of the disease. From time to time, owing to some unknown cause, the infective element becomes more virulent, and a severe epidemic occurs. The second explanation is that the disease is autochthonous owing to the existence of the poisonous element in the soil, water, or other element of the locality affected. By some it is thought probable that the soil is the habitat of the virus, perhaps introduced in the bodies of those who have previously died of the complaint, and the death of animals which live in or on the ground (rats, snakes, etc.), as is said to have occurred in India and China at the outbreak of an epidemic, is supposed to confirm this supposition. Disturbance of the soil has again been said to produce plague, as it does intermittent fever, as if in both these diseases it formed the breeding place of the virus. Whatever the first origin of plague may have been, the idea that it now arises from a spontaneous or independent cause is quite untenable, and it can scarcely be doubted that in the same way as many other specific diseases it invariably has a parasitic origin.

Diagnosis.—The diagnosis of plague is facilitated by the fact that it usually occurs as an epidemic disease, but, notwithstanding this, in almost every outbreak the true nature of the disease has been at first unknown. There is naturally great hesitation in announcing the existence of such a fatal and contagious disease, and plague may therefore prevail in a district for some length of time before being recognised in its true character. The frontal headache, the tongue covered with a thick white fur, the appearance of the face, the staggering gait, the stupor, delirium, and coma, may exist in other affections in which typhoid symptoms occur. The glandular swellings are the most characteristic sign, and when this symptom is associated with severe headache, fever, and prostration, and the disease is epidemic, its real nature is more easily recognised.

Differential Diagnosis.—Typhus and intermittent fever are specially distinguished from plague by the absence of glandular swellings, which rarely complicate those diseases. Scrofula and syphilis, in both of which the glands enlarge, are not epidemic diseases.

In dengue the glands may be slightly enlarged in the groins, axillæ, and neck, but the initial eruption which is erythematous, the locality of the pains which is not that of the glands, the absence of delirium (except in children), and the terminal eruption, resembling that of measles or scarlatina, enable this disease to be recognised.

Prognosis.—The prognosis of plague is most unfavourable, this disease being fatal to by far the greater number (70–90 per cent.) of those whom it attacks. The most favourable signs are the absence of fear and alarm, the urine having a healthy character, the existence of constipation, often profuse sweating, and the appearance of buboes having a tendency to suppurate upon the third or fourth day. On the other hand, there is less hope of recovery if great terror exists, or the vomiting or diarrhœa are profuse, if the buboes become smaller or disappear, or if they develop in the cervical region or iliac fossa. Carbuncles on the back of the neck, or along the vertebral column, delirium, convulsions, abundant hæmorrhages, cyanosis, petechiæ, or suppression of urine, are unfavourable symptoms. Should the patient survive the seventh day recovery is probable, though death may still occur owing to some complication.

Treatment.—Numerous remedies have been employed in the treatment of plague, but none have been found which can arrest the disease or check its progress. Invocations to the stars, amulets, deer's horns, the skin of snakes, human urine, friction with olive oil, bleeding, scarification, purgatives, sudorifics, opium, alcohol, ammonia, mercury, carbolic acid, chlorine water, coffee, quinine, and many other drugs, have been shown by experience to be useless. Locally the application of caustics to the buboes, or incision before suppuration has occurred, have been found of no service. The symptoms can alone be alleviated by the treatment. Thus high fever should be treated by quinine or other antipyretic, and vomiting, diarrhœa, and hæmorrhage in the same way as when they occur in other diseases, while the external treatment should be as far as possible antiseptic. Free ventilation and fresh air are of great importance in the treatment of this disease.

Prophylaxis.—The prophylactic measures which are advisable when an epidemic of plague occurs are of different kinds. Quarantine, which undoubtedly prevents the importation of plague by arresting communication with the country where plague exists, is undoubtedly effective, and the lazaretto furnishes the most successful means of arresting the extension of the disease. Notwithstanding this fact, the requirements of commerce prevent quarantine establishments from being brought into use to more than a moderate extent, though some quarantine must be imposed upon foreign

arrivals from infected places, owing to the fact that other nations refuse to trade with a country unless this is done, and the disease as far as possible prevented in this way from entering the land.

Medical inspection of the vessels which reach our harbours, the isolation of any cases of plague which exist in them, and disinfection of the ship, are also necessary as preventative measures.

The personal disinfection of the patient and his surroundings should be effected as in the case of other contagious diseases. A separate room or suite of rooms should be occupied, if possible on the top floor, well ventilated, and from which all unnecessary articles of furniture have been removed. The persons who visit the patient should not sit upon the bed, or put themselves in closer contact with him than is necessary. The duration of their visit should be short, and not exceed a few minutes if possible, experience having shown that prolonged exposure to contaminated air is specially dangerous. A sheet should hang outside the door which is kept moist by means of a solution of carbolic acid or other antiseptic. The excreta must be carefully disinfected, and all articles used for conveying food should be placed in boiling water before being removed from the room. After recovery and before leaving the house the patient should be carefully washed with antiseptic soap, and upon doing so should wear clothes which have not been in the room which he was occupying. The bedding must be taken to pieces and exposed in a hot air chamber to a temperature of from 250° to 300° F., while the walls and ceiling of the room should be scraped, and repapered or whitewashed, while the furniture and the floor should be washed with some disinfectant and then with soap and water. The air of the room in which micro-organisms may float should be disinfected by means of the fumes of sulphur or chlorine gas, and the windows and door of the room should finally be left open during several days.

The following books and papers refer to plague, some of which have been already mentioned:—

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Haeser, Lehrbuch der Geschichte der Medicin und epidemischen Krankheiten, Jena, 1875–82, vol. iii. 97. Noah Webster, A Brief History of Epidemic and Pestilential Diseases, Hartford, 1799. Edward Bascome, A History of Epidemic Pestilences from the Earliest Ages, London, 1851. The four latter books include other forms of epidemic disease.

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Excellent articles have been written on the plague in the *Encyclopædia Britannica*, 1885, vol. xix., art. "Plague," by Dr. J. F. Payne; in the *Dictionnaire Encyclopédique des Sciences Médicales*, 1887, art. "Peste," by J. Mahé; and by A. Hirsch in the *Handbook of Geographical and Historical Pathology*, New Sydenham Society, 1883, vol. i. p. 494. In these reference is made to numerous other writings upon the subject of plague.

CHAPTER X.

CHOLERA.

BY C. N. MACNAMARA, F.R.C.S.

History of Asiatic Cholera.

We learn, from the description given by Sanskrit authors writing some 400 B.C., that Asiatic cholera existed in India in their day. They have described the symptoms of the disease, which are precisely similar to those met with at the present time. In spite, however, of repeated inroads of warlike races from the north-west into India, cholera does not appear to have spread with them beyond the confines of that country. Probably few of these conquering races overran Lower Bengal, which we may therefore suppose was, as it now is, the home of cholera. As we pass from the study of these ancient records to comparatively recent history, we learn that, in the year 1438, Ahmed Shah was compelled to raise the siege of Mando, in Mehwah, in consequence of an outbreak of a disease which is said to have destroyed some thousands of his troops in the course of a few days; the disease is described as "*waba*," a word still employed to designate Asiatic cholera. In 1490 Vasco de Gama visited the Malabar coast, and has left us a full account of the symptoms of cholera as it affected the natives and his own sailors. For centuries past, in the Gangetic Delta, the "Oolee Beebe," or Goddess of Cholera, has been worshipped; and temples still exist in that part of India containing emblems of this goddess.

In 1781–82 cholera was very deadly in Calcutta, and the Government reported to the home authorities that it had "pursued its course northwards." During this year epidemic cholera visited Ganjam; it attacked a division of our troops passing through that district, and in the course of a few days no less than 1143 sepoys, out of a force of 5000 men, were down with cholera. Colonel

Pearce remarks that "death raged in the camp with horrors not to be described, and all expected to be devoured by this pestilence."

In 1782–83 the whole of Madras and Ceylon were under the influence of Asiatic cholera; at the same time the disease broke out in Burma; and we hear of it in the north of India at Hurdwar, where it was reported to have destroyed 20,000 pilgrims. It seems, therefore, certain that within twenty years of the Battle of Plassey, imperfect as our knowledge then was of many parts of India, that a vast area of the country was under the influence of a virulent form of Asiatic cholera. In fact, from the year 1438 to 1817 we have references, by sixty-six independent observers, to the occurrence of Asiatic cholera in India; and of these authorities ten refer to epidemic outbursts of the disease.[1]

Asiatic Cholera of 1817–23.

This outburst of the disease seems to have commenced in August 1817 in Jessore. The Medical Board of Calcutta, however, asserted "that it was the usual epidemic of this period of the year," and was always present in Calcutta. Before the end of October cholera appeared throughout the whole of Lower Bengal. The Marquis of Hastings was then in Bundelkund with his army, and he notes in his diary the following facts:—"13th November 1817.—The dreadful epidemic, which has been causing such ravages in Calcutta and the southern provinces, has broken out in camp. It is a species of cholera morbus. It has gradually ascended the River Ganges to Patna, Ghazeepore, Benares, and Cawnpore. There is an opinion that the water of the tanks, the only water which we have at this place, may be unwholesome, and add to the disease; therefore I march to-morrow, so as to make the Pohooj river, though I must provide carriage for 1000 sick. November 15th.—We crossed the Pohooj this morning. The march was terrible, for the number of poor creatures falling under the sudden attack of this dreadful infliction, and from the quantities of bodies of those who died in the waggons, and were necessarily put out to make room for such as might be saved by the conveyance. It is ascertained that 500 have died since yesterday. 16th November.—This day has exhibited an abatement of the contagion. 17th.—Still more improvement. 19th.—We marched fifteen miles to the banks of the Bitwah, a clear broad stream with lofty banks. 21st.—There is an unquestionable diminution of the disease. 22nd.—No one can comprehend my sensation on hearing laughter in our camp this morning, after

[1] *History of Asiatic Cholera*, by C. N. Macnamara, p. 22.

witnessing the dismay and melancholy which have lately visited my soldiers."[1]

During the cold season of 1817–18 cholera subsided throughout the greater part of the tract of country it had visited in the hot season and rains; but in April and May 1818 it burst out with renewed violence, and advanced to Delhi and all the intervening country. Westward, it extended along the valley of the Nerbudda and Tapti rivers, and appeared at Poonah in July, and at Bombay in August 1818. Eighteen months later we hear of it in the Punjab.

Cholera was very prevalent along the whole of the western coast of India. In 1819–20 Captain Thomson was despatched from Bombay to the assistance of the ruler of Oman, and in the following year another force landed from Bombay at Sur, in Arabia. Shortly after the landing of this expedition, cholera broke out in Oman. The Arabian historian states that "this year (A.D. 1820–21) a plague first broke out in Oman; it attacks a man's abdomen, and matter is ejected from the mouth and anus till he dies; great numbers fell victims to it in Oman; it prevailed also in India, in Sind, and in Mekrain." In these few words we have the record of the introduction by our troops of Asiatic cholera into a country situated beyond the confines of India. Cholera spread during 1821 to Muscat, and in that and the following year over the whole of Persia and into Asiatic Turkey. In 1823 it appeared at Tiflis, and extended to Astrakhan (Sept. 22nd). The Russian Government put forth all their strength to check the diffusion of the disease into the neighbouring provinces from Astrakhan. It is impossible to determine how far the severe quarantine imposed by Russia against the importation of the disease into her territories saved its inhabitants at this time; but it is certain that, as cholera appeared and spread through Arabia on the landing of our troops from cholera-stricken Bombay, so certain is it that the disease did not penetrate into Russia, guarded by severe quarantine regulations.

Turning to the opposite coast of India, we have a repetition of the same story. Cholera, in a virulent form, existed along the coast from Calcutta to Madras and Ceylon.[1] During the latter part of 1819 the disease spread in Burma, and in 1820 in Siam, which was absolutely devastated by cholera; it broke out at the same time in Malacca, into which place it was "said to have been brought by a vessel from the coast; the fact is undeniable that the disease occurred shortly after some persons, ill of the complaint, had been landed from such a vessel."[2] Penang and Singapore were affected,

[1] *A Treatise on Epidemic Cholera*, by F. Corbyn. Calcutta, 1832.
[2] *Transactions of the Medical and Physical Society of Calcutta*, 1842.

but cholera broke out in neither of these islands until it had previously become planted in the neighbouring mainland, with which there was constant intercourse.

M. Huc, who was in China at this time, states that this malady, "formerly unknown," made its appearance in 1820, when it committed frightful devastations in the Celestial Empire. This statement corroborates the testimony of the Arabian historian, and points to the fact that the countries to the east, as well as those to the west, of India, first suffered from Asiatic cholera during the epidemic which originated in Hindustan in 1817–18, and was carried by English vessels to Arabia, China, and other countries. Before leaving the history of this outburst of cholera, it is well to refer to the appearance of this disease in the island of Mauritius in 1819. From the journal of the surgeon in charge of H.M. ship *Topaze*, we learn that she sailed from Trincomali, where cholera prevailed, on the 9th of October, and immediately after sailing cholera broke out on board, and four men out of seventeen attacked by the disease died. The ship arrived in Mauritius on the 29th of October, thirty-six men were taken on shore and placed in the military hospital at Port Louis; four of them died from cholera. On the 17th of November cholera, which had never before visited the island, burst out among its inhabitants, and spread in all directions. A Government Commission was appointed to report on the circumstances of the disease, and its members arrived at the unanimous opinion "that the disease was contagious, and was imported into Mauritius."

The Governor of Bourbon (now Réunion) was convinced that the disease was contagious, and he therefore took active measures to prevent its being carried from Mauritius into that island. In spite of his efforts, a boat from Bourbon communicated with a vessel from Port Louis, and within a few days cholera broke out in Bourbon, but did not spread, the French Governor adopting stringent measures to isolate the sick from the people surrounding them. The disease, therefore, appears to have been stayed in its progress into Russia and also in Bourbon, during this epidemic, by means of quarantine.

The Indo-European Cholera of 1826–34.

Asiatic cholera gradually died out in the countries over which it had extended in 1817–22, and in Bengal little was heard of it throughout the years 1823–24–25, except in its endemic area. But during the year 1826 cholera was again on the increase in Lower Bengal, especially in the Presidency Circle; it extended to Dinapur and throughout the Benares and Cawnpore Divisions. The

stations on the right bank of the Jumna, viz. Delhi, Meerut, and Agra, were slightly affected; during the cold season the disease was comparatively quiescent. In May 1827 cholera, in an epidemic form, occurred with violence throughout the region affected in the previous twelve months; it was especially severe in the North-Western Provinces, and spread to Ajmere and Jeypur. The Punjab and some of the Himalayan hill stations were likewise affected. From the returns of our European and native troops we learn that a recurrence of cholera took place throughout India in 1828. And in 1829 Lieutenant Conolly, who was at Herat, reported that the disease during that year was extremely violent throughout the kingdom of Cabul, and extended through Khorasan to the Persian capital. In Teheran it destroyed a vast number of the inhabitants. Lieutenant Conolly also stated that cholera had spread to Bokhara and to Khiva, with terrible consequences to the inhabitants of those countries. So that Asiatic cholera, during 1829, occupied the whole extent of country, west and north-west, from Cabul, through which the trade routes from India pass to reach the great Russian marts of Orenburg and Nijni-Novgorod.

On the 26th August 1829 the disease broke out at the Russian frontier trading town of Orenburg; it was then stated to have been carried to that place by caravans arriving from Bokhara; it rapidly extended to various places in the province of Orenburg, but almost disappeared in the winter of 1829–30.

In the meantime Europe was threatened by an invasion of cholera *viâ* Persia. It broke out with great virulence throughout that country in the spring of 1830, and appeared at Tabriz and Resht on the Caspian. In July it spread to Tiflis, and soon afterwards throughout Astrakhan; and here the stream of cholera which entered Russia in 1829 from Bokhara and Khiva seemed to have united with that passing to the north-west through Persia.

In August 1830 cholera appeared in the great annual gathering of Nijni-Novgorod; it reached Moscow in September, and speedily passed on to the western provinces of Russia, where, in consequence of the war going on between Poland and Russia, it was disseminated quickly through both armies and communicated to the inhabitants of the former country.

The cholera epidemic of 1830 was in full force again in the spring of 1831, when it spread throughout Russia and passed thence to Sweden; on the 3rd of August it reached Vienna and Berlin, France remaining free of the disease until the following year. About the end of October cholera appeared at Sunderland, and was supposed to have been imported from Hamburg; the persons

first affected resided on the quay, and had constant intercourse with the shipping. The epidemic broke out soon afterwards at Newcastle and Gateshead; it appeared at Edinburgh about the 22nd January 1832, and in London in the month of February. The disease was most deadly throughout almost the whole of Europe during the year 1832–33.

On the 8th of June 1832 cholera appeared in the cities of Quebec and Montreal in the following circumstances:—The *Carrick*, in April, sailed from Dublin, then affected by cholera, with 173 emigrants on board. The disease appeared among these people soon after leaving Cork, and forty-two of the emigrants on board died before the 3rd of June, when the vessel arrived at Quebec. The emigrants were landed at Grosse Island, a few miles from Quebec but no effective measures were taken to prevent intercourse between them and the city. The first cases of Asiatic cholera ever seen in Canada appeared in Quebec on the 6th, 7th, and 8th of June. The disease passed from Quebec into the United States, reaching New York and Philadelphia in July; it spread over the greater part of America before the end of the year.

With regard to this epidemic, never were greater efforts made by the combined Governments of Europe to exclude an invading disease from their territories by means of quarantine regulations. In Spain infringement of these rules was punishable by death. But land quarantine utterly failed in its object.

In France it was stated that of 55,000 persons affected with cholera, only 164 had been employed on duties which obliged them to be near the sick. On the other hand, there were numerous instances of the following kind, given by persons who had no possible motive for stating what was untrue on the subject. At Moor-Monkton, six miles from York, John Barnes, a labourer, died from cholera on the 28th of December; the disease did not exist within thirty miles of his residence. Barnes' wife and two other persons who visited the sick man were seized with cholera. The son of the deceased then arrived; it appears that he had been apprenticed to his uncle, a shoemaker at Leeds, and that his aunt died of cholera fifteen days before; her effects were sent to the Barnes' to be washed. The trunk containing the contaminated things had been opened by Barnes in the evening, and the next day he was taken ill with cholera and died.

The history of cholera in India, from 1830 to 1845, though presenting features of much interest, need not detain us, so that we may proceed to refer to the second epidemic visitation of cholera, which extended from the East, through Europe, to America.

Asiatic Cholera of 1841–46.

On referring to the returns of the Medical Board in Calcutta, we find that, with regard to Singapore, Penang, and Malacca, from 1827 to 1840, not a single death from cholera had occurred, either among the troops or convicts, at any one of these places. The Madras records confirm the fact that epidemic cholera was unknown in the Straits throughout the thirteen years above referred to. Cholera was very deadly in Bengal and Madras during the year 1840; and it was at this time the Government of India had collected a considerable body of troops in Calcutta and Madras to embark for service in China. The vessels constituting this expedition, starting from Calcutta and Madras, met at Singapore in April 1840, and the superintending surgeon of the Straits, residing in Malacca, reported to the Calcutta Medical Board "the remarkable circumstance that epidemic cholera broke out, early in the year, along the seashore towns bordering the Straits, and slowly advanced to Malacca." On the 4th of July our troops arrived in the harbour of Chusan. Dr. Francis states that our soldiers had hardly landed before twenty cases of cholera occurred among them, and it spread to the inhabitants of the island. He states in his report, written in July 1840, "that, from inquiries made from the missionaries and inhabitants, he assured himself the disease did not exist in the island before the arrival of our troops."[1]

From July till the end of the year, constant supplies of men and materials of war arrived in China from cholera-stricken Bengal and Madras; "the disease was most malignant" among our sepoys at Ningpo in August. Dr. Bryson reported that cholera broke out on our vessels in the Wongoo and Nanking expeditions, and that "this was quite a new feature in the medical history of our fleet in China seas." During the following years, 1841 to 1843, epidemic cholera spread from the coast over the greater part of China, committing devastation and terror among the inhabitants, which, an eye-witness observes, the "pen of a Defoe would fail to describe."

The late Professor E. Parkes was at this time in Burma with his regiment, and reported that "early in 1842 cholera appeared in the *north* of Burma, and, passing in a southerly direction, committed great ravages at Ava; it passed down towards Rangoon, pursuing the course of the Irawada." This invasion of cholera into Burma evidently extended from China along the trade routes into the valley of the Irawada.

The first envoy from Kashgar who had visited India came to

[1] *A Treatise on Asiatic Cholera*, by C. N. Macnamara, p. 141.

meet the Viceroy in Calcutta about the year 1869; he was under charge of Dr. Cayley (now of Netley), then the British representative at Leh. On questioning the envoy as to the existence of cholera in Chinese Tartary, he informed us "that the disease was almost unknown in Yarkand, but that in the year 1844 cholera came from the side of China; that during the summer it attacked all the places on or near the main line of traffic from China; that in Kashgar, Yarkand, Khokan, and Bokhara it killed thousands of people; it lasted for a few weeks in each place, and the people died by hundreds every day; and that such a disease, so far as he knew, had only once before visited Khokan and Yarkand, some fifteen years previously (p. 356), when it lasted for a year, and came from the East. At the time of the epidemic of 1844 there was free traffic between China and Turkestan; every year several thousand horse-loads of tea passed from China through Kashgar to Bokhara." It is evident, therefore, that Asiatic cholera was imported into China by our troops, arriving from Calcutta and Madras in 1840–41; it extended throughout that empire and spread through Burma, and also to the north of the Himalayas to Yarkand, and so to Bokhara by the trade route. Our agent on the frontier of Cabul reported that "cholera travelled south from the countries north of the Hindoo Koosh in 1844, and that in Balkh alone 25,000 people were said to have been destroyed by it; it extended throughout Afghanistan and passed to Peshawar and the Punjab." In Lahore it carried off 22,000 people. So that the Indo-China cholera of 1840 spread not only south into Burma, but also into Afghanistan, and from thence to the Punjab and the North-Western Provinces, along the Indus to Sind and Karachi.

General Ferrier endeavoured to travel from Teheran into the Punjab to join the army under Runjeet Sing, and when to the north of Herat, in June 1845, he first met with cholera; he states that, although he had for some years resided at Bagdad, and passed through Teheran to Meshed and Herat, he had heard or seen nothing of the disease, which, as he observes, was passing from the east, that is, from Balkh and other parts of Afghanistan, westwards. Cholera quickly spread to Teheran and Tabriz, and reached as far north as the town of Derbend, on the Caspian.

At the time this disease was advancing from Bokhara and Persia towards Europe, it had, as in 1821, been imported into Arabia from Bombay; it was extremely severe in Western India in 1845–46, and broke out in May 1846 at Aden, Mocha, and Jeddah; "it even penetrated as far as Oman, and spread up the Tigris and Euphrates, so that Europe, as in the epidemic of 1826–32,

was penetrated by two streams of an invading cholera, one passing from China, the other from Bombay, both of which had originated in Bengal in 1840–42.

At its appointed season, April (1847), cholera broke out again at Orenburg, and soon afterwards at Nijni-Novgorod and Moscow. By October it had covered the greater part of Russia and Turkey, and in the following year almost the whole of Europe. In Edinburgh it first appeared on the 4th of October 1848. Three pilots from Newhaven went to the Isle of May to look out for a vessel; one of them went on board a ship from Cronstadt, where cholera existed; they both remained on the ship at Leith; one of them died of cholera the following Sunday. During the next eight days several cases occurred among relations and immediate neighbours of the pilot who died, and these were the first cases in Scotland; it spread throughout the country. In like manner cholera broke out at Hull and Sunderland immediately after the arrival of vessels, cases of the disease having occurred on board during the passage from Hamburg. The first case reported in Ireland was that of a man who arrived at Belfast from Edinburgh on the 2nd of December; he was sent to the workhouse, and cholera spread to its inmates, and from thence to the town.

America had been free from cholera for thirteen years, but on the 9th of November 1848 a vessel, carrying emigrants from parts of Europe affected with cholera, sailed from Havre. On the 24th of the month, being very cold, there was a general overhauling of baggage to obtain warm clothing. The next day cholera broke out on board. One of the men employed to remove the sick from the ship to a quarantine hospital at New York died of cholera on the following day. In a dirty German lodging-house three persons who escaped from quarantine were attacked with cholera; but the disease did not spread, the weather having suddenly become exceedingly cold, with sharp frost.

At New Orleans, cholera was imported at the same time by German emigrants, who sailed in a vessel from Havre on the 3rd of November. Cases of cholera were brought into the hospital in New Orleans from this vessel; the disease rapidly spread through the city. "The weather was very warm and damp, the streets were as muddy as possible, and heavy fog overhung the city." The disease extended from town to town along the Mississippi, and covered the United States and Canada during the following year.

This, probably the most fatal epidemic of cholera that has ever spread from India throughout Europe, Asia, and America, had almost completely died out by the end of 1851. In England alone it

killed no less than 53,293 men, women, and children. In a report drawn up by the Royal College of Physicians, London, regarding this epidemic, they observe: "It is not probable that in the case of cholera the influence of water will ever be shown to consist in its serving as a vehicle for the poison generated in the bodies of those who had suffered from the disease."[1]

The Indo-European Epidemic of 1848–53.

India was, unless in its endemic area, free of cholera in 1847, the epidemic of the previous years having died out; but in 1848 the accounts of cholera at Dinapore, Cawnpore, and Agra were bad. Throughout 1849 we have a repetition of the old story—cholera of a virulent type in the Mirzapore district, "the devastation of the disease was terribly fearful; the inhabitants fled for refuge to other districts, forsaking their habitations, cattle, and property." Central India was subjected to a most awful visitation of cholera, and Dr. Leith reported that "cholera made its approach to Bombay from the east towards the end of the rains of 1849, in spite of the south-west monsoon which blew during the same week with a force varying from 1½ to 5 lbs., or on an average of 3½ lbs. on the square foot, which is equivalent to a velocity of more than 15 miles an hour, in a direction contrary to that in which the cholera advanced."[2]

Throughout 1850 the disease was reproduced over the Bengal, Madras, and the Bombay Presidencies; in the town of Bombay, for instance, the number of deaths from this disease in 1848 had amounted to 69, in 1850 to 4729, and in 1851 to 4020. In 1851 cholera broke out in the port of Bassorah, having direct communication with Bombay, and from thence extended by two routes, the one towards the west to Hillah, the other east to Mohammera. That pilgrims spread the disease seems more than probable, for it was reported that from the 1st of December 1849 to 1st December 1850 there passed through the station of Khaneguin 52,053 pilgrims, with 64,138 beasts of burden, 4504 muleteers, and 2837 *loads of human corpses*, which, giving three dead bodies to a load, would amount to 8000 *corpses*, to be buried at Kerbela." In 1852 cholera spread from Persia into Russia, along the borders of the Caspian Sea, when, as in previous years, it met with a stream of the disease extending from the Punjab through Cabul and Meshed to Teheran. In 1853 the disease broke out over the whole of Persia and Asiatic Turkey, Russia, and the north of Europe. It appeared

[1] Dr. Baly and Sir W. Gull's *Report*, p. 213.
[2] *Bombay Cholera Report*, 1891, p. 9.

in England first at Gateshead, afterwards in London, during the months of August and September. New York and New Orleans were both visited by cholera in 1853; Mexico and the whole of the West India Islands were during the year under the influence of the disease. In 1854 Asia, Europe, and America were covered by the epidemic which had originated in Bengal during the year 1848-49, and travelled thence to Bombay and to Cabul, and so to Persia and westward, as in previous years, in its progress causing untold misery and death. The disease died out of Europe and America in 1856-57.

In 1854 the death-rate from cholera in the district of St. Ann's, Golden Square, was at the rate of 128 for every 10,000 persons, the general death-rate of the metropolis being only 60 to the same number. It seems that at 40 Broad Street (St. Ann's), a child, having been ill for three or four days, died from cholera on the 2nd September, her fæces having during her illness been emptied into a cesspool only 3 feet from the well supplying the pump used by the people in Broad Street. The contents of this cesspool drained into the well, as was subsequently discovered. On the night of the 31st August, cholera broke out among the inhabitants of Broad Street, the greater number of cases occurring on the 1st of September. "Nearly all the persons who had the malady during the first outbreak drank of the water from the Broad Street pump, and very few who drank of this water during these days escaped having cholera." [1]

In the weekly return of deaths for 9th September the following was recorded as occurring in the Hampstead district: "At West End, on the 2nd September, the widow of a percussion-cap maker, aged 59 years; diarrhœa two hours, cholera epidemic sixteen hours." Dr. Snow was informed by this person's son that she had formerly resided in Broad Street, but had not been in the neighbourhood for many months. A cart went from Broad Street to West End every day, taking out among other things a large bottle of water filled from the pump in Broad Street, the lady in question preferring this to any other water. The bottle of water was carried out to Hampstead as usual on Thursday the 31st of August, and she drank some of it that evening, and more on the following day. She was seized with cholera on the evening of the latter day, and died on Saturday. A niece, who was on a visit to this lady, also drank this water; she returned to her residence, in a high and healthy part of Islington, was attacked with cholera, and died. There was no cholera at the time either at the West End or in the neighbourhood. Besides these two persons, only one servant partook of the water at Hampstead, West End, and she did not suffer, or only to a slight extent.

With regard to this remarkable case, the committee appointed by the Board of Health to conduct a scientific inquiry into the circumstances of the epidemic of 1854, remark: "It seems probable that the water of this well did really act as a vehicle of choleraic infection; but (assuming the absence of fallacy in the case) this probability might easily be admitted, without its therefrom resulting

[1] *Report of Committee for Scientific Inquiries into the Cholera Epidemic of* 1854, p. 50.

that infection depended on the specific material alleged (by Dr. Snow). The water was undeniably impure with organic contamination; and we have already argued that if, at the time of epidemic invasion, there be operating in the air some influence which converts putrefiable impurities into a specific poison, the water of the locality, in proportion as it contains such impurities, would probably be liable to similar poisonous conversion."[1] The committee argue: If, therefore, the specific action caused by the Broad Street pump water be admitted as a fact, it did not arise from its containing the dejecta of those who had died of cholera, but because it had participated in the atmospheric infection of the district.

The committee had by no means neglected to investigate the state of the water consumed by the inhabitants of London during the epidemic of 1854. It was shown that in two large sections of the population of London, "breathing the same atmosphere, comprising the same classes, and averaging the same habits of life," in the one class the death-rate from cholera was 130, and in the other only 37 per 10,000. The former, however, were supplied with water "impregnated with the sewage of the metropolis," and the latter with pure water. From a comparison of the mortality in the epidemic of 1849 with that of 1854, it appears that the population to which the Lambeth Company's water was distributed in the latter epidemic suffered a mortality less than one-third of that sustained by the drinkers of the water purveyed by the Southwark and Vauxhall Company, and that the tenantry using the purer water supplied by the Lambeth Company in 1853-54 suffered not a third as much as the same tenantry had done in 1848–49, when the water was impure. On the other hand, the Southwark and Vauxhall Company, which pumped an impure water in 1848–49, pumped even worse water in 1853–54, and the mortality in the houses supplied was 10 per cent. higher.[2] It was a consideration of facts such as these that led to the passing of "The Metropolitan Water Supply Act" in 1852, and which came into operation from the 31st of August 1855. Previous to this time the several water companies took water for domestic use from tidal and impure portions of the Thames, simply straining it through wire screens on its way to open settling tanks, from which it was pumped for use. By the Act of 1852 no company could draw water from the Thames below Teddington Lock; all reservoirs in which water was stored were to be roofed in, and on its way from any such reservoir to the mains or pipes for delivery, the water was to be filtered. The only

[1] *Report of Committee for Scientific Inquiries into the Cholera Epidemic of* 1854, p. 52.
[2] E. H. Greenhow, "On Cholera," *Medico-Chirurgical Review*, 1857, p. 71.

exception to this rule was in the case of water which was pumped from wells into a covered reservoir or aqueduct, without exposure to the atmosphere.

The practical effect of this Act was to induce sand-filtration, and the supply of water from covered water tanks to the entire metropolis.

In 1854 cholera was not unknown in the county of Bedford, where it broke out in the village of Ridgmount, and eleven cases occurred, all of which were fatal. It was ascertained that the first case occurred in a man whose son had died of cholera in London a week or two before, and whose clothes were sent down to the country. The poor man unwrapped the bundle of clothes himself; he was seized with the disease and died. His case was the nucleus of the others.[1] An instance of a similar nature was reported from Munich; the first case of cholera was generated in the house of a labourer, one of whose daughters was in service in Munich. The latter sent her parents clothes belonging to a family, some members of which had just died of cholera; these old clothes were at once appropriated and worn; three days afterwards (21st September 1854) the father and mother were seized with cholera and died; on the 22nd and 25th other members of the family took the disease.

Dr. Lebert reports the case of a man who was attacked with cholera, having worn the clothes of a person who had died of the disease two months previously. Other instances are cited of persons having been seized after sleeping in beds previously occupied by cholera patients, but which in the meantime had been kept locked up.[2]

In the neighbourhood of Tavatola the wearing apparel, the bed-clothes, and the mattress of a cholera patient were washed at one of the fountains of the city; the waste pipe of the fountain being broken, this foul water became mixed with the drinking water. On the following day sixty persons in this small and up to that time healthy district were stricken with the malady.[3]

Cholera in India from 1858. Punjab Epidemic of 1861.

Our information regarding cholera in Bengal in 1857–58 is necessarily defective on account of the disturbed state of the country; we know, however, that the disease appeared among our troops engaged against the mutinous native army before Delhi, from June to September 1857; and some sixteen cases and eleven deaths

[1] *On Malaria and Miasmata*, p. 140, by J. Barker. London, 1863.
[2] *Constantinople Cholera Conference*, p. 95. Calcutta, 1868.
[3] B. W. Richardson, "On Cholera," *Transactions of Epidemiological Society*, vol. ii. p. 427.

took place among the prisoners in the Delhi jail in 1858.[1] The Lucknow garrison also suffered to a slight extent from cholera in 1857.[2]

I would only refer, however, in detail to the history of one outbreak of the disease which occurred during this year in connection with its appearance at Kalka.

Kalka is a small village at the foot of the Himalayas, on the road leading to the Simla, Sabhathu, Dugshai, and Kassowli sanitaria. This village was infected with cholera when our troops marched through it, suffering greatly from thirst induced by the sudden change from the cool climate of the hills to the scorching plains. H.M.'s 75th passed through Kalka on the evening of the 12th May; soldiers were attacked with cholera of the most virulent type at Umballa on the 15th May, probably within sixty hours after they had left Kalka. The 1st Bengal Fusiliers passed through Kalka about midnight on the 13th May; two men were attacked with cholera at 10 P.M. of the 14th, or twenty-two hours after leaving Kalka. The 2nd Fusiliers passed through the place on the morning of the 15th May, and suffered greatly from heat and thirst; the soldiers drank copiously of every stagnant pool they passed; on their arrival at Umballa severe cholera broke out. These regiments were perfectly healthy before leaving their stations in the hills.

The water of Kalka is obtained from a spring, which discharges into a square open masonry reservoir. The reservoir is enclosed on three sides by a wall, the fourth side of the enclosure is formed by the hill at the back. At one side of the reservoir, and enclosed with the surrounding wall, there is a piece of ground about 6 by 5 feet, on which the people stand when drawing water. I found this piece of ground polluted with fæcal matter and other impurities, which were being slowly washed by the dripping water into the reservoir. The spring itself was situated in a watercourse below the level of the village, and its produce was liable to admixture with the drainage and sewage of the village. This is the only spring in the neighbourhood, and people come to it to draw water from a distance of a mile or more. The circumstances stated show that every facility existed for the infection of the troops through the drinking water.[3]

During the year 1859 cholera was widely disseminated throughout Bengal and to the north-west as far as Cawnpore; but it did not extend further in this direction, although it spread directly west and south, attacking the whole peninsula, as Mr. Cornish remarks, from Karachi to Cape Comorin; it was very severe in Bombay during the month of May; at the same time its progress through the Deccan was as steady as in 1818–19, and also along the eastern and western ghauts; but it covered this vast space of territory in a very much shorter time than it had done thirty years previously.[4]

[1] *Punjab Selections*, vol. v. No. 8, p. 39.
[2] Greenhow, "Notes during the Siege of Lucknow," *Indian Annals*, vol. x. p. 336.
[3] De Renzy's *Report for* 1869, p. 23.
[4] Cornish, *On Cholera in Southern India*, p. 16. Madras, 1871.

Throughout the year 1860 cholera prevailed in Bengal proper, and, in fact, from Assam to Oude, and from the seashore of the Bay of Bengal away into Central India and Bombay, as well as over a very large portion of Madras; it also spread far up the Himalayas to Darjiling. The number of deaths from cholera among the prisoners confined in the jails eastward of Cawnpore was nearly four times as numerous as in 1859. Among the small European force at Morar there were 89 deaths from cholera; at Jhansi, 13; at Saugor, 4; Nagode, 15; and Jubalpur, 5. The prisoners in those stations, together with the civil population, suffered in an equal degree. So that we have evidence of cholera of a virulent type, and extensive power of diffusion, having been raging over the enormous tract of country above indicated during the early part of the year 1860. And, as we might have expected, it spread at the same time to Agra.

Dr. Walker informs us that cholera appeared in the city in July, and extended slowly among the natives: "Rain had fallen sufficient to soak the ground, and even to be lying in pools in many places."[1] On the 10th of August cholera broke out among the prisoners at Agra; at the same time there were twenty-four casualties from this disease among the European troops at Muttra.

Dr. Walker was of opinion that the epidemic influence appeared to have been on this occasion more widely spread, and more generally fatal than in former years,—a statement which is borne out by his figures; and from the history of the disease in 1859, together with its terrible virulence over the whole of Bengal proper, the Central Provinces, and as far to the north-west as Muttra, we should naturally have expected to have heard of its immediate dissemination throughout the North-Western Provinces and the Punjab with the setting in of the rains of 1860.

I would call the reader's attention, however, to the exceptional state of these provinces. Throughout this year they were subjected to unprecedented drought, which converted an enormous tract of otherwise fertile country into a desert; this arid waste was bounded to the east by the Agra district, to the west by Sirhind, to the north by Dehra, and to the south by Goorgaon; and although cholera spread from Bengal and Central India up to the very borders of these districts, it extended in no single instance that we know of into this barren area, which constituted what Colonel Baird Smith describes as the famine tract of 1860–61, and which is very clearly defined in Chart No. II. of his report on the subject; section 28 refers to the mortality attributable to the famine; but among the

[1] *Prison Returns for the North-Western Provinces for* 1860, pp. 123, 124.

diseases which affected the starving people, he makes no allusion to cholera. Throughout the whole of the jails in the famine districts not one instance of cholera occurred; and there were only one or two cases among the troops, and some of these are described as "cholera biliosa." Dr. B. Smith, who at this time was in medical charge of the civil station at Delhi, and therefore in the midst of all the suffering, expressly states that the first instance of cholera he heard of among the famine-stricken people was in May 1861. Smallpox and fever raged among the starving people; but in all the reports and returns I have read on the subject, the existence of cholera is never once alluded to during the year 1860 in the famine-stricken districts.

I am justified, therefore, in asserting that in 1859 a very considerable portion of the peninsula of India was under the influence of epidemic cholera; throughout the following year it was reproduced over the whole of this area, with the exception of that part of the country which had been affected by a grievous drought, and thereby converted into a sandy desert.

It is almost impossible for those who have not experienced the influence of the annual rains in the north-west of India to realise the condition of the country after such a year as 1860. Colonel Baird Smith says: "It would be difficult to exaggerate its forlorn dreariness: it seemed denuded of its inhabitants; that monotonous brown tint of the untilled soil suppressed everything else. It was only by some inquiry it could be learnt that, even in this great waste, there was cultivation in plots round the villages, and round the wells remote from villages." This is truly a faithful picture of a desert; and in this country cholera never gained a footing during the continuance of the drought, although the disease raged around it.

It is not my province now to discuss the bearing of this fact on the etiology of cholera; but when taken in conjunction with the circumstances I have related as occurring in the north-west in 1831, it is very significant, and well worthy of our serious consideration.

This remark is strengthened by what follows, for no sooner had the rains of 1861 set in over the famine-stricken districts, than cholera burst out among its inhabitants with terrible virulence. I shall now proceed to demonstrate this fact from documentary evidence bearing on the subject.[1]

In 1861 cholera was reproduced over the whole of Bengal proper; out of fifty-two jails in this province only eleven escaped the disease. In May the convicts and European troops at Cawnpore and Allahabad were attacked with

[1] *A Treatise on Asiatic Cholera*, by C. N. Macnamara.

cholera, and in July those at Gwalior and Jubalpur suffered very severely. It is evident, therefore, that cholera was reproduced over the area in which it was principally prevalent during the previous years; and this remark is applicable to the circumstances of the inhabitants of the Agra and Muttra districts, where, as I previously stated, cholera had been very severe in 1860.

Dr. Smith informs us that "the first heavy fall of rain at Delhi in 1861 occurred on the 31st of May," at which time cholera appeared amongst the inhabitants of the southern portion of the Goorgaon district, extending from the direction of the Bhurtpore territories. The disease rapidly spread among the famine-stricken people of the district, and reached Delhi on the 11th of June.[1] "It is important to note that at this time there was not a single case of diarrhœa in the jail, and the amount of sickness in the station generally seemed to be below the usual average; it is well known that many cholera epidemics are preceded, introduced as it were, by the occurrence of a great amount of generally prevailing diarrhœa; it was not so in this instance as regards the city of Delhi."

It appears that among the prisoners one patient only sank from the effects of the disease within four hours of the time he was attacked by it; of the others, none died under an illness of less than nine hours.

In H.M.'s 82nd Regiment, eighty men were seized with cholera, and fifty-seven of these were in a state of collapse on admission into hospital. "One man had no vomiting or purging throughout; but after death the intestines were found filled with rice-water fluid."

The men of H.M.'s 107th Regiment, and the prisoners, were attacked by cholera on the same day at Agra (7th July); the disease spread with alarming rapidity both among the Europeans and natives; indeed, it had existed among the latter from the middle of June.[2] Dr. Banister writes from Muttra that the disease appeared among the Europeans on the 14th July: "The weather being very close, the rain was unusually heavy, the wind continuing to blow from the east."

On the 26th of July the station of Meerut was visited by a heavy fall of rain, which flooded part of the prison enclosure. On the 27th the first case of cholera occurred among the prisoners, and the malady did not cease until the 24th of August; during this time there had been 664 admissions, and 344 deaths from the disease. Cholera had, however, appeared among the Europeans in this station since the 30th of June.

On the 6th of August and five following days, fifteen cases of cholera, all of which were fatal, occurred among the European troops at Mian-Mir; by the 14th of the month all the regiments in cantonments were more or less affected; and Dr. W. A. Green (Inspector-General of Indian Medical Service) strenuously urged their removal into camp. Unfortunately, it was found impracticable to march the whole of the troops out of cantonments at once—the country for miles round being under water; besides, the commissariat was unprovided with carriage and other appurtenances for a camp of the kind at a moment's call. The military authorities, however, did all in their power to forward Dr. Green's views, and on the 15th of August three companies of H.M.'s 51st Regiment left the station; at the same time the artillery marched to Shahdera, on the banks of the Ravi, 10 miles to the north of Mian-Mir. Subsequently, one single case of cholera occurred among the men of this party; but among the troops who

[1] *Punjab Selections*, vol. v. No. 8, p. 44, "Cholera in the Delhi Division."

[2] Murray's *Report on the Epidemic Cholera at Agra*, 1861, p. 3.

remained in the station there were no less than 457 cases, and 261 deaths from the disease within the following ten days. In fact, after the 15th of August, cholera increased with such fearful rapidity, that the soldiers in a few days were panic-stricken and hopeless.[1]

In one regiment, out of a total strength of 1002 men, 863 were employed as hospital orderlies; and of these no less than 428 were seized with cholera. In the other European regiment at Mian-Mir, of 203 cases of cholera, 137 occurred among hospital orderlies. It was not found possible, however, to determine if these hospital orderlies were more liable to be attacked than men who had not been exposed to cholera in the hospital, because all the men in the station had been on duty of this kind at one time or another. On the other hand, we cannot overlook the fact that the medical officers and the whole of the medical establishment, together with the native servants, almost entirely escaped the influence of the disease, although prostrated by the fearfully harassing nature of their duties. And what is more remarkable, when it was discovered that the European orderlies were unable to work any longer, some thirty Sikhs of the 31st Regiment were daily sent to take their place in the European hospitals, but not a single instance of cholera occurred among these natives.

The Commission, a member of the Indian Civil Service being their president, was appointed by the Government of India to report on this terrible outbreak of cholera, and they stated: "That the mere contamination of drinking water may cause disease, but will not cause cholera,"—an idea which, under the leading of the gentleman appointed as head of this Commission, the Government of India adhered to for a long time.

The Epidemic Cholera of 1863-64.

The reports from the various jails throughout Lower Bengal for the year 1863, demonstrate the fact that this province was subjected to a most severe visitation of epidemic cholera during the early months of the year. These returns show cholera swarming up the Ganges valley in the east, and covering the Behar provinces; in which province, we are told, not a village of any note escaped. The disease spread to the Central Provinces and into Oude; it broke out in an epidemic form among the pilgrims assembled at Punderpur in November 1863, and was by them communicated to the natives of Poonah. Cholera was reproduced over the whole of India during the year 1864; the western coast was specially affected; in the town of Bombay the deaths amounted to 4588. Early in the following season the disease appeared at Karachi, Sind, and along the shores of the Persian Gulf, also in Yeman, Mocha, and Aden. Cholera was carried to Mecca by pilgrims; after a fall of rain it burst out among the assembled multitude, and some 30,000 of them are said to have fallen victims to the disease.

[1] *Report on Cholera of* 1861. Published by the Government of India. Appendix, p. xvii.

At this time there was not a trace of epidemic cholera in Egypt, Europe, or America.

On the 19th of May a ship, bringing 1500 pilgrims from Jeddah (Mecca), arrived at Suez. Many of the passengers had died of cholera during the voyage; the captain of the vessel and his wife were attacked by the disease on the 21st of May at Suez. The pilgrims were at once forwarded from Suez to Alexandria by rail, and on the 22nd of May the first case of cholera was noticed in a body of these people on their way to the port. Numerous pilgrims from Mecca followed by this route, and many of them embarked at Alexandria in vessels provided to carry them to their homes.

Cases of cholera occurred in Alexandria early in June, and the disease spread rapidly over Egypt, destroying 60,000 of its inhabitants during the following three months. The panic was great, and people fled from Egypt to Constantinople, and to various places along the Mediterranean coast. At the same time, the disease was extending through Persia to the north and north-west, and so along the shores of the Black Sea, and up the Danube into Europe. In July, Turkey, Russia, Italy, France, Spain, and Portugal were under the influence of cholera. The disease reached England in September *viâ* Southampton, the port at which the Peninsular and Oriental vessels from Alexandria landed goods and passengers. Cholera was imported into America by the steamer *Atlantic*, from Havre; she carried 540 steerage passengers, who had come from Paris and other infected places. So that the Indian cholera of 1863–64 was, in the course of twelve months, disseminated from that country over the greater part of the civilised world by means of persons from infected areas passing in steamers and railroads to localities previously free of the disease; as in the case of the pilgrims from Mecca already referred to, or as in the following instance:[1]—

The wife of a German workman left Odessa on the 16th of August for Altenburg, in Saxony, with her child suffering from diarrhœa. On the 24th, after a journey of nine days, she arrived at her father's house. On the 27th, the child's diarrhœa having become considerably aggravated, the mother called in Dr. Genitz. The mother was in perfect health on that day. At nine o'clock on the evening of that same day she fell ill of cholera, and sank under the attack on the morning of the 29th. At eight o'clock on the evening of the same day her sister-in-law, who lived in the house, was attacked in her turn, and died on the 30th. The house in which these two women died became the primary focus of infection,

[1] *History of Asiatic Cholera*, by C. N. Macnamara.

whence the disease spread throughout the town. The family of a workman who had died at Altenburg on the 18th of September imported the disease into Werdau. The dwelling occupied by this family became the starting-point of an epidemic which carried off 2 per cent. of the population of the town.[1]

The following details are from a despatch to the Foreign Office from H.M.'s Minister at Florence, dated October 26, 1866. He reports: "The outbreak of cholera at Palermo has taken place under circumstances which merit some remark. Last year cholera prevailed at Naples, Malta, Marseilles, and other places where the intercourse with Sicily is most frequent; but a quarantine of the most stringent, not to say exaggerated, form was enforced throughout the island, and the disease never appeared there. The same thing occurred again this summer; and notwithstanding the prevalence of cholera at Marseilles, Genoa, and Naples, it did not make its appearance in Sicily, where quarantine was as before rigidly enforced. Then came the disturbance at Palermo, and the necessity of bringing troops at once from Naples, and of landing them without delay. In a few days it began to be whispered that cases of cholera had occurred among them, and shortly afterwards some of the townspeople were attacked, till, by last returns, above a hundred deaths had taken place within the twenty-four hours."

The first case of cholera reported in London in 1866 was traced by Mr. N. Radcliffe to a family at Bromley; the discharges passed by this patient were emptied into the River Lea, 600 yards below the open reservoirs at Old Ford.

This took place on the 27th June; the water from this reservoir was distributed to certain parts of London; early in July, out of every 10,000 of the persons who consumed this water, 27 were attacked with cholera; whereas in other parts of London only 5 in 10,000 persons were attacked by the disease.

The Cholera Epidemic of 1867 in (Hurdwar) India, and in Persia and Europe in 1868–70.

From the jail returns of Bengal for 1867, we find that no less than 368 per 1000 of the prisoners in Chyabapa, 132 per 1000 in Cuttack, and 100 per 1000 in Raipore, died from cholera; fearful as is this death-rate, it was probably trifling as compared with that of the surrounding population. The disease was very severe in the Central Provinces, and appeared among the people assembled at the Viceroy's Durbar at Agra. Hurdwar is a town situated on the

[1] *History of Asiatic Cholera*, by C. N. Macnamara.

banks of the Ganges in a gorge of the Sewalick hills, about 13 miles from where the river escapes from the Himalayas. Here pilgrims collect from all parts of India at a certain day of the year, to the number of about three millions; they camp on a space about 22 square miles in extent. The year 1867 was the year of the *Kumble mala*, which occurs every twelfth season, when the blessings derived from bathing in the Ganges are supposed to be unusually great, and Hindoos flock to Hurdwar in vast numbers from all parts of India.

The pilgrims began to pour into the camp from the 1st of April in vast numbers from the plains, and to settle themselves down in the blocks laid out for them. On the 3rd of April the fair may be said to have commenced, though dense living streams stretched backwards for a very long distance into the plains, and with a volume steadily increasing up to the auspicious bathing hour of noon on the 12th of April, continued to concentrate themselves in Hurdwar, and to pour out their multitudes on the encamping ground. It is important to notice here that, on the night of the 11th of April, a very heavy thunderstorm burst over this vast unsheltered multitude; the rain lasted the whole of the night and throughout the following day.

Those only who have been exposed to these hill storms in the tropics can realise what a night of misery these three million pilgrims must have passed on the open plain of Hurdwar, cold and drenched to the skin, the water running in streams off their half-naked bodies over the rocky ground into the river; and however perfect the conservancy may have been, this downfall of rain must inevitably have washed excrementitious matter from the latrines and surface soil into the Ganges during the night of the 11th of April.

With the exception of a case of cholera on the 9th, under the care of Dr. Kendall, the entire mass of pilgrims appears to have remained in good health up to the 12th of April, and I cannot do better than describe what then occurred in Mr. Cutcliffe's own words. He says, the bathing place of the pilgrims was a space 650 feet long by 30 feet wide, shut off from the rest of the Ganges by rails, which prevented the people from getting out into the river further than the limits of the space thus enclosed. Into this long, narrow enclosure, the pilgrims from all parts of the encampment crowded as closely as possible, from early morn (the rain still beating down over them) till sunset. The water within this space during the whole time was thick and dirty, partly from the ashes of the dead, brought by surviving relations to be deposited in the water of their river god, and partly from the washing of the clothes and

bodies of the bathers. Pilgrims at the bathing ghaut, after entering the stream, dip themselves under the water three times or more, and then drink of the holy water whilst saying their prayers. The drinking of the water is never omitted; and when two or more members of a family bathe together, each from his own hand gives to the other water to drink.

Observe what follows;—On the evening of the next day, 13th April, eight cases of cholera were received into one of the hospitals at Hurdwar. By the 15th the whole of this vast concourse of pilgrims had dispersed, and the encamping ground was again left a barren waste. Dr. J. Murray has given a detailed report of the events that occurred after the pilgrims left Hurdwar. He states that the immense crowd at Hurdwar having entirely dispersed on the 15th, the pilgrims passed chiefly on foot at about the rate of 15 miles a day. "The moving mass crowded the roads at Meerut in a continuous stream for nearly a week. This pilgrim stream carried cholera, which lined the road with victims, whose funeral pyres studded the surrounding fields, or whose corpses were thrown into the canal or collected by the police and buried. The disease was communicated to the neighbouring towns and villages, and the pilgrims carried it to their homes over the whole of Hindostan." Dr. Murray, in his able report, traces the appearance of cholera over districts, otherwise free of the disease, to the advent of bands of pilgrims; the date and particulars of each outbreak is given in detail.[1]

Cholera spread to Peshawar and to Cabul, from which country many pilgrims travelled from Hurdwar; it broke out with fearful violence in Cashmere, and at Teheran in the summer of 1867. It appeared in Meshed in July 1868; the place was then crowded with pilgrims, and with them it was dispersed over the whole of Persia and Asiatic Turkey. In the same circumstances Kiev, the "Jerusalem of Russia," when full of pilgrims was attacked with cholera (August 1869), and in the three succeeding years in Russia alone it destroyed no less than 241,808 people, and throughout Europe probably not less than one million human beings.

It was after this terrible visitation of cholera that the second International Sanitary Congress was opened at Vienna (*vide* Appendix), the first Congress having met at Constantinople in 1866. The Vienna delegates affirmed their belief that epidemic cholera invariably spread to Europe from India; that it is not likely to become localised in Europe or America; that it travels with man, and only to a limited extent by the atmosphere; and that it may be spread by means of drinking water, soiled linen, or merchandise

[1] *Report on the Hurdwar Cholera of* 1867.

coming from an infected locality; that the period of incubation of the disease did not last beyond a few days.

I may mention an instance of the disease, the first occurring in Calcutta, on the 25th December 1871, regarding which I published the following details in the *Indian Medical Gazette* for March 1872:—

Nos. 3, 4, and 5 Russell Street form as fine a block of three-storied houses as any in Calcutta; they stand well apart from one another, and are, in fact, nearer to a parallel row of houses overlooking the Chowringhee maidan than they are to one another. The locality is admirably drained, and supplied with water from the Calcutta municipal works. The three houses above referred to form a boarding establishment, presided over by a lady living in No. 5 Russell Street, and all the European lodgers on the premises are provided, not only with food and milk, but also with water, from a kitchen in No. 5; for it is remarkable that although the pipes from the municipal waterworks run along the side of the street, the water is not laid on to any of the three houses in which the cholera occurred, but the supply of drinking water is brought in bheesties, dirty leathern bags, and emptied in an open filter kept in No. 5 Russell Street, from which filter the drinking water for all the persons in this boarding establishment is drawn.

There has not been a single case of Asiatic cholera among the Europeans residing in the immediate neighbourhood of Russell Street within the past four years.

On the night of the 26th December, a gentleman who had lately been living at Bhowanipore among a community free of cholera, although the disease existed in the neighbourhood, was seized with cholera in No. 4 Russell Street; he passed rapidly into collapse, and died the following morning. The wife of this gentleman, and a Christian servant in their employ, who partook of the food and water[1] consumed by his master, were attacked with cholera on the 27th; they both recovered. At the same time a lady in No. 5 Russell Street was seized with cholera, and during the night of the 27th another lady residing in No. 3 Russell Street was attacked by the disease. Archdeacon Pratt was living in rooms in No. 5 Russell Street; he left the house on the night of the 25th, and travelled away some 300 miles by rail to Ghazeepore. He was there seized with cholera on the 27th, and died from the effects of the disease on the following day.

It appears, therefore, that of inmates of Nos. 3, 4, and 5 Russell Street, who were all in good health on the 25th of December, six were attacked by cholera within the next forty-eight hours. Since that time there has not been a single case of the disease in the neighbourhood. We cannot overlook the fact, that of the multitude of native servants on the premises, only one ate and drank the food and water consumed by Europeans, and he was attacked with symptoms of cholera. Further, the three houses in which the cholera occurred were respectively nearer to the houses overlooking the Chowringhee maidan (where no cholera occurred) than they were to one another.

We must bear in mind the fact that the one thing which the European inhabitants of Nos. 3, 4, and 5 Chowringhee had in common was the kitchen, from which they all received their food and water; and it is very

[1] It was proved by subsequent analysis that this was hydrant water, but containing just double the amount of organic matter existing in water drawn from stand pipes close by.

important to observe that the bheestie who carried the water (it may have been from a municipal stand pipe) in his *mussuck* to the common filter in No. 5 Russell Street, resided in a suburb of Calcutta called Bhowanipore; further, the milkman who supplied milk to the establishment (Nos. 3, 4, and 5 Russell Street) also lived in Bhowanipore, and we have since ascertained that within a stone's throw of the tank from which the milkman gets his water there is a large house in which no less than eight persons were attacked with cholera between the 18th and 23rd of December, and of these four died. Cholera was, in fact, very deadly in parts of Bhowanipore during the weeks previous to the 25th of December; and it is quite possible the infecting matter was introduced through the medium of milk, and distributed to the people residing in Nos. 3, 4, and 5 Russell Street on the 24th and 25th of December. It affected those susceptible to its influence within three days from the time it was swallowed.[1]

The Indo-European Cholera of 1879–83.

During the years 1876, 1877, and 1878 there were respectively 196,590, 155,305, 95,193 deaths from cholera in the Bengal Presidency; in 1879 the mortality rose to 136,363, so that in these four years in one of the three Presidencies of India no less than 583,451 of its inhabitants were carried off by this terrible malady. In the Bombay Presidency, from 1875 to 1878, about 183,667 people died from cholera; while in 1883 no fewer than 37,954 deaths occurred in Bombay from this disease.

The epidemic of 1879 was remarkable for its terrible virulence in the North-West Provinces and in the Punjab, and appears to have been disseminated by pilgrims returning from Hurdwar in the same way as the cholera of 1867 had been spread by them over that part of India. As I before remarked, every twelfth year is supposed to be exceptionally sacred at Hurdwar, and as in 1867 pilgrims collected to the number of about three million to bathe on a certain day in the Ganges, so in 1879 another *Kumble mala* was held in this place. Cholera broke out among this vast assemblage of pilgrims about the 12th of April, and from that time it spread over the north-west of India. On the 15th of April, after the return of pilgrims from Hurdwar by rail, the first cases of cholera were observed at Lahore; the disease broke out among our troops in the Peshawar valley; 284 died from it in the month of June; it spread throughout Afghanistan during the year 1879, and also into Cashmere.

Cholera was very destructive over the greater part of Oude and the North-West Provinces in 1880.

In 1881 the disease killed 16,694 persons in the Bombay Presidency, and in 1883 no less than 37,954 people, as we have said, died in this province from cholera.

[1] *History of Asiatic Cholera*, by C. N. Macnamara, p. 383.

I have not attempted to describe the course of this epidemic over India, for year by year a vast area of that country had gradually been opened up by railways, facilitating the rapid transit of people through extensive tracts of country; and so complicating the circumstances of the distribution of cholera, as to render the outlines of its progress from one part of the country to another almost impossible to follow. We must, however, refer to the spread of the disease beyond India during the year 1881–82. In the month of November 1881 the feast of Kurban Bairam was held at Mecca; some 60,000 pilgrims assembled in and around the holy city on this occasion. Many of these people arrived at the port of Jeddah early in September to be ready for the festival. In July the *Columbria* left Bombay with 650 pilgrims on board bound for Jeddah. This vessel made no less than five trips with pilgrims between April and November, carrying in all 3566 pilgrims from India to Arabia. In September the *Columbria* touched at Aden to unload a quantity of rice she had taken on board in Bombay (then under the influence of epidemic cholera); three of the natives employed to unload this rice were within a few hours after commencing their work attacked with cholera, and from their homes the disease spread to natives living in surrounding villages, and so throughout the Hedjaz. The *Columbria*, having discharged her cargo of rice, proceeded on her voyage to Jeddah, and landed her 650 pilgrims; they passed on to Mecca about the middle of August. Soon after these people arrived in Mecca, that is, on the 29th of August, cholera commenced among the pilgrims in the holy city, and it is believed killed some 6000 or 7000 of them. On the 27th of November a shipload of these cholera-stricken people arrived in Egypt, the vessel being at once subjected, together with the pilgrims, to strict quarantine regulations at El Wedj. Although the disease existed more or less constantly in this station, being frequently imported by fresh arrivals from Mecca, cholera did not spread to Egypt in 1881, some batches of pilgrims being detained four months in quarantine.

On the 25th of July 1882 the *Hesperia* entered the port of Aden, after leaving Bombay about the 12th, with 501 pilgrims and a cargo for England. At Aden the stoker of this vessel died of cholera. Three-fourths of the pilgrims on board came from Bokhara and Afghanistan, where cholera existed at the time of their departure. Several of them died of cholera on their journey, and the disease again broke out at Mecca in October. During this year a brigade of Indian troops landed in Egypt from Bombay, and took part in the occupation of that country by our forces, and the Egyptian quarantine regulations had to be relaxed in consequence of

the exigencies of our military operations. At the end of June 1883 cholera appeared at Damietta, and afterwards at Rosetta, Port Said, and Mansourah. During July it spread from these localities; at Cairo it was very fatal, and also in Alexandria. It is estimated that from the 22nd of June to the 1st of September 1883, cholera destroyed 50,000 people in Egypt. There can be little doubt that the Egyptian cholera of 1883 originated in the 1882 epidemic of India and Arabia. "The French delegates to the Sanitary Council had urged that pilgrims who reached Suez without previous quarantine should be isolated and kept under surveillance for three days; but, owing to the opposition of the English delegates, these measures were not enforced. The Council did not meet again, and so no protective system was adopted."

Dr. W. T. Simpson, the Officer of Health in Calcutta, has given an admirable account of this outbreak of cholera in Damietta and Egypt, and, after the most careful consideration, arrived at the conclusion that there was room to suspect the introduction of cholera into Damietta by pilgrims; but, as in India so in Egypt and Europe, in consequence of the vastly increased facilities given of late years to the transit of human beings and merchandise from the East, it is now extremely difficult to follow the course of the disease from one country to another.

About the end of June 1884 cholera broke out in Toulon, and soon afterwards at Marseilles. I cannot pretend to settle the question as to the exact route by which cholera reached these places. It is quite certain Egypt was under a widespread epidemic in 1883–84, and that France, which had before that year been free from Asiatic cholera, became affected by the disease, it may be directly from Africa, or, as some authorities hold, by vessels arriving at Toulon and Marseilles from Saigon; but it seems almost certain that during the summer of 1883 cholera was carried from Egypt to Marseilles. The disease spread over Southern France in 1884, and appeared in Genoa, Naples, Palermo, and various other places in Italy, where it remained until 1886–87.

As early as 1884, Spain was slightly affected with cholera, and in 1885 it broke out with considerable virulence over a portion of that country. The disease commenced in Spanish ports having direct communication with infected places in France, and from the seaboard extended inland; according to the best authorities it was disseminated principally by means of polluted drinking water.

On the 17th of February 1883 I addressed a letter to Earl Kimberley, then Secretary of State for India, asking him to grant me the services of an officer of the Indian Medical Department to

assist me in working out the bacteriology of cholera. Lord Kimberley on the 9th of April declined to grant my request. Fortunately, however, in the summer of 1883, Germany, France, and subsequently America sent men possessing the highest scientific knowledge to investigate the cause of cholera in Europe and Egypt. Among these, Dr. Robert Koch, the Director of the Pathological Laboratory of the Imperial Board of Health, Berlin, was commissioned by his Government to proceed to Alexandria, and subsequently to Calcutta, to investigate the nature and cause of Asiatic cholera. The French Government deputed experienced pathologists and bacteriologists to proceed to Egypt on the same errand. America sent Dr. E. O. Shakespeare, of Philadelphia, on a like mission to Spain, Italy, and to India. The report of this physician to his Government is one that does honour, not only to the gentleman who compiled it, but also to the United States, who have published his exhaustive and admirable work on cholera in Europe and India. The labours of Dr. Koch have justly received recognition by his own Government; and by men of science throughout the world their value are now fully acknowledged. But no sooner had his views become known in Europe, and been confirmed by many of the best authorities on the subject, than the Home Government sent a Commission, with Dr. Klein at its head, to Bengal, "for the purpose," as Dr. Shakespeare remarks, "of investigating the grounds of the opinions announced by the German Commission." Dr. Shakespeare adds: "After having spent some time in Calcutta and Bombay, the English Commission returned in due time to London, and published a report, in which they related observations and conclusions directly opposed to those of the German Commission. At the same time, they incidentally advanced statements concerning the dejecta of cholera patients which would seem to warrant the inference that they were imbued with a notion, entertained by some medical officers high in authority in India, and connected closely with the Government of India, that there is nothing specific or contagious about these dejecta, and that it is extremely doubtful if this disease is at all infectious or contagious in the ordinary acceptation of these terms."

The Indian Government, since the unfortunate Commission on the Mian-Mir cholera of 1861,[1] had adopted views regarding the preventive treatment of cholera which differed from those held on this subject by the medical and sanitary authorities of Europe and America. In this way it came to pass that when Asiatic cholera extended beyond India westward, England was accused of neglecting the proper precautions to prevent the disease spreading from its

[1] *Treatise on Asiatic Cholera*, by C. N. Macnamara, p. 194.

home. This idea was unfortunately fortified by some expressions of opinion used by Government on the subject. For instance, in 1883 the Sanitary Commissioner of Madras, in his annual report, recorded facts bearing on the spread of the disease in Southern India by pilgrims and by passengers travelling over our railroads. With reference to this communication, which was in accord with all that we learn from science and the history of cholera, the Government of India, on the 6th March 1883 (No. 83, Home Department), observes: "We are not called upon to enter into theoretical discussions as to the causation of disease, but there can be no doubt that the publication of such theories as are contained in this report is likely to prove most embarrassing, especially at the present time, when the International Sanitary Board (p. 57) at Constantinople and Alexandria not only accept these theories, but immediately proceed to put them in practice, to the great disadvantage of India." Expressions of this kind necessarily gave rise to the ideas I have mentioned regarding England's proceedings in this matter. So strong had this feeling grown in 1883, that the Foreign Office felt it necessary to publish a note to the French Government repudiating the idea that England was in 1881–84 "responsible for the outbreak of Asiatic cholera in Egypt and Europe." But so long as views such as those I have quoted find their way into Government despatches, we must remain under suspicion as to our motives and action in the preventive treatment of epidemic cholera.

Since the year 1869, Drs. T. R. Lewis and D. D. Cunningham have to a large extent led the Government of India in matters connected with the origin and spread of Asiatic cholera; these gentlemen in 1882 had expressed views on the subject precisely similar to those enunciated in the Government despatch of 1883, above quoted. Dr. T. R. Lewis, writing on the cholera which broke out in Aden in 1882, observes that "the influence of the promulgation of current theoretical views (see p. 373) has many disadvantages. What the essential cause of cholera may be is wholly unknown; but surely," he adds, "it is wiser frankly to avow our ignorance than to promulgate purely theoretical doctrines which tend to direct the attention of Government and individuals from the necessity of getting rid of known local causes of ill-health, and which, if carried to their logical conclusions, would seriously interfere with personal liberty, and prove very embarrassing to the commercial intercourse of nations."[1]

In this country, Sir Joseph Fayrer, Physician to the Secretary of State for India in Council, maintained similar ideas to those above

[1] *A Reprint of the principal Scientific Writings of the late T. R. Lewis, M.B.*, p. 327.

referred to regarding Asiatic cholera. In an address delivered in February 1886, Sir Joseph Fayrer "rejected the theory of contagion of cholera by personal intercourse, and therefore condemned in strong terms the inability of all coercive measures of quarantine and cordons." He observed "that the British and Indian Governments, who based their action in the matter on well ascertained facts, had wisely discarded all quarantine measures on both sea and land, and relied solely upon sanitary laws." [1]

In the *Lancet* for May 19, 1888, Sir Joseph Fayrer observes:—

> The cause of cholera is still unknown; but so much, however, has been learnt of its habits, that in Europe and India we have come to know that action based on any theory of contagion is as useless as it is unprofitable—that the rate and direction of an epidemic are not influenced by facilities of communication, or by the greatest streams of human traffic. Many circumstances attending the outbreak of the disease and the pathological conditions these develop seem opposed to a specific poison as being a cause of the disease.

Sir Joseph Fayrer remarks that "cordons and quarantine have utterly failed to prevent the spread of cholera, but, on the contrary, have done harm"; that unripe fruit, saline purgatives, fear, and anxiety are powerful predisposing causes; the former, he states, may "bring on the disease." Sir Joseph "demurs to a microbe being accepted as the solution of a problem as the cause of cholera." On the other hand, he remarks "that Dr. Bryden maintained that cholera was due to a miasm, and has a perennial abode in certain areas of India, and in other districts is renewed by invasion from these areas; that it is earth born and aerially conveyed."

Epidemic Cholera of 1891–92.

During the early part of 1891 cholera was very prevalent in parts of Lower Bengal. Surgeon-Captain D. G. Crawford (the *Indian Medical Gazette* for February and April 1892) has published an account of an epidemic that occurred in 1891 in the Purnia district, which is situated within the endemic area of cholera, between Nipal and the Ganges: it contains a population of about two million people. There was comparative freedom from cholera in this district during the year 1890. On the 8th of February 1891, a great bathing festival took place on the banks of the Ganges at Karagola. Surgeon Crawford writes that this festival (Suan) is said to occur only once in thirty years, and it took everybody by surprise. The railway authorities appear to have been unprepared for the enormous quantity of passenger traffic suddenly thrown on

[1] *Brit. Med. Journ.* p. 457, March 6, 1886.

their hands, and were unable to concentrate sufficient rolling-stock to carry pilgrims applying for transport, especially as this unexpected demand was made in more directions than one at the same time; crowds of pilgrims poured down the N.B.R. line to Sara, as well as along the Assam-Behar Railway to Manihair. Nor were any preparations made in this district, at least for dealing with the sickness that might be expected to occur among the assembled masses at Karagola. On the 8th of February 1891, cholera broke out among these pilgrims, and by the 12th about 60,000 of them had been despatched by train, some of them, it is reported, dying of cholera in the railway carriages. A great number of the pilgrims came from the hills, that is, from the lower ranges of the Himalayas. In March the disease had become general over almost the whole of Purnia, 2187 deaths being registered from this cause. In April the deaths from cholera reached 10,730; in May, 6668. Dr. Dawson Williams, in the *British Medical Journal* for September 17, 1892, states that towards the end of 1891 cholera was imported into the Peshawar district from Swat, and spread through the Khyber Pass to Cabul before the close of the year.

In 1892 our attention is fixed on another outbreak of cholera among pilgrims assembled at Hurdwar, which, as I have before stated, is situated near the exit of the Ganges from the Himalayas. From the *Indian Medical Gazette* for April 1892 we learn that in the year 1891, when the great fair was held at Hurdwar, at which an extraordinary number of pilgrims assembled, the Government had the satisfaction of recording the fact that no cholera had broken out either at Hurdwar or among the returning pilgrims. This fortunate result was attributed to the excellent, but costly, sanitary and other arrangements organised and carried out by the Government. "This year (1892), however, we are informed that with a smaller number of pilgrims cholera has broken out in a severe form at Hurdwar, necessitating the breaking up of the fair. It is reported that this year the sanitary and food arrangements were defective. The Punjab Government has taken steps to establish dispensaries at every large station on the N.W. Railway for the purpose of inspecting all special trains conveying pilgrims from Hurdwar back to their homes. Already, however, we hear that cholera has reached Saharanpur; and, if we may judge of the past history of the disease caused by returning pilgrims from Hurdwar, this is only the first stage of its journey, the first *act in the drama*." I have quoted this passage because it might seem that I was harping unnecessarily on the dissemination of cholera over the north-west of India by affected pilgrims.

The outbreak of cholera occurred among the pilgrims assembled at Hurdwar about the 22nd of March 1892; on the 25th of the month the Government issued orders preventing the railroads from taking any more pilgrims to Hurdwar, and those who had assembled there were dispersed as speedily as possible. This action of the Government was the subject of much comment in the native papers, but, as the *Pioneer* at the time observes, it is impossible to sympathise with "the complaints which reach us from Delhi and elsewhere regarding the action of the authorities in dispersing the pilgrims at Hurdwar when cholera appeared among them. It is argued that whereas the course taken was meant to stamp out the epidemic, it has had the opposite effect, returning pilgrims carrying the disease with them wherever they appear in large numbers. This is unfortunately the case, but it was inevitable. What the authorities really did was to minimise the local outbreak, to stop hundreds of thousands of persons congregating instead of only the tens of thousands who had already gathered when cholera of a most virulent type broke out. If one imagines the terrible results that would have followed had not the 70,000 pilgrims been hurried from the bathing-place, the action of the authorities will be justified a hundred times over. It has been proved again and again that pilgrims, imbued with the spirit of fanaticism and filled with that fatalistic belief so common among Eastern races, will incur every risk from pestilence sooner than abandon the religious object they have in view. Thus at Hurdwar several Hindus in the last stage of cholera were actually lifted from the sacred waters, only to die a few minutes later. Their companions resented all interference with their 'right' to bathe, and it was with the greatest difficulty that their dispersal was eventually effected. Had not the strongest measures been taken, there would have been an epidemic probably unequalled in the century. Less than 100,000 persons had assembled when the order was given to break up the fair, and we know what has followed. What would have been the return of mortality if 500,000 had made their way to Hurdwar? The latter figure is well within the mark, for in all probability the attendance this year would have been exceptionally large. Not all the appliances of sanitary science, and no amount of medical skill, can check an epidemic of true cholera when hundreds of thousands of people are camped together in a small space. There is nothing for it but to disperse the gathering as quickly as possible. It is the less of two evils, and the only question is when the order for the breaking up of the pilgrims' camp should be given."

From the 1st of April to the 7th there were twenty-four cases of cholera between Simla and Kalka. By the 20th of the month it had broken out at Mian-Mir; it prevailed to an alarming extent at Peshawar and in the surrounding country. Seven thousand deaths from cholera were reported in the Punjab since the second week of April. During the month of May cholera spread to Kashmir, and in Srinagar alone 5736 out of a population of 124,000 died from the disease.[1] Dr. R. Harvey, who was deputed by the Government of India to investigate the circumstances of this epidemic in Kashmir, reported "there can be no doubt the disease was imported" into the country. By the 11th of May cholera was "raging at Cabul"; the disease spread rapidly over Northern Afghanistan, and, in spite of the strict quarantine of forty days imposed on their frontiers by the Russian and Persian authorities, cholera reached Meshed by the 27th of May, 700 deaths occurring in that city every day. The Persian Government proceeded to establish *cordons sanitaires* round Meshed, to prevent pilgrims from moving from place to place. The town of Turbaty-Shan is described as presenting "a terrible picture. The streets of the place strewn with unburied corpses. The inhabitants flying in terror to the hills;" although the Russian authorities had sent an armed force to the Persian frontier to keep the disease out of their territory. Places westward of Meshed were speedily affected; the disease reached Astrabad, Yezd, and Resht, and by the 26th of June we hear of it at Baku and in various other towns on the shores of the Caspian. At the same time, the disease had broken out with great severity throughout Northern Khorassan and at both termini of the Transcaspian Railway—from Samarcand along the road to Taskend on the east, and from Urun-ada on the Caspian, and so by steamer to Baku, on the west. The Russian Government made strenuous efforts to prevent cholera spreading along the banks of the Volga; but it broke out in Astrachan, and by the 1st of July appeared at Isaritzin, far up the river, and by the 4th of the month at Saratov, some 500 versts up the Volga, and so it reached the famine-stricken districts of Russia. Cholera passed on to the University town of Kasan, and into the province of Orel. It appeared at Nijni-Novgorod on the 11th July. On the 13th of July, persons travelling from Moscow to St. Petersburg were stopped *en route* and sent back, as they were believed to be suffering from cholera. In Moscow it was officially notified on the 24th July, and in St. Petersburg on the 1st of August.

The disease extended over a great part of Russia during the months of August and September, destroying not less than 300,000

[1] Surgeon-Colonel Harvey, M.D., *Brit. Med. Journ.* August 13, 1892.

of her inhabitants during this period. Professor Virchow, who visited Russia at the time, on his return to Berlin reported "that the preventive measures taken by Russia against the dissemination of cholera over the country were simply magnificent. The best possible means of checking the disease were everywhere at the disposal of the local authorities." Virchow adds: "In some respects it is evident the Russians are ahead of us in Germany and Berlin. It is true the disease is raging in some districts of the Volga (September 8), and the arrangements are not so perfect there as they might be; but the disease there finds a favourable soil among the famine-stricken population, and Germany could do no more than the Russians have done." I can endorse the views here expressed by Virchow, for I visited Russia and the Volga not many years since, with the object of observing the condition of the people, and the preparations made by Russia with reference to the passage of cholera from India into Europe. There can be no doubt her sanitary authorities were aware of the danger, and, as far as practicable, were prepared for an invasion of cholera from the east, such as has this year overtaken their country.

There is a larger emigration of Russians, especially Jews, as well as Germans, from Hamburg than from any other port in the north of Europe. The sanitary condition of the city is most defective, and the water of the Elbe, into which the drains of Hamburg empty themselves, was delivered unfiltered for the use of the population. In these circumstances, Asiatic cholera broke out in the city about the 18th of August 1892, and continued until the end of October, during which time 17,972 persons were attacked by cholera, and 7610 of them died from the effects of the disease. Asiatic cholera appeared in a number of places in Holland, Belgium, and the north of Europe; it also broke out in Poland, Cracow, Buda-Pesth, Vienna, and other towns in the east of Europe; but, with the exception of Hamburg, no place beyond the confines of Russia was seriously visited by the disease. In all doubtful cases the nature of the affection was determined by the presence, or absence, of Koch's cholera bacillus in the discharges passed by the patient.

The news of the outbreak of cholera in Hamburg served to increase the vigilance of our Local Government Board. Dr. Thorne had for months carefully watched the progress of the cholera from Asia through Europe. Hamburg had always been a suspected place, owing to the large number of emigrants from Russia and Eastern Europe generally who annually arrived there *en route* to England and America. In anticipation of an outbreak of cholera in the great German port, medical men, by order of the Department, had, in the spring of 1892, been closely watching the ports on the eastern coast of England, with which Hamburg is in almost daily communication. As soon

as the Local Government Board received official notice that Asiatic cholera had actually broken out in Hamburg, the Board telegraphed the news to every medical officer of health at every port from the Tyne to the Solent. Three medical inspectors were also despatched without delay to the chief ports at which ships from Hamburg might be expected to arrive. The Board received telegrams from those gentlemen showing that they were actively at work in co-operation with the various port sanitary authorities, which bodies, in many cases, are constituted by the town councils. Orders were issued to all port sanitary authorities, all urban and rural sanitary authorities, officers of Customs, masters of ships, and other persons concerned, reminding them of the powers they possess, and the responsibilities attaching to them. Suspected persons could be detained, and, if released, they were obliged to give their names, addresses, and other particulars, so that their subsequent movements could be followed. As a matter of fact, every new arrival suspected of having been in any way in contact with cholera was followed to his destination and closely watched, with a view to prompt medical attention and isolation in the event of the disease developing itself. Dr. Thorne added that, short of absolute quarantine, which would practically mean the suspension of all commerce, every precaution had been taken to prevent the importation of the dreaded disease.

On the 25th of August the Hamburg steamer *Gemma* arrived off Gravesend with three suspicious cases of Asiatic cholera on board. The patients were at once removed to the floating hospital; two of them died the same evening of cholera. The next case occurred at Middlesborough on the 27th of August.

The steamship *Geona* arrived at Middlesborough from Hamburg, and reported a clean bill of health. She went into dock, and most of her hands were paid off and went to their respective homes. Later in the day a seaman named Goodlad, of Leith, who had steered the vessel into dock, became ill, and Dr. Malcolmson, of the port sanitary authority, was sent for. He found Goodlad suffering from the symptoms of cholera, and ordered the vessel into quarantine. Goodlad died on Saturday night. Dr. Thompson, local Government inspector, confirms the opinion that the case was one of cholera.

At Hull on the same day, August 27th,

"Two German firemen on board the steamer *Hamburg*, of and from Hamburg, which was lying in the Queen's Dock, Hull, at which port the vessel had arrived the previous day, were found to be unwell, and were examined by the medical officer of health (Dr. Mason), who pronounced them to be suffering from diarrhœa. Under the circumstances the authorities deemed it prudent to remove them to the Garrison Side Hospital, which is set apart for cases of infection, whither they were at once taken and isolated. The vessel having received free pratique, and there being no reason for her detention, she sailed the same night for Southampton. All vessels arriving at Hull were rigidly inspected, and closely watched both by the Customs officials and those of the port sanitary authority, no person being allowed to land until they had satisfied the authorities they were in good health." [1]

Since the 25th of August until the end of October 1892, twenty-

[1] *The Standard*, August 27th, 1892.

nine cases of Asiatic cholera have been brought to our shores in vessels arriving from infected places on the Continent. Not one single case of cholera had arisen out of these twenty-nine importations of the disease; nor had a single case of Asiatic cholera besides those imported occurred in England.

On the news arriving in America of the outbreak of cholera in Europe, President Harrison prohibited any further emigration into that country from infected places on the Continent; in the same way old rags and clothes were excluded from the United States. A considerable number of vessels, however, arrived at various American ports, principally at New York, from Hamburg, with cholera cases on board. The passengers and crew of some of these vessels had suffered severely from cholera during the passage across the Atlantic. All such vessels were placed in strict quarantine on their arrival in American waters. No cases of cholera occurred throughout the United States during the year 1892, except in the instances of persons who, as in England, had embarked from cholera infected places on the continent of Europe.

In short, Asiatic cholera of a virulent type broke out within its endemic area in March 1891, and was widely disseminated over districts to the north of the Ganges, and into the Himalayas. During March 1892 the disease appeared among the pilgrims assembled at Hurdwar, and, on their being dispersed, was rapidly carried over the Punjab into Kashmir and Cabul. During the month of May, cholera spread into Persia and Samarcand, and in June it had reached various towns bordering the Caspian Sea. The disease was officially notified on June 18th in Astrakhan, and somewhat later the cities along the banks of the Volga were under the influence of the epidemic; the disease had spread before the 6th of July into the provinces of Saratov and Kostroma, some 700 miles up the Volga, in the centre of Russia in Europe; and in the south to Shusha, near the Turkish frontier, and so to Tiflis. In August cholera had broken out in the north of Europe, and had been carried westward as far as New York by the 3rd of September. This rapid dissemination of the disease may be accounted for by the fact that pilgrims leaving Hurdwar about the 23rd of March would have reached our Afghan frontier by rail in the course of a few days; and the communication between Samarcand and the Caspian was equally expeditious, and from Europe to America; so that in this, as in former epidemic outbursts of the disease, cholera has not outstripped human intercourse in the rapidity of its progress over the countries in which it appeared.

We cannot conclude the history of cholera during the year

1892 without reference to the outbreak of the disease in Paris, which has been in existence since the 10th of May; it was reported on that day that a man had died in the Necker Hospital of cholera nostras. Since then a number of cholerine and cholera cases have taken place at Puteaux and other suburbs of Paris. In July the disease was spreading at St. Denis and Aubervilliers, about seven deaths per diem having taken place from cholera in these localities. The season had been a peculiarly hot and dry one in Paris, and the river water was drunk by no small portion of the suburban population. The disease, however, was considered sufficiently serious by our Local Government Board to call for a General Order on the subject, dated July 12, 1892. In this Order it is stated, that "Whereas cases of an infectious disease, alleged to be cholera, now exist in certain parts of France, and it is expedient that regulations should be made, as hereinafter mentioned, with reference to ships having on board bales of rags from that country,"—and then follow regulations preventing the import of rags from France into this country. It seems that the cholera of Paris in the early part of 1892 was not an invading cholera, such as that which, during the spring, spread from India to Europe, destroying thousands of people within a few months in its progress. I am disposed to believe that the Paris disease is a revival of the South of Europe epidemic of 1884–87, unless cholera had been imported in the meantime directly from Tonquin into Paris. There seems to be no doubt in the minds of a Commission of Spanish bacteriologists and physicians sent to examine into the matter, that the epidemic disease existing since May in Paris is Asiatic cholera. But it has comparatively feeble infective properties, and in this respect is not at all like the epidemic which in the course of six months has spread from India to Europe and America, and which is likely to overlap the disease which during the summer has been prevalent in France.

Cholera appeared at Havre on the 5th July, and deaths from it were known from the 15th of that month. Energetic preventive measures of treatment were at once enforced to combat the disease. All bedding, clothing, and the effects of the sick were destroyed; disinfectants were freely used. Although the disease hung about the town until October, it never attained large proportions. So far as it is possible to judge, the Havre cholera was an offshoot of that of Paris, and hardly a branch of the Indo-European cholera of 1892.

While cholera was thus widely disseminated over India and Europe in 1892, the disease had during the summer shown a tendency to extend towards the east. Later in the year we have information

from Chung-kung, *viâ* Shanghai, that Asiatic cholera of a virulent type was raging at Cheng Ten and the neighbouring districts of China.

Bacteriology.

Morphology and Cultivations of the Comma Bacillus.—The question we have to consider is,—if in persons suffering from Asiatic cholera there is a specific micro-organism in the evacuations, or in their bodies after death; and if so, what are the distinctive characters of the parasite?

In order that we may arrive at a satisfactory conclusion on this subject, it is necessary to define the meaning which we attach to the term specific organism. Dr. R. Koch has supplied us with precise information on this point. He states that a micro-organism must fulfil the following conditions before it can be admitted to be the specific agent which effects definite pathological alterations in the body. The parasite must be present in all cases of the disease. It must exist in this and in no other form of disease. This micro-organism must occur in such quantities, and be so distributed within the tissues, that the symptoms of the disease can reasonably be attributed to its action.

As far back as 1866, in a work published on Asiatic Cholera, I insisted on the fact that the disease was caused by the introduction of specific organic matter into the small intestines of human beings, and that this "vibrio" was destroyed by the acid of the healthy gastric juice; that it was necessary for its development that it should rest in an alkaline medium, having a certain temperature and moisture. I stated my conviction that this organic matter might, if swallowed, be digested in a healthy stomach; on the other hand, supposing this organ to be in an unhealthy condition, or that the germs of cholera largely diluted in water gained speedy access to the small intestines, that it would then develop and cause the symptoms of cholera. These opinions were based on experience and a careful study of the disease, extending over nineteen years' continuous residence in Lower Bengal.[1] It was almost impossible in those early days of bacteriology to advance the subject further than this; in fact, it made but little progress until 1883, when, through the labours of Dr. Koch in Egypt and in India, a most important step forward was taken regarding our knowledge of the nature of cholera. After the most careful study of the micro-organisms contained in the evacuations of persons suffering from this disease, Dr. Koch arrived at the conclusion that in all cases of

[1] *A Treatise on Asiatic Cholera*, by C. N. Macnamara, pp. 418–433.

cholera there was present in the evacuations during life, and in the contents of the intestines after death, a bacillus which in his opinion presented clearly defined characters of its own. He further asserted that this micro-organism was never present in the evacuations or contents of the intestinal canal unless in persons suffering from Asiatic cholera. Dr. Koch believes, and I concur in his opinion, that the presence of this bacillus in the intestines of human beings is sufficient to produce the symptoms of cholera; these symptoms being the result of a poison formed by the cholera bacillus in the intestines.

The appearances presented by the cholera bacillus under the microscope are not characteristic, and they vary with the medium in which the micro-organism has lived. This bacillus, vibrio, or spirillum is about half as long, but thicker than a tubercle bacillus; it has rounded extremities, and a more or less pronounced curve along its longitudinal axis (Fig. 37). The bacillus is supplied at one

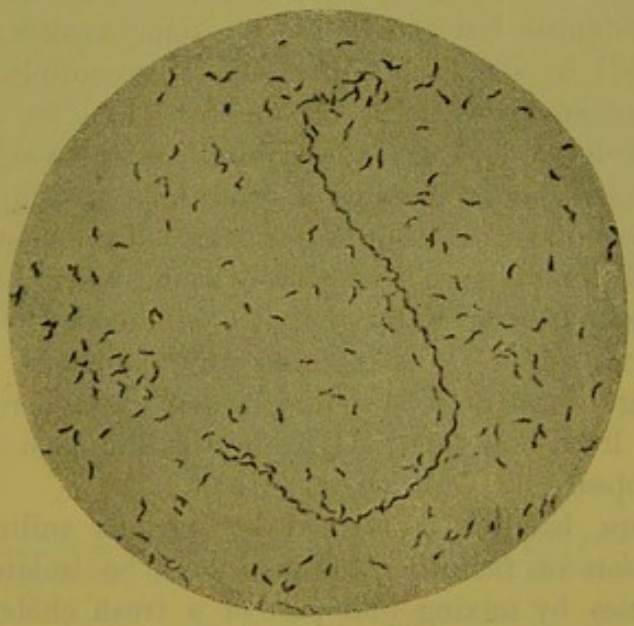

FIG. 37.—Cholera bacilli from a pure culture, twenty-four hours old, stained with fuchsine. Magnified 1000 diameters. (From Gaffky and Koch's Report.)

end with a flagellum, by means of which it exercises rapid movements across the field of the microscope. It may be isolated, but, as a rule, remains closely aggregated, often assuming an S or screw-shaped form; in old cultivations these are found with bulgings along their axis. On the whole, the cholera bacilli increase with extraordinary rapidity. Their vegetation reaches its highest point very soon; it remains stationary only for a short time, and then quickly diminishes. The growth of the bacilli is perhaps most rapid if placed in moist earth or on linen kept moist. In these conditions

even in twenty-four hours it grows extensively, and a pure cultivation of the bacillus may thus be quickly obtained; but they die after two or three days, being replaced by other organisms.

The cholera bacillus is killed if exposed to a temperature above 50° C., or to one below 15° C. It is, as a rule, destroyed after being kept free of moisture for a few days; but there is reason to believe that if the fluid in which it was contained gradually dries on moistened wool or cotton material, the bacillus may retain its vitality for some weeks, and if then removed to favourable conditions of moisture, heat, and oxygen, it may develop. The cholera bacillus multiplies by fission; it does not seem to be capable of producing spores by which its vitality can be preserved against surrounding deleterious influences. In any circumstances it thrives best when supplied with moisture and oxygen and a proper temperature. Koch has proved that the bacillus of cholera is destroyed by healthy gastric juice, and by most acids, and many chemical substances, but it will live in some weak vegetable acids. It thrives well in sterilised milk without perceptibly changing this fluid. In non-sterilised milk it can only live for a short time. The bacillus develops also in sterilised water, reaching its most luxuriant growth under favourable conditions of temperature, light, and free access of air in about seven days. In non-sterilised water it is destroyed more speedily. Koch found when he planted the bacillus in ordinary well water that it continued to live for upwards of a month; planted in dirty canal water, it lived for a shorter time; in bilge water of a ship and in salt water, Dr. Nicati found the bacillus lived for thirty days; in the drainage of cesspools it quickly disappears.

The cholera bacillus is stained by various anilines and in a watery solution of fuchsine. It may best be isolated for microscopic purposes by mixing one part of a fresh cholera evacuation with five of alkaline beef broth, and allowing the mixture to stand for ten or twelve hours at a temperature of 40° C. The bacillus rises to the surface of the liquid and forms a film, a drop or two of which shows almost a pure cultivation of the comma bacillus.

It is by means of cultivations of the cholera bacillus that we can best distinguish it from other micro-organisms. The reader is referred to works on bacteriology for directions regarding the necessary steps to be taken in cultivating the bacillus; these differ in no way from the methods employed for other micro-organisms. But I may observe that for cultivation purposes we may mix a little water in which cholera bacilli have been planted (or plant the bacilli directly) in some properly prepared and slightly alkaline

gelatine which has been rendered liquid by heat. The mixture, having been shaken up, is to be poured over a glass plate and allowed to set. In the course of from twenty-four to thirty-six hours a microscopic examination under a low power, say, fifty diameters, of the gelatine plate, shows colonies of the cholera bacillus. These present a rough granular centre of a grey or brownish-yellow aspect, having the appearance of a collection of highly refracting finely broken glass, surrounded by a clear zone, in which are scattered some dark refracting granules (Fig. 38). The bacilli as they develop liquefy the gelatine, and then sink to the bottom of the small

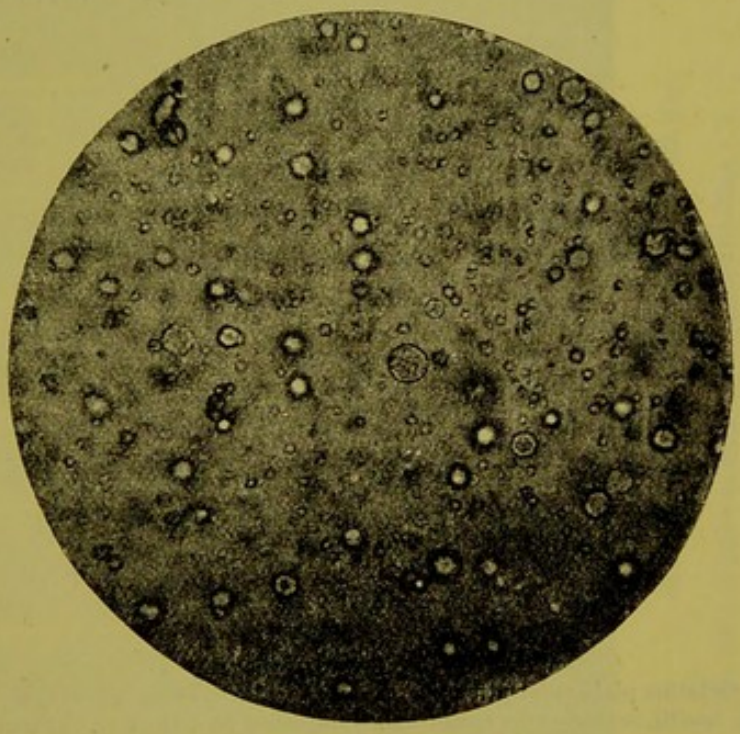

FIG. 38.—Gelatine plate cultivation of cholera bacilli, showing growth of colonies of the bacilli, forty-eight hours old. Magnified 50 diameters.

depression thus made. This change is best seen in about seventy-two hours after the bacilli have been planted; the surface of the gelatine then looks as if it had been perforated by a number of minute holes or air-bubbles; funnel-shaped depressions they really are, found in the position of the original colonies of bacilli; these pits, due to the liquefaction of the gelatine, increase in depth, and at the bottom of each a small whitish-yellow clump of bacilli may be seen, each one about the size of a pin's head (Fig. 39). From the fourth to the fifth day a halo appears round the cluster of bacilli, and, at the same time, the group assumes a reddish tinge; liquefaction extends, and soon the gelatine is completely liquefied.

Test tube cultivations of the cholera bacillus are best carried on in slightly alkaline gelatine. In the course of twenty-four hours after inoculation the track of the needle is indicated by a slight opaline line, due to a number of scattered points at which the bacillus is growing. At the end of forty-eight hours the surface of the gelatine is seen to dip. The upper portion of the culture is more or less funnel-shaped, with the appearance of an air bubble at the top. The fluid contained in the funnel-shaped cavity beneath the apparent air bubble is clear, or only slightly opalescent. At the bottom of the funnel and along the narrow neck below, greyish finely granular masses of the micro-organism may be seen, which have

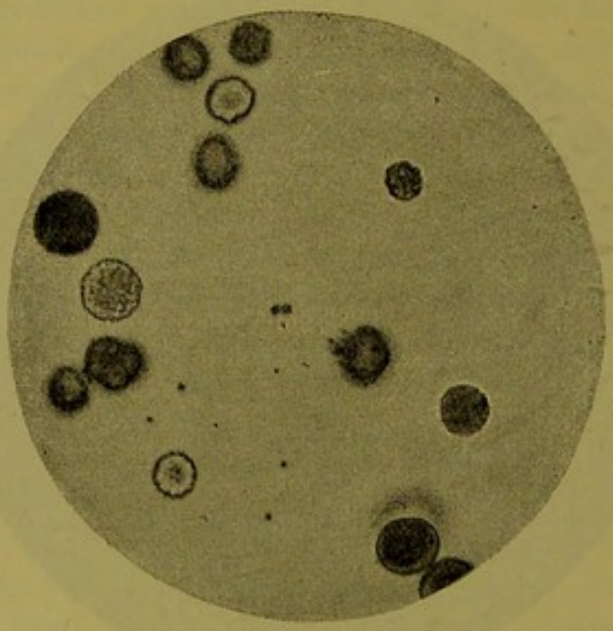

FIG. 39.—Gelatine plate cultivation of cholera bacilli, showing growth of colonies of the bacilli, seventy-two hours old. Magnified 50 diameters. (From Gaffky and Koch's Report.)

subsided from the growth of bacilli above and fallen down to the bottom of the liquefied gelatine. Under the microscope these greyish masses may be seen to consist of comma-shaped bacilli. At a later stage most of the bacilli pass into the lower extremity of the needle puncture, where they settle as yellowish-white clusters. At the end of a few weeks the upper half of the gelatine contained in the tube is converted into a turbid yellowish alkaline liquid, having a faintly ethereal odour. In about two months the whole of the gelatine in the tube is liquefied (Fig. 40).

Hueppe recommends the following method of obtaining the bacilli as the shortest. He inoculates bouillon and places it in the incubator (37° C.) for eight to ten hours, after which time there is often a development to be seen, and the cholera bacilli

may be recognised. In doubtful cases he then prepares, from the bouillon plate cultivations on agar-agar, and obtains growths after keeping them twelve hours in the incubator. Sometimes, however,

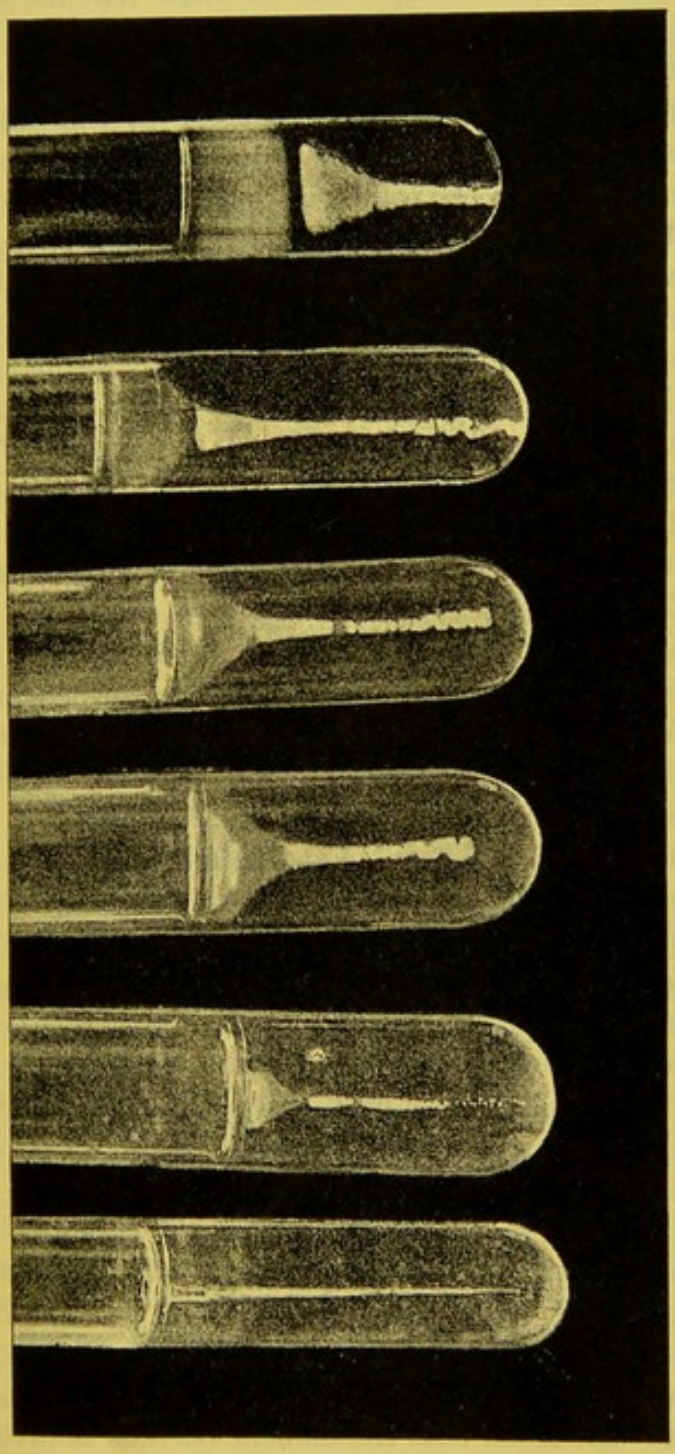

FIG. 40.—Gelatine culture of the cholera bacillus.
(From Report on the work of the German Cholera Commission in Egypt and India in 1883, by Dr. G. Gaffky, assisted by Dr. R. Koch.)

the bacilli on the agar are not of characteristic form, and he therefore also inoculates from the bouillon gelatine plates, which he keeps twenty-four hours at 20° to 24° C. Then the bacilli are

examined. The nutrient media must be of distinct though feeble alkaline reaction. Hueppe says that, as the diagnosis depends upon the appearance of the growth and the microscopical shape of the individual microbes, it is important to know that the comma bacilli vary very much in shape, and do not always grow in the typical way described by Koch. This variation partly depends upon the nutrient medium, and may be explained by the difference of the nutrient medium in the intestines of different individuals.[1]

If the surface of a slice of potato is inoculated with cholera bacilli, and placed for twenty-four hours in the culture oven, a growth of curved bacilli will be found to have developed in this faintly acid medium. To prevent the admixture of bacilli from the surrounding air, it is well to cultivate the cholera bacillus on chips of potato, which after inoculation can be placed in a test tube, its mouth being protected by a plug of cotton wool. S-shaped bodies may often be seen in these cultivations, caused by the attachment, end to end, of two bacilli curved in opposite directions.

The cholera bacillus may live on agar-agar for months, developing as a moist film of white lustre along the line of inoculation.

Blood serum is gradually liquefied by the cholera bacillus. It thrives luxuriantly in nutrient bouillon, and on the surface of such a mixture a wrinkled film forms; a growth of this kind flourishes best if the bouillon is kept at blood heat. The bulk of the mixture is rendered only slightly turbid when shaken, a few heaps of bacilli arise from the bottom, and are distributed throughout the fluid.

By treating cholera bacilli cultures grown in bouillon containing peptone (or in common nourishing gelatine) with a small quantity of pure sulphuric acid, there will shortly appear in the solution a reddish-violet, frequently purplish-red, discoloration. Bouillon cultures exhibit this reaction after remaining ten or twelve hours in the incubator, while the gelatine cultures furnish it only after a few days, liquefaction having taken place in the greater part of the culture medium.[2]

Dr. R. Koch and the greater number of bacteriologists hold that we have in the above tests definite and easily recognised features, which are characteristic, when taken as a whole, of the cholera bacillus, and that these characteristic tests differentiate this micro-organism from any other bacillus as yet described. Further, these observers hold with Dr. Koch that the cholera bacillus is invariably present, and is intimately associated with definite changes in the

[1] *Brit. Med. Journ.* vol. ii. 1892, p. 540.

[2] *Text-Book of Bacteriology*, by Carl Fraenkel, M.D. Translated by J. H. Linsley, p. 266.

intestine to be found in all cases of Asiatic cholera. Lastly, this bacillus is seldom if ever met with in the evacuations or in the intestines, either in health or in any form of disease except Asiatic cholera. During the epidemic of 1892, throughout Russia, Germany, France, and, in fact, in every part of Europe, the presence of the cholera bacillus in the discharges of patients suffering from the disease, has been received as evidence of the malady being Asiatic cholera.

Some of our most eminent English bacteriologists, however, decline to accept the conclusions of Dr. Koch as to the specific nature of the cholera bacillus.[1] Among them I may mention the names of Professors Klein and Lankester. With reference to these opinions, Professor Koch writes as follows:—

> Klein was sent to India by the English Government to carry out investigations into the etiology of cholera. From the reports by Klein which have as yet been published, we must conclude that he has exclusively busied himself in upsetting my statements. At least, I have as yet found nothing in Klein's publications but what stands in direct contradiction to my results. Any other result could scarcely be expected; for before he went to India, his judgment of my statements was decided. He attempted, at that time, to show that I had contradicted myself; that I had, in Egypt, compared the bacteria found in the wall of the small intestine with the bacilli of glanders, but that the latter were not curved, but straight bacilli; then all at once, in India, the straight bacilli had become curved ones. This objection has later, also, been brought forward by others; but—in order at once to settle this point—he who asserts this has evidently never seen side by side sections containing cholera bacilli and glanders bacilli —I have taken the liberty of placing here such preparations, and you will be able to convince yourselves that it is very difficult to distinguish these two kinds of bacteria from one another in these sections. The glanders bacilli are, no doubt, generally straight, but they are by no means rigid bodies, but, on the contrary, are soft and yielding, and very often assume more or less curved forms in the tissue when lying between cells closely packed together, to which they must accommodate themselves. On the other hand, the curved form of the cholera bacilli is not so well marked in sections. I would not hesitate a moment, if I wished to convey quickly a general idea of these bacteria to any one who had not yet seen the cholera bacilli, but who knew the bacilli of glanders, to select, as a comparative object, sections containing glanders bacilli; and it was only of the appearance in sections which I spoke when I made my returns from Egypt. With what astounding ignorance of bacteriology this matter has been treated will be seen from this drawing, which was published by Lankester, in *Nature* of December 25, 1884. In this question Lankester takes up the same standpoint as Klein, and says that it is a horrible error on my part to compare glanders bacilli with comma bacilli. In order to illustrate this, he figures here a glanders bacillus, and side by side a hay bacillus and a tubercle bacillus. As you see, these different kinds of bacilli are shown as

[1] "Further Researches on Cholera," by Dr. R. Koch, *Brit. Med. Journ.* January 1886, p. 6.

of about the same size. In fact, the glanders bacillus seems to be longer and thicker than the hay bacillus. The differences in size are, however, in reality very considerable, the glanders bacilli and the bacilli of tubercle being both very much smaller than the hay bacillus.

A further objection of Klein's, which, however, is of very small consequence, but which characterises the style of his controversy, is that the cholera bacteria are not bacilli but spirilla. It is all the same to me whether the cholera bacteria are called bacilli or spirilla, so long as one pays attention to and lays stress on their other characteristics; the name is, in this case, of the least importance. I can, however, here show you that a capable botanist, namely, De Bary, still calls curved rod-shaped bacteria bacilli.

Further, Klein states that he has found the cholera bacteria, not only in the deposits on the teeth of healthy men, but also in other diseases, more especially in persons who had suffered from diarrhœa; for example, in phthisical and dysenteric cases. He further asserts that they are present in cholera in only quite small numbers, and that the earlier the post-mortem examination is made, in so much the smaller quantity are they found, large numbers being only present in bodies where the necropsy has been delayed. In this point Klein stands in opposition, not only to me, but to all other investigators who have found the comma bacilli in numbers a very short time after death.

He further ascribes to me the assertion that the comma bacilli are killed by weak acids—a statement which is quite erroneous. I have not, in my former communications, spoken of the death of the comma bacilli, but, as you will remember, only of the hindrance to their development caused by different substances, and among others, of the absence of growth in gelatine of an acid reaction.

In India, Klein states that he found comma bacilli in the same tank in which we found them, but at a time when those who lived in the neighbourhood of the tank were free from cholera. No one knows what Klein found; his report has in England been subjected to a very thorough and able criticism by Dr. Watson Cheyne. Klein was compelled in consequence to withdraw most of his assertions, or almost all which are of importance; more especially, he had to admit that the cholera bacilli differed from those occurring in phthisis, in dysentery, and in the mouth; and he has further admitted that he has found true cholera bacilli in all cases of cholera. Thus he finally comes, under compulsion however, exactly to the same result as I did—namely, that the cholera bacteria are a specific variety, and seen exclusively in cholera. Klein will not be able to escape from all the conclusions which follow from these facts, unless he again involve himself in contradictions.

In May 1888, Sir Joseph Fayrer, writing on this subject, observes that—

In 1883 Professor Koch, after investigating cholera in Egypt, and later in India, discovered a bacillus in the alvine discharges of cholera patients, which was announced to be the germ which caused the disease. The doctrine of contagion received thereby an impulse by which the dread of it became enhanced, and Southern Europe for a time was almost demoralised by fear, whilst the old measures of coercion and quarantine threatened to be reimposed with greater severity than ever. In May 1884

the Secretary of State for India at my instance despatched a Commission (Drs. Klein and Gibbs) to investigate the subject in India. In March 1885 they submitted their report, and a committee of physicians and pathologists was convened to consider it. The following conclusion was arrived at: that comma-shaped bacilli are usually found in the dejecta of persons suffering from cholera, but that there is no ground for assuming that they are the cause of the disease, that they are, in fact, but epi-phenomena; thus confirming the conclusions of Lewis and Cunningham, arrived at years before, after a long and careful microscopic study of the disease in India.

There can be no question that the vibrio of Metchnikoff is nearly related to that of the cholera bacillus of Koch; there is this difference, however, that whereas the cholera bacillus can only be transmitted to animals by artificial means, that of Metchnikoff has the most pernicious influence on almost all animals; vast quantities of the bacteria being found after death in the blood and tissues. Other forms of vibrio, such as Emmerich's, Deneke's, and Finkler-Prior's, have one after the other been described as identical with Koch's bacillus of cholera; but they have each and all of them, when examined by proper tests, been found to differ from it in various ways, their study having clearly demonstrated the fact that the cholera bacillus is a specific micro-organism.

It is evident that in its growth the cholera bacillus changes the media in which it grows, liquefying gelatine and producing a definite chemical substance of basic character, which, in all probability, when absorbed into the blood causes the symptoms characteristic of cholera. Substances thus produced from the action of the cholera bacillus are poisonous if inoculated in full doses into various animals, and in pure cultures in very minute quantities it causes peculiar symptoms in man, which I shall subsequently describe; it is asserted that a person so inoculated is, for the time, immune to cholera.

Inoculation Experiments.—Dr. Koch has introduced pure cultures of the cholera bacillus directly into the intestinal canal of animals through the stomach, that is, without opening the walls of the abdomen. He first neutralised the acid contents of the stomach by injecting into it a 5 per cent. solution of carbonate of soda. After the contents of the stomach have thus been rendered faintly alkaline, Koch introduced into it, through a tube, a solution of a pure cultivation of cholera bacilli; he then injected a small quantity of opium subcutaneously, so as to lessen the peristaltic action of the intestines, his object being to retain the cholera bacillus as long as possible within the intestinal canal. Thirty-five guinea-pigs were experimented on in this way, and "thirty of them died from

cholera." The symptoms and post-mortem appearances of the animals were the same as those in which the injection had been made directly into the duodenum. The animals treated as above described, on the day after the bacillus had been injected into their stomachs, appeared to be ill, looking shaggy; they did not eat. On the following day they suffered from paralytic weakness of the posterior extremities; the animal no longer supported itself on its hind legs, but lay quite flat, with its limbs stretched out. The respiration was weak and slow. The head and extremities were cold, the pulsation of the heart hardly perceptible, and the animal died after it had lain for a few hours in this condition. Immediately after death an examination was made of the contents of the intestines, and they were found to contain a pure culture of the cholera bacillus. The walls of the abdomen were flaccid, the internal surface of the small intestines markedly infected. The stomach and cæcum contained a quantity of fluid. The epithelial pulp found in the intestinal canal was in many instances crowded with comma bacilli. The urinary bladder was empty, the gall bladder full of bile.

Dr. Shakespeare, from numerous observations, confirms Dr. Koch's facts as above stated; he remarks that "guinea-pigs are susceptible to the influence of the cholera bacillus introduced through the stomach—after a period of three days' incubation, accompanied or not by cyanosis, prostration, algidity, spasmodic muscular contraction, and death." The post-mortem changes above referred to may always be found.

Other observers have confirmed these observations; and it seems almost impossible to doubt the truth of the statement that pure cultures of the cholera bacillus are capable of causing in animals symptoms similar to those produced by this micro-organism in the human subject, and that animals dying under these conditions present the same pathological changes as are found in the bodies of human beings after death from Asiatic cholera.

With reference to human beings: during the course of instruction on the bacteriology of cholera carried on in the laboratory of the Imperial Board of Health, Berlin, one of the gentlemen attending the course became infected with the bacteria; he had been five days in Berlin, when he was attacked by slight digestive disturbance, accompanied with diarrhœa. The evacuations were loose, and occurred several times a day; so that his condition excited no alarm. But, on the last day of his attendance here, more frequent, quite thin, watery evacuations occurred. He thought, however, that he was able to travel; he did so, and reached home, but was then

seized with a true attack of cholerine. For two days he had very frequent watery and colourless stools; there were great weakness and unquenchable thirst; the urinary secretion was reduced to a minimum. True cramp of the calves of the legs did not occur; but there was marked contraction of the sole of the foot, and cramp in the toes. As he felt too weak to examine his evacuations himself, he put a small quantity in a well-cleaned flask, and sent it to Berlin. The vessel was sent off in the evening, arrived on the following morning, and was at once investigated. As the transit only occupied a night, and that in the cold season of the year, the contents could not have been materially altered by the transport. The investigation of the dejecta, which was made by means of cover-glass preparations, and also by plate-cultivations and cultivation in cupped slides, showed by each method the presence of very numerous true cholera bacilli. One of the pure cultivations of cholera bacilli shown in the laboratory came from the dejecta of this case.

A case of a somewhat similar kind came under my own observation, in which cholera dejecta became mixed with water. This water remained for a whole day exposed to the heat of the sun, and was then drunk by nineteen persons, five of whom were taken ill with cholera within thirty-six hours.[1]

As I shall subsequently have to refer to Dr. Ferran's inoculations of human beings with the products of the pure culture of the cholera bacillus, it is unnecessary in this section to describe the remarkable results which he appears to have obtained by this system of treatment.

The Comma Bacillus Pathogenic.—From the evidence at our command, therefore, it seems impossible to question the truth of Koch's views regarding the existence of a specific bacillus in cases of Asiatic cholera. This bacillus is present in every case of cholera, and in such quantities in the dejecta and contents of the intestinal canal as to account for the symptoms of this disease. The history of the disease is perfectly in accord with this statement, for we find that no instance has ever yet been recorded in which Asiatic cholera has manifested itself among people, until they have been brought into relation with those suffering from the disease, or to articles of consumption, or fabrics tainted with the discharges from persons affected with cholera.

It is perfectly true, as Virchow remarks, with respect to the silk-worm disease (mus-cardine), an undoubted mykotic affection, studied now for many years, that "we cannot, even to-day, state with certainty what are the reasons for its appearance alternately in greater or less extent, nor can one say what one must do to suppress

[1] *A Treatise on Asiatic Cholera*, by C. N. Macnamara, p. 196.

it." Virchow adds: "During my studies of natural science, I have always been inclined, whenever an observation has been made in a single concrete case under every guarantee of certainty, not to make the acknowledgment of the correctness of such an observation depend on its ability to account for everything." With regard to cholera, Virchow remarks: "Its cause is a specific bacterium. This passes from man to man by means of moist agencies, especially drinking water; is received with the food, and, being developed in the intestines, gives rise to cholera. Its entrance and increase are probably facilitated by certain favourable conditions of the intestinal canal, an individual predisposition, to be accounted for, perhaps, by a diminished acidity of the stomach, and inertia of peristalsis."

In answer to the question whether Asiatic cholera can be ascertained in cases in which no bacilli are found, Virchow said: "The bacteriological investigation is of such a nature that parts of the liquids evacuated by patients are placed on a soil favourable to the development of bacilli, after which the investigator waits to see whether they develop. In this procedure, as in that of the sower, it may happen that hope is disappointed, owing to the unfavourableness of the soil. As science, however, can hold nothing fast but facts, the investigator can only state that he has found "no comma bacilli; the case, therefore, is not one of Asiatic cholera."

Etiology.

Relation to Ground Water.—From the history of Asiatic cholera we learn that the endemic area, or home of the disease, is located in Lower Bengal, from a line drawn through the district of Pooree north to the Himalaya, and from the delta of the Brahmaputra River through Tipperah to the east. The mortality from cholera in this region is, as a rule, highest during the dry season, that is, from October to May. Throughout the rains the death-rate from cholera decreases. At first sight it might seem as if there were some connection between the rise and fall of the underground water, and the increase or decrease of cholera in Bengal. But on examining into details it appears that the connection is incomplete. For example, the underground water in Lower Bengal reaches its lowest point in May, whereas the maximum cholera mortality by no means takes place in that month. During December and January the level of the underground water subsides, and yet the mortality does not rise; on the contrary, it falls. The connection between the changing state of the underground water and the rise and fall of cholera in other parts of India is not found in

any way to coincide, and is unimportant with reference to the spread of the disease in India.

Relation to Rainfall and Winds.—In the same way numerous statements have been advanced as to the supposed effect of the rainfall in India upon the death-rate from cholera. There is no reason, however, to believe that the rainfall in itself has any direct influence on the spread of the disease; excessive rain seems at times to hinder its extension, and so does the absence of rain (p. 366).

It has often been assumed that the south-east wind, blowing over Lower Bengal for several months of the year, is a means by which cholera is carried from its endemic area over Northern India. There can be no question as to the fact that before the days of railroads cholera spread from Lower Bengal northward at that season of the year when the prevailing wind blows from the south-east. It was by means of this wind, however, that large country boats laden with goods and passengers passed along the Ganges from Bengal to the north-west of India; and consequently, as the Marquis of Hastings observed in 1817, "the disease gradually ascends the river from the lower provinces." Beyond this the monsoon seems to have no special influence in disseminating cholera over the hill country immediately to the north-west of the endemic area of the disease; the aborigines inhabiting these hills are, in fact, singularly free of cholera until they leave their homes for the plains of Bengal. Again, Dr. Leith (p. 36) informs us that cholera advanced from the east into Bombay in 1849, in the teeth of the south-west monsoon. Quarantine has frequently, by isolating an infected locality, or by separating a healthy place from an infected one, demonstrated that Asiatic cholera does not spread by means of the atmosphere beyond, we may affirm, a distance of about 100 yards. We can readily understand this, because we know that the cholera bacillus is a delicate plant, incapable of producing resisting spores, and is destroyed if kept for any length of time without moisture. On the other hand, the disease may be conveyed from one place to another by an infected individual, breaking out where he stops, and passing over intermediate places through which he has passed without stopping; although the two places may present identical conditions of air, temperature, moisture, and so on.

Altitude and Temperature.—From the history of the disease we learn that although cholera manifests itself with virulence among the inhabitants of over-populated, badly ventilated, and dirty, low-lying towns, nevertheless from time to time it breaks out with deadly effect in places situated 7000 feet above the level of the sea. It stands to reason, however, that an enormous majority of the popula-

tion of the globe are located in and about large commercial towns generally built on rivers. Temperature has, doubtless, much to do in arresting or in aiding the growth of the cholera bacillus. We know that this micro-organism is killed if exposed to a temperature of 15° C., or to one of over 50° C., and from the history of Asiatic cholera we have noticed that year after year it dies away in the cold weather of the North-Western Provinces of India and the Punjab. The effect of cold in arresting the progress of epidemic cholera is still more marked during the continuance of a European winter; when spring returns the disease reappears, becoming most vigorous in summer. As an example, we may refer to the disappearance of cholera after being introduced into New York during the winter of 1848; and of its spread at the same time of the year from New Orleans during hot damp weather. Cholera has, however, sometimes extended its ravages in the winter, as was the case among the troops of the Russian army in Poland in 1830–31; but we must take into account the high temperature at which many of the houses in Russia are maintained during their long winter.

We are, therefore, in a position to affirm that Asiatic cholera is endemic among the inhabitants of Lower Bengal, but that it does not extend from its endemic area by means of the wind—in other words, it is not carried by the wind from Lower Bengal over India. When introduced by human beings suffering from the disease into a locality, no kind or condition of soil can prevent it from spreading among the inhabitants of the place. No race of men are exempt from attacks of cholera; those living at 7000 feet above the level of the sea may suffer equally from the disease as persons dwelling at or below the sea level. But people existing under bad sanitary conditions are more liable to suffer from cholera than those who live in healthy localities.

Influence of Sanitary Conditions.—The influence which imperfect sanitary conditions have on the dissemination of cholera is twofold; in the first place, people dwelling in these conditions are less capable than their more favourably placed brethren of resisting the development of a micro-organism like that of the cholera bacillus, when it enters their stomachs mixed with water or food. Beyond this, there is every reason to believe that the cholera bacillus is dangerous, not only according to the dose received into the stomach, but also in proportion to the favourable or unfavourable conditions under which it has been developed. The cholera bacillus, I repeat, is a delicate organism; its vigour depends on the medium in which it is sown, and upon the temperature in which this medium is placed, upon the free access of oxygen, and, I believe, of light. Moisture is

essential to its development, and probably certain salts of lime as well as organic matter. If the bacillus is sown, as Watson Cheyne has observed, in a particular locality, what happens? It is deposited in the soil, in the water, etc., and, according to the conditions in which the bacillus finds itself, it grows or it does not grow. If it is deposited in a place where the sanitary conditions are good (in other words, where the conditions are adverse to its growth), it either dies out or it grows with difficulty. If it grows at all, it may be that small numbers or a weak dose finds its way into the intestines of some of the inhabitants through water or food. But these quantities, being small and of low vitality would be insufficient to cause virulent disease, and would either produce no effect at all or only a mild diarrhœa. Hence we find that in places where the sanitary conditions are good, cholera does not spread to the same extent as it does among people living under the reverse conditions. Impure water supply and other results of defective sanitation may also favour the dissemination of the germ.

Mode of Dissemination of Asiatic Cholera.

Human Intercourse.—From the history of the disease we learn that before the English entered India, epidemic cholera extended over that country from time to time; and we have every reason to believe that it then had its endemic area in Lower Bengal (p. 351). But we may be equally sure that Asiatic cholera had, until 1830, never spread from India east and west. The reason of this seems to be that India was practically cut off from the rest of the world by its position. It is true, goods reached Europe from India, but only after something like a year's passage up the Red Sea and across the desert to Koptos, a distance of 280 miles; they were then carried 500 miles down the Nile to Alexandria.

The first steamer that navigated the Red Sea was the *Hugh Lindsay*, which left India in the same year that cholera spread from that country to Europe, 1830. The vessel took thirty-two days to reach Suez. In 1834 a Committee of the House of Commons reported that it was practicable to carry on steam communication between Suez and Bombay during the north-east monsoon; but they were doubtful as to its practicability at other seasons of the year. The route from India to Europe *viâ* the Persian Gulf, or through Cabul, was no less protracted, and for long periods was strictly prohibited. Consequently, it was almost impossible for cholera to have been communicated to Europe in these early times, supposing it spreads from infected persons to healthy

communities. No sooner, however, had England taken possession of India, and opened up relations with Persia, Arabia, Europe, and China, than Asiatic cholera manifested itself in localities along the routes followed by our men and merchandise. First breaking out among the inhabitants of seaport towns into which vessels from infected localities discharged their passengers and cargoes, and from these seaport towns the disease spread inland.

In like manner, from the first epidemic outburst of cholera of which we have any authentic record in 1782, up to the present time, the assemblage of a large concourse of pilgrims at places like Hurdwar or at Mecca has afforded a favourable breeding ground for cholera; and when the disease has broken out among such an assemblage of human beings, it has spread far and wide with them to localities previously healthy.

From these, and, in fact, from the whole history of the disease, we arrive at the conclusion that Asiatic cholera extends from one place to another through means of human intercourse. We may go a step further, and assert that no amount of overcrowding, of famine, poverty, filth, or any other condition, have ever originated an epidemic outburst of Asiatic cholera beyond its endemic area. Before the disease can occur among people outside the home of the disease, its seeds must have been carried to them by persons suffering from cholera, or by articles of clothing which have been soiled by the excreta which patients have passed during their illness. In other words, living cholera germs must be planted among the inhabitants of an uninfected place before Asiatic cholera can develop among them.

Drinking Water.—The influence which drinking water has in disseminating cholera among an otherwise healthy population, is a fact which no unprejudiced person can deny at the present time. The first authentic account we have of a widespread epidemic of this disease was given by the Marquis of Hastings in 1817; and it is remarkable that he should have attributed the spread of cholera in his camp to the consumption by his soldiers of impure water. Acting under this impression, he removed his camp from this source of drinking water to the banks of a running stream, and, in the course of a few days, again to the vicinity of a rapidly flowing river, with the result that cholera disappeared from among his men as if by magic. Striking as are the details of this incident, no one seems to have profited by its teaching, or to have applied the principle to the relief of bodies of men similarly affected, until within the last few years, when our military authorities in India have followed the example set them by the Marquis of Hastings in 1817; and, on the occurrence of an outbreak of cholera among troops in a

station, they are marched out into a cholera camp, that is, a spot previously selected and kept in readiness for the occasion. One of the chief objects held in view when preparing such a camp for the use of our soldiers is, that the water supply should be unexceptionable, and be strictly guarded from all chance of contamination. The establishment of these cholera camps in India, has been attended by a marked improvement in the death-rate from this disease among our European army located in that country.

In the year 1857 Dr. Snow published the particulars of the Broad Street case (p. 362), and I have referred to other instances of the same kind. In these cases, water contaminated with the excreta passed by persons suffering from cholera caused the disease. We may, however, turn to the other side of the picture, and refer to the remarkable results following the supply to a body of men living in the endemic area of cholera of pure drinking water. From the year 1826 until 1864 our European soldiers in Fort William, Calcutta, were, year after year, subject to cholera; in some seasons, as in 1857, out of a strength of 700 men, no less than 73 died from Asiatic cholera. The average mortality, however, for the above period from this disease was 20 per 1000. From the year 1863 up to the present time, the death-rate per 1000 of our troops in Fort William from cholera has fallen from 20 per 1000 to 1 per 1000. In the year 1863 the fort was, for the first time since it was built, supplied with pure drinking water, with the result referred to. In the same way the death-rate from cholera among the inhabitants of Calcutta has decreased in a marked manner since the year 1870, when a pure supply of water was provided for the town. The decrease in the death-rate from this disease among the inhabitants of Calcutta has not been so marked as that which has taken place in Fort William, because, in many parts of the city, the municipal water supply is imperfect, and people still consume contaminated tank and well water.

Dr. Guttmann, chief physician of the Moabit Hospital in Berlin, delivered an interesting lecture, on the 23rd September 1892, on "Cholera," at a meeting of the Society for Internal Medicine. He pointed out with satisfaction that the epidemic has not assumed any great dimensions in Berlin. The cases that have occurred prove beyond doubt that the water for drinking purposes is one of the chief sources of infection. The fact that Professor Carl Fraenkel, of Marburg, has found the comma bacillus in the docks at Duisburg, on the Rhine and Ruhr Canal, shows how far the cholera infection has travelled along the rivers. Thus the disease has broken out on board vessels which had had no intercourse either with the

inhabitants of infected districts or with other vessels. Dr. Guttmann further urged the great importance of sending persons who are only suspected of cholera to the hospital. Many such patients, though not suffering from cholera, when received, afterwards fell ill of that disease. The sending of them to the hospital enables them to be isolated at once. Without this they would have become centres of infection. He emphasised the diagnostic importance of the comma bacillus. There are, he said, real cases of cholera so mild that nobody would take them for such, and again there are other cases having nothing to do with cholera which sometimes take a very malignant course. He mentioned a case of antipyrin poisoning, for instance, which showed all the external symptoms of Asiatic cholera. But the presence of the comma bacillus in any case at once cleared up the nature of the disease from which the patient was suffering.

Milk.—However pure the drinking water consumed by the inhabitants of a cholera infected place may be, it is certain that milk, which forms so large an element of the diet, is liable to be contaminated with cholera excreta by being mixed with impure water. I gave the particulars of a case of this kind which occurred under my own observation (p. 374); and Dr. Simpson, the present able Officer of Health in Calcutta, has supplied the details of similar localised outbreaks of the disease, caused by persons drinking milk which, there was good reason to believe, had been adulterated with contaminated water.

Other Modes of Dissemination.—It is possible that the germs of cholera may pass into the circulation by other paths than the stomach, but we have no reliable evidence on this subject. From the details of numerous cases, to some of which I have referred (pp. 360 and 364), it is more than probable that the disease may be communicated through articles of clothing which have been contaminated by the evacuations of persons suffering from it. In like manner the bodies of individuals who have died of cholera may become a source of infection.

The attendants upon cholera patients are, as a rule, safe from fear of contracting the disease during their care of the sick. It is under peculiar conditions, such as those to which I have referred as existing at Mian-Mir in 1861 (p. 368), that attendants on persons suffering from cholera generally contract the disease. In this case the air of the hospital was foul in the extreme, and doubtless charged with the germs of cholera, which, I suppose, were absorbed by the drinking water, contained, as it was, in large open, porous vessels standing in the cholera wards. This water was largely consumed by the European orderlies in attendance on their sick comrades, and many of

these poor fellows were soon down with cholera. On the other hand, when the European orderlies were relieved by native troops none of these latter took cholera; for the obvious reason that, although inhaling the same atmosphere, and performing duties exactly similar to that of the European orderlies, the native was prevented by his caste from drinking the water, or allowing anything to pass his lips in the shape of food, so long as he remained in the hospital.

That the immunity enjoyed by hospital attendants is not so complete as is generally supposed, has been shown by Dr. Davidson, who has taken the trouble of analysing the figures bearing upon this point, as furnished by the reports of the Indian Government.[1]

Cholera, like typhoid fever, is not communicable by contact. A person may rub, wash, and administer to the necessities of a patient suffering from the disease without risk of contracting it, provided the room in which the sick person lies is properly ventilated. The disease is communicable from the sick to the healthy in the manner above described, but, in the proper meaning of the term, it is not a contagious disease.

Preventive Measures.

Quarantine.—Before entering on the more immediate consideration of this subject, it is necessary to make a few observations on maritime and land quarantine. I take it that the meaning now attached to the word "quarantine" is that given in Webster's *English Dictionary;* it is "*A period of time, variable in length, during which a ship or vessel supposed to be infected with certain diseases is not allowed to communicate freely with the shore.*" This is the sense in which the term was employed by the delegates of the International Sanitary Congress of 1874 and of 1892. In the report drawn up and signed by the delegates of all the European Powers, including England (of the Congress of 1874), the second section is devoted to rules and regulations appertaining to "*questions relating to quarantine.*" The first rule under this heading refers to the examination of all vessels arriving from infected parts by duly constituted sanitary officers. These officers are allowed either to grant a vessel free pratique, or to order her and everybody, and everything she contains, to be isolated from communication with the shore. If cases of cholera have recently occurred on board, the "crew and passengers, after the removal of the sick and dead, are to undergo a careful process of disinfection." Any one on board having cholera, or who may be attacked by the disease after the vessel has been placed in quarantine, is to be removed to a hospital

[1] *Geographical Pathology*, vol. i. p. 434.

specially provided for the care of such patients. At the expiration of five days, if no fresh cases of cholera have occurred on board the vessel, provided her passengers and crew can give satisfactory references as to the places in which they propose to reside on shore, they may by permission of the medical officer leave the vessel, but they remain under inspection for a week or so longer.

The Sanitary Congress of 1874 further appointed a committee to consider and draw up rules as to the management of "*the quarantine establishments* in those States which intended to erect such." It is obvious that the sanitary authorities of Europe draw a marked distinction between quarantine regulations, and quarantine establishments—they are treated under different sections. Regarding the former, the delegates were almost unanimous; as to the latter, there was a considerable difference of opinion. A system of quarantine establishments has kept cholera out of isolated localities, but it means lazarettos, the crowding together of people indiscriminately who have arrived from infected countries, and the complete cessation of free intercourse between a country where cholera exists and unaffected places. It seems to me therefore that the Right Hon. H. H. Fowler, President of the Local Government Board, is inaccurate when he states that quarantine "has never been resorted to in this country since 1848," its regulations are in force at the present time; but England does not approve of quarantine establishments, and I believe she is quite right in the theory and practice she follows.

As far back as 1869, in a work I then published on Asiatic cholera, I urged in the most forcible language at my command the following ideas regarding the preventive treatment of cholera; I stated that "the fundamental principle upon which all such treatment must be based is, that Asiatic cholera can only spread through means of food or water contaminated with the infecting matter passed by persons suffering from this disease;" that "cholera is not a contagious disease in the ordinary sense of the term, but it is nevertheless eminently a communicable disease, through means of the infecting matter passed in the evacuations of persons suffering from cholera, which organic matter must gain access to the intestinal canal of another person before it can produce the symptoms of cholera. In this lies the secret of preventive treatment; for if this specific matter is destroyed immediately it is passed by patients suffering from cholera, the disease cannot spread. In the same way articles of clothing, or, in fact, any and everything soiled by the evacuations of a person suffering from cholera, as well as his body, if he dies, must be destroyed, in order that we may effectually stamp out the disease. . . . It is evident the more perfect quarantine is enforced round India, the better as regards pilgrims and native traffic, which we cannot otherwise control; but," I added, "this rule does not apply to well-conditioned vessels under the order of European officers and crew. The reason for this is obvious, for we can insure the destruction of the organic infecting matter in the latter instance, whereas in the former it would be impossible to make

certain of any such result. Supposing a first-class steamer starts from Bombay or Calcutta, and cholera occurs among the crew within four days of her leaving India, if proper precautions are taken to destroy the infecting matter, and no cases occur within six days of the arrival of the vessel at Suez, there could be no reason for detaining the passengers, provided the quarantine officer could satisfy himself that the reasonable precautions above indicated had been faithfully carried out, and all the apparel of the patients, and everything likely to have been contaminated by the infecting principle, destroyed. Supposing a case of cholera to have occurred within six days of the ship's arrival at Suez, it would be the duty of the quarantine officer to examine into the circumstances of the case, and to use his judgment as to the course to be pursued; for instance, the fact of one of the crew being affected, if the passengers were healthy, would hardly be a valid ground for detaining them. It might be a different matter if the vessels were going into a crowded port, or still more, into dock, in which case I would certainly allow no communication with the shore until six days had passed without any fresh instances of cholera occurring on board. I have before stated my firm belief that cholerine is a mild form, but the same disease as cholera. The dejecta, therefore, of patients suffering from cholerine must be destroyed in the same way as those of cholera."[1]

I did my best, so long as I remained in Bengal, to get these ideas recognised and acted upon by the Governor of India; but the Government and their sanitary advisers had other views on these matters (p. 379). It is, however, satisfactory to find that the Dresden International Sanitary Conference of 1893 have expressed opinions on this subject precisely similar in principle to those above quoted. And what is of still more importance, all the great Continental Powers are now determined that these principles shall be carried into practice as regards the preventive treatment of cholera.

The quarantine regulations which England now enforces are based upon the knowledge we possess regarding the cause which produces Asiatic cholera, and the means by which it spreads over the world; so long as we can keep the bacillus of cholera from our shores, England is quite safe from this disease. We may go a step further and assert that if the germs of cholera obtain a resting-place among us, that if medical men will take the proper steps to kill the baccilus as it passes from patients under their care, by means of chemical agents, that epidemic cholera cannot spread among the people of this country.

The precautionary measures to be adopted against Asiatic cholera may be considered under three heads: first, those intended to prevent the transport of cholera from India to surrounding countries and to Europe; second, the measures to be adopted to prevent the spread of the disease after it has been imported into places beyond the confines of British India; and, lastly, we have to consider whether by inoculation it is possible to render individuals immune, or insusceptible to the disease when it has broken out among the surrounding population.

[1] *A Treatise on Asiatic Cholera*, by C. N. Macnamara, pp. 464–474.

With reference to preventive measures in general, the International Sanitary Congress of 1874 laid down the following principles:—*General sanitation and isolation, real and complete, of everything which might introduce the disease, are the best means for preventing the importation and subsequent propagation of cholera.* The object being by complete supervision of the intercourse between persons, or soiled things from an affected locality, to prevent the passage of cholera germs into unaffected places.

Cholera having become epidemic beyond its home in Bengal under peculiar conditions of soil, temperature, and other circumstances which we do not understand, extends by human agency over India, and so to Cabul and Persia, or to Bokhara; it may be carried directly from Bombay by trading vessels up the Persian Gulf, or to Arabia. The way through Cabul, or up the Euphrates, has been the most frequent route by which an invading cholera has travelled to Europe. It would be difficult by land quarantine to stop the progress of epidemic cholera westward, when it has reached Bokhara, Persia, Arabia, or Turkey in Asia. The countries also bordering on the west of India, through which the disease extends towards Persia and Russia, are inhabited by half-civilised fanatics, and beyond the control of the British or any other civilised Government. Nevertheless, I feel sure that an effort should be made, not only to mitigate the ravages of cholera in India, but to prevent it passing from that country into Cabul and Kashmir.

The evidence of Asiatic cholera having been carried directly from India *viâ* the Suez Canal to Europe is incomplete; and if we exclude the outbreak in Southampton in 1885, I know of no evidence which demonstrates the fact of cholera having been introduced into England by vessels passing through the Canal from India. The cholera of Southern Europe of 1885 is said to have originated in Tonquin, and to have been imported by French vessels to the South of Europe; if this were the case it may account for its exceptional character, in that it appeared to have possessed but feeble power of spreading far from the Mediterranean coast; it was hardly therefore an invading cholera. Doubtless, as time goes on, other countries beyond British India may become endemic foci of the disease; and as railways and steamers give facilities for the transmission of men and goods from these areas, cholera may be more frequently imported into Europe.

The history of this disease hardly sanctions this idea, so far as the trade carried through the Suez Canal is concerned; for the Canal was opened in 1869, and the last widespread epidemic of cholera in Europe, excluding the present outbreak of the disease, occurred in 1865–66. Pilgrims returning from Mecca to Egypt and Turkey may,

and should, be prevented from disseminating cholera in places along their homeward journey by quarantine regulations and establishments enforced at the entrance of the Suez Canal. Quarantine at Suez is a necessary safeguard to Egypt and to Europe from infection by this class of people. But it is equally certain that vexatious measures of quarantine, applied to steamers carrying European passengers from the East to Europe will defeat their own ends. Harsh measures of this kind lead to the concealment of the truth regarding doubtful cases of cholera, in order that the suspected vessel may escape detention at Suez. I believe that if sounder knowledge as to the nature and the history of cholera were diffused among the officers and men of our Mercantile Marine, it would be a more effectual means of guarding Europe from cholera than quarantine regulations of a harassing character enforced at Suez. When once the officers in charge of such steamers as those which pass through the Egyptian Canal understand the terrible consequences likely to arise from landing passengers suffering from cholera, either in Egypt or in Europe, or articles of clothing used by the sick, we shall have gained the best protection against the spread of cholera by such vessels from India or other places.

It is beyond my purpose to enter into minute details regarding quarantine regulations: these have been settled by the International Conferences of 1874, 1890, and 1893; the principles upon which they are based have been referred to at the commencement of this section. They include isolation on board ship of persons suffering from cholera, and on arrival in harbour of the removal of the sick on shore under the supervision of constituted sanitary authorities. A vessel in which cases of cholera have occurred must be thoroughly disinfected, and retained in quarantine for five days, after which, if no fresh cases have occurred on board the ship, she may be permitted to proceed on her voyage. The management of the excreta passed by patients suffering from cholera on board ship, and of articles of clothing they have used during their illness, is to be effected in the same manner as when the case occurs on land.

Measures for Preventing the spread of Cholera in a Community.—To prevent the spread of cholera in a community into which the disease has been carried, the first cases of cholera must be isolated. The premises in which the patient resides are to be thoroughly disinfected. Especial care is necessary to watch and isolate cases of this disease travelling over a line of railway, or by river steamers. Rigorous cleanliness should be observed upon trains and at railroad stations. Disinfectants must be employed, both to patients after recovery from cholera and also to their clothes and

bedding. The bodies of persons dying from cholera should be cremated, or enclosed immediately after death in sheets soaked in disinfectant fluids, after which the corpse must be covered with chloride of lime, and buried as soon as possible.

The following is a summary of the chief provisions of the Convention of 1893:—

"The Governments engage to notify to the contracting States every appearance of a focus of cholera on their territory. They shall specify the locality and the extent of the invasion, and also what measures have been adopted for its limitation.

"The Conference has specially stipulated that foreign countries shall only put in force restrictions against the productions of the infected area, and not against those of the whole country of which that area forms a part.

"Prohibitions against imports shall only be applicable to objects capable of transmitting the disease, and a list is given, enumerating by name the things considered dangerous. We have reason for believing that this list contains only a comparatively small number of articles, and that it in no way interferes with the transport of manufactured goods, textile or woollen fabrics, food supplies, or even with the exportation of fruit.

"The measures prescribed by which a Government may protect its frontiers, or which it may take against an infected area within its boundaries, are founded on medical inspection, disinfection of soiled linen, sanitary passports, and surveillance of passengers in the country to which they go during the time which corresponds to the incubation of cholera. The Conference decided that this period should be taken as five days, which, although not perhaps scientifically accurate, is practically sufficiently near the mark to cover the great majority of cases.

"As regards ships, the passengers will not be isolated or disembarked unless there either is cholera on board, or there has been a case within seven days. But we may assume that if it should be put in operation by any country, it would be carried out on the plan accepted by the Venice Convention, in regard to the Suez Canal, in which it was arranged that people should be isolated in small groups (*aussi peu nombreux que possible*), so as to avoid the detention of a large number of people if any secondary outbreak should occur.

"In the case of emigrant ships, special precautions may be taken, and special regulations made by the country receiving them, which is, in fact, only what we have done in England by the Special Orders issued last September by the Local Government Board.

"In regard to the navigation of the Danube, provisional regulations have been made until such time as the town of Sulina at its mouth shall have provided itself with a proper water supply."[1]

Dr. Cornet, of Berlin, believes that a person apparently perfectly convalescent from cholera may carry about with him in his intestines active, living comma bacilli.

"The case in which this was noticed was that of a man whose mother, wife, and son died of cholera. He himself had a slight attack, and was put under the care of Dr. Carl Lauenstein of the Seemann's Hospital. He was nine days in hospital, and received five tannic intestinal injections against his diarrhœa, which was not of a very severe type. The patient recovered

[1] *Brit. Med. Journ.* April 29, 1893.

perfectly, and was on full diet. On Friday he was up and very anxious to go home, but was induced to stay. On Saturday he was still better, and had no motion at all, and it was with difficulty he was induced to remain. On Sunday Dr. Cornet discovered that the stools passed on Friday still appeared to contain comma bacilli.

"The importance of the discovery that a patient apparently perfectly convalescent may still be the means of disseminating the disease cannot be overrated. The fact will probably be as unwillingly recognised and accepted as the kindred one, well known to those who have investigated the subject, that the milder so-called cholerine, which so often precedes cholera epidemics, is true Asiatic cholera."[1]

Cholera bacilli have been found alive in the bodies of persons eleven days after death; at the latest they have been detected in the stools of persons ten days after the commencement of the attack of cholera; as a rule, they disappear from the evacuations on the fourth day after the first symptoms of the disease have set in. By far the most effectual means of preventing the spread of cholera is the isolation of the patient, and the disinfection and effectual disposal of the evacuations immediately they are passed by the patient. Our object being to prevent living cholera bacilli passing into rivers or any sources from which the supply of drinking water is drawn.

From my own observation, and from all that has been learnt on the subject, we can affirm that Asiatic cholera cannot remain latent in the system for more than five days. If, therefore, a person coming from an infected locality has no symptoms of the disease for five days, he may be permitted to pass with freedom among his fellow-creatures.

Disinfection.—Regarding the disinfection of soiled articles of bedding and clothing, steam at a temperature of 100° C. is most effective. A 5 per cent. solution of carbolic acid, or of chloride of lime, may be relied upon; a weaker solution being used to disinfect persons. Vomited matter and the stools passed by the sick person should be received in vessels which contain a disinfectant; or, immediately they have been passed, they should be mixed with a disinfectant, and emptied, if possible, into properly prepared holes in the ground, away from wells or other sources of drinking water. If in any locality there is a suspicion of the water supply having been contaminated, or if cholera exists, it should be boiled before being swallowed; the same remark applies to milk: too much force cannot be placed on this most necessary precaution against cholera.

Latrines must be carefully and frequently disinfected if used by patients suffering from cholera, even in its premonitory stages. It is necessary to bear in mind the fact that all cases of cholera do not pass on to collapse, and that the evacuations in the case of *cholerine*

[1] *Brit. Med. Journ.* October 8, 1892, p. 814.

are capable of causing the disease. An instance of this kind was reported as far back as 1849, in the case of a soldier who arrived at his house in the commune of Hamil from Paris, where cholera was prevalent. This man on reaching his father's house suffered from severe diarrhœa, from which he recovered; his youngest brother was constantly with this man, and on the day after his arrival the lad was seized with Asiatic cholera; the disease extended to other members of the family, and soon throughout the neighbourhood.

Measures to be Enforced in case of an Outbreak among Troops.—The measures at present enforced, on the outbreak of cholera among European troops stationed in the various cantonments of India, are much the same in principle as those adopted by the Marquis of Hastings in 1817. They practically consist in removing the men from the locality in which cholera has appeared, and placing them in such circumstances as will effectually exclude the possibility of their obtaining water or food of any kind contaminated with the germs of the disease. In fact, the bulk of the soldiers under these circumstances are marched off from the place in which some of their comrades have been attacked with cholera, into camps previously prepared for their reception, proper precautions having been taken to guard the drinking water from the possibility of being impregnated with cholera bacilli. Isolation of the sick, and the careful disinfection of their evacuations, are also enforced. I have already referred to the remarkable decrease in the mortality from cholera among our European troops stationed in Fort William which has followed the introduction of a pure water supply into that place, although surrounded, as they are, by a population from among whom Asiatic cholera is never absent (p. 405).

Principles of Prophylaxis.—So important is this subject, that although the following memorandum of Dr. Thorne contains much I have already insisted on, I am convinced the reader may with advantage study it, coming as it does from the head of the medical department of the Local Government Board. Dr. Thorne writes:—

> Cholera in England shows itself so little contagious, in the sense in which smallpox and scarlatina are commonly called contagious, that, if reasonable care be taken where it is present, there is almost no risk that the disease will spread to persons who nurse and otherwise closely attend upon the sick. But cholera has a certain peculiar infectiveness of its own, which, where local conditions assist, can operate with terrible force, and at considerable distances from the sick. It is characteristic of cholera (and as much so of the slight cases where diarrhœa is the only symptom as of the disease in its more developed and alarming forms), that the matters which the patient discharges from his stomach and bowels are infective. Probably, under ordinary circumstances, the patient has no power of infecting other

persons except by means of these discharges; nor any power of infecting even by them except in so far as these matters are enabled to taint the food, water, or air which people consume. Thus, when a case of cholera is imported into any place the disease is not likely to spread, unless in proportion as it finds, locally open to it, certain facilities for spreading by indirect infection. In order rightly to appreciate what these facilities might be, the following considerations have to be borne in mind:—First, that any choleraic discharge cast without previous thorough disinfection into any cesspool or drain, or other depository or conduit of filth, is able to infect the excremental matters with which it there mingles, and probably, more or less, the effluvia which those matters evolve; secondly, that the infective power of choleraic discharges attaches to whatever bedding, clothing, towels, and like things have been imbued with them, and renders those things, if not thoroughly disinfected, capable of spreading the disease in places to which they are sent for washing or other purposes; thirdly, that if, by leakage or soakage from cesspools or drains, or through reckless casting out of slops and waste water, any taint (however small) of the infective material gets access to wells or other sources of drinking water, it can impart to enormous volumes of water the power of propagating the disease. When due regard is had to these possibilities of indirect infection, there will be no difficulty in understanding that even a single case of cholera, perhaps of the slightest degree, and perhaps quite unsuspected in its neighbourhood, may, if local circumstances co-operate, exert a terribly infective power on considerable masses of population.

After giving advice as to the water supply and proper condition of the sewers, etc., Dr. Thorne goes on:—

It may fairly be believed that, in considerable parts of the country, conditions favourable to the spread of cholera are now less abundant than in former times; and in this connection the gratifying fact deserves to be recorded that during recent years enteric fever, the disease which in its methods of extension bears the nearest resemblance to cholera, has continuously and notably declined in England. But it is certain that in many places such conditions are present as would, if cholera were introduced, assist in the spread of that disease. It is to be hoped that in all these cases the local sanitary authorities will at once do everything that can be done to put their districts into a wholesome state. Measures of cleanliness taken beforehand are of far more importance for the protection of a district against cholera, than removal or disinfection after the disease has made its appearance.

Protective Inoculation.—The principles above referred to, simple as they appear to be, are effective against cholera if they are promptly and consistently carried out. It is quite certain, unless these principles are enforced, other sanitary arrangements, however perfect, will not stop the progress of the disease. We must, however, bear in mind that desirable as it may be that men and women should have pure water, air, and perfect sanitary surrounding, by far the majority of the human race are unable to command these conditions; and so when epidemic cholera appears among them a considerable number of them will contract the disease, and not

less than 40 per cent. of these will fall victims to cholera. The question therefore arises as to the possibility of protecting such a community from this danger by the inoculations of minute doses of the poison which produces cholera.

I have already referred to the evidence which seems to demonstrate the fact that the symptoms of cholera result from a poison produced by the cholera bacillus in the intestinal canal. Dr. Ferran, following up these observations, proceeded to make pure cultivations of this micro-organism in bouillon; he thus obtained an alkaloid which he injected into the circulation of human beings in minute doses, and he believes that he can thus render them immune for a time against cholera. Dr. E. O. Shakespeare visited Spain during the epidemic of 1885, and carefully investigated Dr. Ferran's practice and method of procedure in obtaining pure cultures of the cholera bacillus. He states:—

> From the Government statistics of cholera throughout the province of Valencia, it appears that among the villages invaded there were 62 attacks per 1000 of the population, and 31 deaths per 1000, which gives a mortality of 50 per cent. of those attacked. It appears, from analysis of the published official statistics of cholera, in twenty-two towns where inoculation was performed the inhabitants were divided as follows:—104,561 not inoculated, 30,491 inoculated; of the latter there were 387 attacks of cholera, or 12 per 1000, and 104 deaths, or 3 per 1000, the mortality of those attacked being 25 per cent.; of the former there were 8406 attacks, or 77 per 1000, and 3512 deaths, or 33 per 1000, being a mortality of those attacked of 43 per cent. It appears, therefore, that among the population of villages wherein anti-choleraic inoculation had been more or less extensively performed, the liability of the inoculated to attacks of cholera was 6·06 times less than that of the non-inoculated; whilst the liability of the inoculated to death by cholera was 9·87 times less than that of the non-inoculated.

Dr. Shakespeare adds:—

> It would seem that there is *prima facie* evidence sufficient to warrant the presumption, first, that the comma bacillus of Koch is the cause of Asiatic cholera in man; secondly, that there is an immunity following an attack of cholera; third, that an immunity of some duration may be established artificially by inoculation of the products of a pure cultivation of the cholera bacillus; fourth, that in the practice of these inoculations extensively among populations suffering greatly from cholera, an epidemic can be rapidly extinguished. The evidence, such as it is, seems to tend almost universally in this direction.[1]

The symptoms produced by the inoculation of the products of pure cultures of the comma bacillus into the circulation in minute doses are as follows:—Some three hours after the injection into both arms, the limbs become painful, and there is inability to use them,

[1] *Report on Cholera in Europe and India*, by Edmund O. Shakespeare, of Philadelphia, A.M., M.D., United States Commissioner.

great prostration follows, with a feeling of intense coldness, and an actual coldness of the extremities, headache, nausea, and frequently diarrhœa, with considerable general malaise; these symptoms increase up till about the twelfth hour after the inoculation, and continue for twenty-four hours, when they subside and gradually disappear. A second inoculation, made a fortnight after the first, produced little or no effect, or any symptoms beyond some local pain. The vaccine is to be kept in a properly constructed flask, so as to prevent the passage of extraneous micro-organisms from the atmosphere into the fluid. In fact, its preservation and mode of injection are similar to those employed for tuberculine, or by Pasteur in his inoculation for rabies. The dose used by Dr. Ferran in his inoculations was one cubic centimetre, into each arm, of a pure culture of the comma bacillus. Five days after the first inoculation, a second one may be used; but, as before stated, as a rule, it then has no effect beyond exciting local irritation at the point of puncture.

M. Haffkine has taken up the subject of vaccination against cholera in a most zealous and scientific manner. He is now engaged in anticholeraic vaccination in India. The progress of this work will be watched with the greatest interest. The reader is referred to the *British Medical Journal* of February 4, 1893, for full particulars as to the preparation and method of vaccination employed by M. Haffkine.

Pathology.

Intestinal Canal.—On opening the abdomen immediately after death from Asiatic cholera, supposing the person to have died during the stage of collapse, we find that the viscera lie back in a compact form deep in the abdominal cavity.

The stomach is empty, in some cases its mucous membrane is red and congested, small spots of ecchymosis may be observed on its surface. But this is by no means a constant feature after death in the collapse state of cholera; on the contrary, the mucous membrane in many instances, beyond a cloudy swollen condition of its epithelial lining, appears to have undergone no pathological changes.

The small intestines, as a rule, contain more or less fluid, which, if death has occurred within a few hours, in appearance resembles thin gruel. In the more acute cases of cholera the contents of the intestines are almost colourless; flakes of mucous, however, of a pale red colour are usually present. But in the majority of cases the fluid is stained by the colouring matter of the blood. Under the microscope, in many instances, a vast number, and in others

only a few, cholera bacilli may be seen, together with other forms of micro-organisms, and generally quantities of epithelial cells in which comma-shaped bacilli may be detected.

The mucous membrane of the small intestines throughout their length is more or less red, congested, and swollen. In some cases it is of a rose-red colour, with marked swelling of its epithelial covering, a considerable portion of this layer of cells being detached, producing the appearance of superficial ulceration, and sometimes of even diphtheritic changes. The ileum is commonly most affected, extensive lesions such as those I have referred to being found immediately above the ileo-cæcal valve. The solitary lymph follicles and Peyer's patches are of a greyish colour, their margins being surrounded by a zone of dilated vessels, among which hæmorrhages may frequently be seen. These latter appearances are noticed even when the rest of the mucous membrane and its epithelial cells are only slightly affected. But in those cases in which the mucous membrane of the small intestines is much congested, Peyer's patches are seen distinctly raised above the rose-red mucous membrane surrounding them.

Koch states that "on examining sections made of the coats of the intestines of persons after death from cholera, that in those cases in which the intestines by magnifying show the slightest changes, the bacilli had penetrated into the utricular glands of the mucous membranes of the intestines, and had caused there a considerable irritation, as the dilatation of the opening of the gland and the collection of granular circular cells in the interior of the gland showed. In many cases the bacilli had found their way behind the epithelium of the gland, and had multiplied between the epithelium and the glandular membrane. The bacilli had also settled in large numbers on the surface of the villi of the intestines, and had often penetrated into their tissue. In severe cases, which terminated in bloody infiltration of the mucous membrane of the intestines, the bacilli were found in very large numbers, and they did not then confine themselves to the invasion of the utricular glands, but passed into the surrounding tissue into the lower layers of the mucous membrane, and in some places right to the muscular skin of the intestine. The intestinal villi were also in such cases penetrated by bacilli. The chief seat of these changes is in the lower part of the small intestine. If this discovery had not been made in perfectly fresh corpses, one could have made little or no use of it, for the influence of putrefaction is able to bring about similar vegetation of bacteria in the intestines."

The large veins of the abdomen, as well as those supplying the coats of the intestines and the mesentery, are gorged with blood.

The mesenteric glands are enlarged, soft, and infiltrated with a whitish granular matter.

Other Organs.—Klebs observes that marked changes are noticed in the kidneys after death from cholera; they present an abnormally pale colour of the cortical substance. On examining stained sections (gentian violet), the tortuous tubes are not stained, or they readily give up their colour to alcohol. The nuclei have disappeared, or at least they contain only traces of the material which stains. The cellular substance is greatly swollen, and is slightly cloudy; if death occurs at the height of the morbid process, the epithelium will be found to fill the lumen of the tubules. This process, Klebs states, is a "coagulation necrosis" which attacks the epithelia, excited in all probability by a toxic substance affecting the cell protoplasm.

It seems quite possible that the arterial thrombi and small hæmorrhages seen in the pericardium, intestines, and other parts of the body are also caused by alterations in the cells lining the walls of the vessels, and the serous and mucous membranes of the body.

The spleen is small and anæmic.

The liver appears to be shrunken, its portal veins being full of dark viscid blood. The gall-bladder is generally distended with bile.

The urinary bladder is empty.

The lungs, like the liver and spleen, are below their normal weight, and in the greater number of cases are collapsed and found lying back against the spine. On section they appear dry, containing but little blood, and that is confined to the pulmonary arteries and their branches; the capillaries and veins of the lungs are empty.

In some instances there is more blood in the minute vessels, and the lungs are then of a darker colour.

The heart, as a rule, is found distended with dark blood on the right side, its left side being empty. The jugular veins and the vena cava, together with the coronary veins, are full of blood, but the aorta and other arteries are empty, with the exception of the pulmonary arteries. The blood is of a dark bilberry-juice colour, the colouring matter leaving the corpuscles and tinging the serum; it still retains its power to take up oxygen and to give off carbonic acid, but it passes so slowly through the pulmonary vessels that only one-third of the usual quantity of carbonic acid is given off from the lungs during life, and but little oxygen is taken in.

The veins of the encephalon are full of dark blood, otherwise no pathological changes are to be found.

Cholera bacilli are not present either in the blood, or in any of the tissues of the body except the intestines.

Action of the Virus on the System.—With reference to the poison which we believe the cholera bacillus produces, several theories have been advanced to explain its action in connection with the pathological appearances found after death. Among these theories, that advocated by Sir George Johnson takes a prominent position, and by many pathologists is held to be best capable of accounting for the symptoms and the post-mortem changes found after death from cholera.

Sir George Johnson states that in whatever way the poison was introduced into the body that it entered the circulation, and there probably underwent increase by the conversion of some blood constituents, which were then excreted through the mucous surface of the alimentary canal, and were ultimately expelled by vomiting and purging; and thus the patient recovered. In the more severe cases collapse occurred. That this was not the result of the liquid discharges from the system was shown, Sir George Johnson considered, by the fact that, in the most rapidly fatal cases there was rather an inverse than a direct ratio between the degree of collapse and the amount of the liquid discharges. A complete arrest of the discharges during collapse was a sign of fatal import; while, on the other hand, there was a continuance of the discharges, in a greater or less degree, during recovery from collapse. The effect of various and opposite modes of treatment, he believed, was also inconsistent with the theory that the worst symptoms were the result of the liquid discharges. Those methods of treatment appeared to Sir George to have been most successful which had been least repressive in their tendency. The sudden onset and the rapid passing away of extreme collapse were inexplicable by copious exhausting discharges. That the main and essential cause of choleraic collapse was, he thought, due to a greatly impeded circulation through the lungs, as was shown by the appearances found after death, and by the explanation thus afforded of the chief symptoms of collapse. When the chest was opened soon after death in collapse, the left cavities of the heart were found nearly empty; while the right cavities, the pulmonary artery, and the systemic veins were distended with blood. Sir George Johnson holds that extreme contraction of the pulmonary arterioles was the only probable explanation of this arrest of the circulation. The thickening of the blood was the consequence and not the cause of the impeded circulation. The small stream of blood through the lungs during collapse, with the resulting defective oxidation, explained the suppression of bile and urine; while, in the case of nursing women, the mammary secretion, which was not an oxidised product, continued. The marvellous temporary relief afforded by injecting a hot saline solution into the veins, he attributed to the warmth of the liquid relaxing the arterial spasm, and thus allowing the blood to pass on; while the beneficial effect which had often resulted from venesection was explained by its lessening the distension of the right cavities of the heart, and so increasing their contractile power. Other causes of arrested pulmonary circulation, such as pulmonary embolism, exclusion of air, and the inhalation of nitrous oxide gas, produced much the same train of symptoms and pathological conditions as those produced by the poison of cholera.

With reference to this theory, I am aware that one of our earliest observers of cholera in India, Mr. Scott, states that in

some cases there are no spasms, hardly any purging, but "a mortal coldness with arrest of the circulation from the beginning; and the patient dies without a struggle." But Mr. J. Annesley, a most talented Indian physician, commenting at the time on this passage from Scott's works, remarks: "This is a type of the disease which I have never seen." After nineteen years' residence in the home of cholera, I must endorse Annesley's opinion. I have never met with a case of cholera such as Mr. Scott describes; like Mr. Annesley, in all the patients I have attended, the disease, even in the worst cases, has lasted ten or twelve hours, and been attended with much more marked symptoms than those referred to by Mr. Scott. Further, I am convinced that a large majority of the officers of the Indian Medical Service would join me in asserting that they had seen many cases in which, to all appearances, they had stopped an attack of cholera in its early stages by means of opium and acetate of lead or sulphuric acid, which checks the elimination of the poison through the intestines. We are bound also to bear in mind the fact, that after death from cholera there is found to be great loss in the normal weight of the lungs, the spleen, liver, and other organs. If the cause of death depended on obstruction to the circulation through the lungs, the blood must have accumulated in other parts of the body; but they all lose in weight, and this loss is due to dehydration of the blood and tissues.

It would seem possible that the condition of the nuclear matter of the cells of the kidneys and other parts of the body, described by Klebs, may be caused by dehydration of the blood and tissues. For we must remember that in Asiatic cholera there is not only a very rapid and copious outflow of serous fluid from the intestines, but absorption of fluid through the stomach is prevented. There is an actual diminution of water in the blood and tissues of the body, and fluid cannot be absorbed by the stomach to replace that which has drained away. A condition of this kind, lasting for some hours before death, must affect the integrity of the protoplasm of all the cells of the body; for it is living protoplasm which has continued to exist for from ten to forty-eight hours deprived of water to a great extent. This living protoplasm is something quite different from dead matter; but its properties must alter to a greater or less extent, although the body of which it forms a part may live, if deprived of water for some hours. It is quite possible that the chemical changes going on in protoplasm in these conditions, may account for its want of power after death to be coloured by staining agents, which in other circumstances is a characteristic feature of this kind of organic matter.

Symptoms.

Onset and Early Stage.—Asiatic cholera is most deadly on the first outbreak of an epidemic, and new-comers into an affected locality are more frequently attacked than persons who have been living in the midst of the disease for some time.

Cholera may come on without premonitory diarrhœa (cholerine), and the patient quickly passes into a state of collapse, too frequently terminating in death within thirty-six hours.

In the greater number of cases, Asiatic cholera commences with diarrhœa; the stools are watery, copious, and frequent; they are coloured with bile, and have an alkaline reaction. The patient complains of extreme lassitude, and a feeling of indescribable sinking at the "pit of the stomach"; he is very thirsty, his tongue is white and clammy, there is great nausea, and not infrequently vomiting. If judiciously treated, many patients recover from this condition; the stools, however, in cholerine contain the infective matter of cholera, and the patient, therefore, and the evacuations should be treated on the principles laid down in the previous section. Should the disease progress, the stools alter in appearance, resembling water in which rice has been boiled; these liquid evacuations are passed very frequently; and the patient vomits water and everything else which he may swallow. He also suffers from excruciating pain caused by cramps in his arms, legs, and abdominal muscles; he is extremely restless, throws off the bedclothes, and complains of feeling hot, although the surface of his body feels cold. Dr. E. O. Shakespeare states that if the thermometer is carefully applied to the axilla for ten minutes, it will indicate a rise of temperature to 100° or 102° F. The temperature of the rectum is always three and even four degrees above its normal point. In this stage of cholera the patient's pulse is rapid and small, his respiration is quick and shallow, his voice is husky and extremely weak; the eyes deep sunk in the orbits, and the features are pinched. The skin of the body not only feels cold but is inelastic, that covering the hands and feet is wrinkled and of a purplish hue. The duration of this stage of cholera is uncertain; it may last for two or three hours, or may continue for a day or two; but so long as the pulse can be felt at the wrist there is good hope of reaction and recovery.

Algid Stage.—Should the disease progress the patient passes into the algid or collapse stage of cholera, which is characterised by continued vomiting and rice-water stools, but to a less degree than in the previous stage; in fact, the blood and tissues of the body are

well-nigh dehydrated, and if the patient is placed in a large bath it is not easy to keep his body under the water, it floats like a cork. In this, the collapse stage of cholera, the lividity of the surface of the body is increased; the integument having a doughy, inelastic feel, the skin of the tips of the fingers and toes are blue and wrinkled. The eyes are surrounded with a dark areola, and the eyeballs deeply sunk in the orbits; the voice cannot be raised above a whisper, the breathing is very rapid. The pulse can hardly be felt, and the heart's action is very weak. No urine is passed. The patient is still very restless, and his constant cry is for water, and that some one should rub his arms and legs to relieve the terrible cramps. Reaction may after twelve or more hours supervene, but the condition of the patient is alarming, and too frequently terminates in death.

The torpid stage is one through which many cholera patients pass before death: it seldom lasts for more than a few hours. The purging and vomiting almost cease, and the patient remains in a semi-comatose condition, with his eyelids half closed, the conjunctivæ are injected; the pulse cannot be felt at the wrist, and the respiration is laboured; the skin is covered with a cold clammy perspiration. The suppression of urine continues.

Stage of Reaction.—Reaction may, as above remarked, come on at any stage of cholera, though very seldom indeed after that last described. The intensity of the cramps and the vomiting and purging diminish, and the pulse and respiration are more natural; above all, the patient can quench his thirst and obtain some refreshing sleep; after, it may be thirty-six hours or so, he passes a small quantity of urine, and after a few days may be up and walking about; the rapidity with which recovery takes place is in many cases very remarkable. Convalescence, however, may be thwarted by various serious complications: of these the most important are suppression of urine, gastritis and enteritis, pulmonary congestion, a clot in the right side of the heart or pulmonary arteries, meningitis, sloughing of the corneæ, abscesses over the surface of the body and hæmorrhage from the bowels, and roseola-choleraica.

Treatment.—In the early stages of Asiatic cholera, we may frequently stay the further progress of the disease by opium combined with acetate of lead, or with dilute sulphuric acid. Twenty drops of laudanum with three grains of acetate of lead, or twenty minims of dilute sulphuric acid should be given, and repeated every hour for three doses (in the case of adult patients) if the purging continues. At the same time, the patient should be kept in bed, and a large mustard poultice applied over his abdomen. If ice is

available, he may suck as much of it as he pleases, but it is unwise to allow him to drink much water or any other fluid.

Supposing, however, such treatment does not succeed, or that on first seeing the patient he has passed into the second stage of the disease, we should still prescribe the opium and acetate of lead, or sulphuric acid as above; mustard poultices must also be employed, and the patient kept in bed with plenty of ice to suck. Having gone through a severe attack of cholera, I can testify as to the unspeakable relief which ice affords to a person passing through an attack of this terrible disease. In my own case the onset of the disease was so sudden, and the vomiting so incessant that everything I took was immediately rejected, and consequently it was useless taking medicine; but I chewed and swallowed pounds of ice, to my infinite comfort. I think water, though urgently demanded by the patient, should be refused. I would restrict the opium to three grains; it is unwise to give more, although we may be well-nigh certain that much of it has been vomited. If this treatment does not stop the progress of the malady, and the vomiting is very severe, a single dose of twenty grains of calomel will sometimes relieve this symptom. A mixture may be added, each dose of which contains two grains of acetate of lead and fifteen drops of dilute acetic acid, to be taken every second hour; and fifteen drops of dilute sulphuric acid in water every alternate hour, so that the patient should take a draught of first one mixture, and then the other, every hour. In this way the alkaline stools become acid, and perhaps destroy the cholera organisms in the intestinal canal. However this may be, these acids seem to be beneficial in the treatment of cholera.

The cramps are best relieved by hand friction. Hot water bottles should be applied to the feet and sides; if the cramps are very severe the inhalation of ether is often a great relief, the more so, I think, after the stools have been rendered acid by medicines such as those above referred to. If the disease has reached the stage of collapse, there is but little we can do for the patient in the way of medicine, but he may continue to take the acid draught. Friction to the limbs will still relieve the cramps, and ice quench the distressing thirst; a small quantity of iced water may be cautiously given from time to time, provided it does not increase the vomiting.

I believe that alcohol is positively harmful in any stage of cholera. When reaction comes on we must be careful not to fall into the error of overfeeding the patient under the mistaken idea of supporting his strength; he will not die of exhaustion, if small

quantities of milk and arrowroot are administered frequently, for two or three days, together with warm beef tea enemas. But enteritis may certainly be induced if food other than the simplest and in the smallest quantities are allowed. The patient requires rest and the most careful nursing after a severe illness like cholera.

By treatment of this kind the kidneys and other organs of the body gradually recover their functions, and in this way suppression of urine and other complications are best guarded against. If suppression of urine or any of the other complications referred to supervene on an attack of cholera, they must be treated on the same principles as are applicable to these various maladies occurring in other circumstances.

I might fill many pages with a description of the various remedies that have from time to time been advocated for the relief of cholera. Many of these drugs would seem to have given much promise of success, but one after another they have so far failed to take their place as recognised agents in the treatment of this disease. Our hope, for the present, lies in an endeavour to stop its progress in the early stages of the disease, and, above all, to master the cause which gives rise to it, and thus endeavour to preserve those committed to our charge from its deadly influence.

Professor Rumpf, head of the Eppendorf Hospital at Hamburg, one of the best institutions of the kind in the world, has published, in the *German Medical Weekly*, an article on the treatment of cholera, and his experience of the most usual methods employed in about three thousand cases, during the present epidemic of 1892. His experience of drugs has been very unfavourable. Of the various preparations, salol, cresline, creosote, muriatic acid, lactic acid, cresol, chlorine water, sulphuric acid, and morphia, not one, he says, was of any avail. Clysters did good in the less severe cases, and after the crisis was over; these clysters consisted of a quart or more of hot water, containing two drachms of benzoate of soda, or thirty grains of tannic acid. But the best results were obtained by doses of calomel in doses of eight grains, to be given by the mouth, and to be repeated every five hours, in five grain doses. Warm baths and subcutaneous injections of solutions of common salt, and of camphor and morphia, have proved the next best remedies. Dr. Rumpf concludes as follows: "There is no specific treatment for cholera yet known. The discovery of a remedy which would annihilate the virus in the body, without damaging its functions would be an essential step forward in the treatment of cholera. Modern bacteriology will, perhaps, help us on in this direction."

CHAPTER XI.

LEPROSY.

BY C. N. MACNAMARA, F.R.C.S.

History.—The history of leprosy is confused in consequence of what we now recognise as distinct and separate diseases having been described under the term leprosy. For instance, leucoderma and elephantiasis have both been included under the heading of leprosy; the Bible mentions a person as "a leper as white as snow," in other words, he suffered from leucoderma. Again, P. Ægineta observes: "If cancer, which is, as it were, an elephantiasis in a particular part, is ranked among the incurable diseases by Hippocrates himself, how much more is elephantiasis incurable, which is, as it were, a cancer of the whole body? Wherefore, those who are already overpowered by the disease must be abandoned; but when the affection commences so as that none of the extremities have fallen off, or the surface of the body become ulcerated, nor the hard swellings appeared, and the face merely looks foul, but not altogether unseemly, we must attempt a cure." The disease here referred to as elephantiasis was evidently a form of leprosy. Isidorus, of Saville, tells us that they called it elephantiasis because it was a mighty disease; sometimes leontium or morbus leoninus, from the supposed resemblance of the eyebrows to those of a lion. Dr. Adams, in his commentary on these observations of Ægineta and Isodorus, remarks that the Greek translators of the Arabian physician Avicenna rendered the Arabic word *juzum* or *judam* by lepra; but unfortunately they called quite a different affection "elephantia," from its resemblance to the leg of an elephant; and the Greeks, who had been accustomed to designate leprosy as elephantiasis, came to mix up elephantiasis with leprosy. To make matters worse, of late years lepra, a form of skin disease classed by Willan under the order of *Squamæ*, has also been described as leprosy.

Out of all this confusion of terms, pathology has at last led us to recognise the fact that leprosy is the effect of a micro-organism,

which, if it becomes located in the skin causes tubercular leprosy; but if its action is confined to the nerves, it produces "nerve leprosy"; frequently these two forms of disease exist in the same individual, in which case the person is said to suffer from "mixed leprosy."

Leprosy must have existed in Egypt from the time of Moses. Manetho, writing 260 B.C., states that a vast number of Jews suffered from this disease, and were in consequence expelled from Egypt. In India, Susruta, 400 B.C., describes leprosy as being common among the inhabitants of Hindostan; the early Chinese authors also refer to the disease. Leprosy, though of rare occurrence, was known in Italy as early as A.D. 53, and before that time had been described by Greek and Arabian physicians.

In England a leper or lazar-house was established at Canterbury in the year A.D. 1096. The disease would appear to have made rapid strides both in this country and in the West of Europe during the time of the various Crusades; and some of our best authorities are of opinion that the evidence is strongly in favour of the idea, that the Crusaders had much to do in disseminating the disease throughout the regions from which they came, and to which many of them returned after visiting Palestine. It is certain that between the time of the foundation of the first leper-house in England and the year 1472, no less than 112 similar institutions were built in this country. Michaud, in his *History of the Crusades*, states "that the historians we have followed are silent as to the ravages of leprosy among the nations of the West; but the testament of Louis VIII. (1226), an historical monument of the period, attests the existence of 2000 hospitals for lepers in the kingdom of France alone." The total number of leper-houses in Europe was estimated by Matthew Paris at 19,000.

Sir J. Simpson observes, according to the record of Edward III., that king sent, in 1346, "a commandment under his Great Seal to the Mayor and Sheriffs of London, willing them to make proclamation in every ward of the city and suburbs, that all leprous persons within the said city and suburbs should avoid within fifteen days, and that no man suffer any such leprous person to abide within his house, upon pain to forfeit his said house, and to incur the king's further displeasure. And that they should cause the said lepers to be removed into some outplaces of the fields, from the haunt and company of all sound people."

The leper was not looked upon in the eye of the law alone as defunct, for the Church also took the same view and performed the solemn ceremonials of the burial of the dead over him on the day on which he was separated from his fellow-creatures and consigned to a lazar-house. He was from that moment regarded as a man dead amongst the living, and legally buried, though still breathing and alive. The ritual of the French Church retained till a late period the various forms and ceremonies to which the leper was subjected on the day of his living funeral.

A priest, robed with surplice and stole, went with the cross to the house of the doomed leper. The minister of the Church began the necessary ceremonies by exhorting him to suffer, with a patient and penitent spirit, the incurable plague with which God had stricken him. He then sprinkled the unfortunate leper with holy water, and afterwards conducted him to the church, the usual burial verses being sung during their march thither. In the church the ordinary habiliments of the leper were removed; he was clothed in a funeral pall; and while placed before the altar between two trestles, the *Libera* was sung, and the mass for the dead celebrated over him. After this service he was again sprinkled with holy water, and led from the church to the house or hospital destined for his future abode. A pair of clappers, a barrel, a stick, cowl, and dress, etc. etc., were given to him. Before leaving the leper, the priest solemnly interdicted him from appearing in public without his leper's garb; from entering inns, churches, mills, and bakehouses; from touching children or giving them aught he had touched; from washing his hands, or anything pertaining to him, in the common fountains and streams; from touching in the markets the goods he wished to buy with anything except his stick; from eating or drinking with any others than lepers; and he especially forbade him from walking in narrow paths, or from answering those who spoke to him in the roads and streets unless in a whisper, that they might not be annoyed with a pestilent breath, and with the infectious odour which exhaled from his body; and last of all, before taking his departure and leaving the leper for ever to the seclusion of the lazar-house, the official of the Church terminated the ceremony of his separation from his living fellow-creatures, by throwing upon the body of the poor outcast a shovelful of earth, in imitation of the closure of the grave.

From the year 1420, and even before that date, there had been a decided diminution in the number of lepers in England. A Commission appointed in the reign of Edward IV. (1470) to inquire into the matter, reported that there were very few lepers left in any of the lazar-houses. The disease continued to linger on in the Shetland Islands[1] until the end of last century, but practically it had disappeared from England before Henry VIII. came to the throne. In Italy, in like manner, leprosy had almost ceased to exist by the year 1510, and somewhat later in France. In the sixteenth century the disease had become so rare in Denmark that the leper-houses were abolished; but it continued to prevail in Sweden until the end of the eighteenth century. Whilst the disease was thus dying out of Europe, lepers everywhere being treated by isolation in the manner above described, it has continued, and still prevails, over the greater part of Asia, where no such system of isolation has ever been enforced.

Dr. G. Thin, in his work on leprosy, published in 1891, p. 42, observes that "it is clearly established that leprosy was introduced into Italy about the time of Christ, and that from Italy it spread

[1] Dr. Edmonston met with a case of leprosy in Shetland in 1809, and Dr. Broadbent exhibited to the Medico-Chirurgical Society of Edinburgh, on the 6th June 1855, a case in a young man from the Hebrides.—EDITOR.

(following the Roman armies and main routes) into countries in Northern and Western Europe. Within a few centuries of its first spreading into these countries, it had multiplied to such an extent as to have inspired the whole of Christendom with horror and fear. The disgust and terror which it evoked, roused the whole populations of these parts to drive the unfortunate leper from their midst. The genius of Christianity, fortunately, was true to itself, and tempered this act by providing "lazar-houses" for the reception of the unfortunate outcasts. The leper everywhere was met with the cry of "unclean"; and to touch him was considered an act only supernatural faith could inspire. This was followed by another circumstance of enormous importance to us in the present day, who have to deal with countries in which leprosy is now as great a scourge as it was in Europe at that time. With extraordinary rapidity, considering the nature of the infirmity, it began to disappear simultaneously with the adoption of the strict measures that were put in force, the disappearance being as rapid and complete as the onset of the disease amongst the populations had been swift and intense."

Present Distribution.—*Sandwich Islands.*—It is during the invasion of new countries and nations by obscure diseases, that some of the laws governing the spread of these diseases may be best studied, because a starting-point with regard to time and place, at least approximately, may be fixed, from which the further growth and spread of the disease can be traced. These conditions given, the laws by which the dissemination of the disease takes place become marked. It is on this account, I think, the following communication received by me from a German physician, Dr. Hillebrand, formerly of Honolulu, is a document worthy of our best consideration. Dr. Hillebrand came to India in 1865 to study the character of leprosy; I then made his acquaintance, and on his return to Honolulu he sent me the following account of the introduction and spread of the disease in that island. Dr. Hillebrand states that—

> In the Sandwich Islands, where I have been living ever since 1851, practising the profession of medicine, and to a great extent among the natives of the country, leprosy was unknown before 1859, and after close scrutiny cannot be traced further back than the year 1852, or at the most 1848. A recent census, taken by the Government, established the number of lepers to be about 230, out of a population of 67,000 natives, or nearly $3\frac{1}{3}$ in 1000. As I have good reason, however, to believe that only cases with marked tubercular development have been reported, the simply anæsthetic form not being generally recognised as being of leprous character, this estimate falls rather short of, than exceeds, the reality, which may safely be estimated at 4 in every 1000. The character of the disease was first recognised in August 1859, shortly after the establishment of the Queen's Hospital and Dispensary. It then occurred to me that I had met

with similar cases occasionally, but rarely before, the first of which I could recollect as far back as 1853. Further inquiry among the natives at large brought to light that a few had been observed in 1852 and 1851; and an old chief, well versed in the history of his country and in everything pertaining to his countrymen, referred the first case known to him to the year 1848. In 1859, when I first brought the existence of lepra amongst our people to the notice of Government and the public, only a few cases became known, but with every subsequent year the leprous patients presenting themselves at the public dispensary increased in number, until during 1864 and 1865 it was considered of quite ordinary occurrence that lepers should apply for relief. It is worthy of notice that, soon after the character of the disease became known, the natives began to call it "mai pake," the Chinese disease. Whether this name, derived from a belief that the disease had been imported through Chinamen, of whom there have been a considerable number settled at the island for years, or if it simply owed its origin to the circumstance that they learned from the Chinamen that the disease was common in China, I have not been able to ascertain.

Here, then, we have the important fact of the leprous disease introducing itself amongst a clean nation, spreading slowly at first, so as not to attract attention for many years, but multiplying faster as years roll on, until, after the lapse of at most seventeen years, it has invaded almost every district of our island group, alarms the people, and seriously occupies the attention of the Government and Legislature, who, during the Session of 1865, voted the comparatively large sum of 30,000 dollars for the establishment of a secluded hospital and a leper colony in an isolated locality on one of the smaller islands; in fact, it is regarded as a national calamity. And mark well, in all this, hereditary taint, from the nature of the case, has no share, or, if any, only a most subordinate one. I can only remember a single child under the age of six years, among the great number of cases which have come under my observation. Only one instance, where father and child were affected, can I recall to memory, and in that case the child was born clean, before the disease had broken out in the father.

The question next arises—Have changes taken place in the habits or ways of living of the people for the worse? Do they live on poorer and less wholesome food now than formerly? Are they clad more scantily? Do they live in worse constructed houses?—in a word, are they exposed to the inclemencies of the weather? Are they borne down by oppressive taxation, by forced labour, or anything tending to lower their vital forces, and thereby to prepare a soil well adapted for the spontaneous generation of such a disease? Quite the reverse; on all these points they are better off now than at any time before. Their food is the same which it used to be—viz. a paste formed of the tuber of the *Colocasia esculenta*, a tuber richer in gluten than any other. The country is well stocked with cattle, sheep, pigs, fowl, fish, etc., and animal food is within the reach of every one; for labour is in great demand, and highly paid. While, in former times, a girdle round the loins constituted their whole wearing apparel, now they are decently dressed like Europeans; their former dark and damp straw-huts are rapidly making room for pretty wooden structures, raised from the ground, and well aired. The climate is perhaps the finest in the world, the thermometer ranging between a minimum of 60° and a maximum of 88° F., the trade winds blowing uninterruptedly during summer; malaria is all but unknown. They live under a free constitutional government, and taxation is light. It is true syphilis has, as in most Polynesian tribes, sapped the

life of the nation, and is the main cause of the lamentable decrease of the population; but syphilis and the decrease of population were going on long before the appearance of leprosy, and are making less progress now than before. And, moreover, although lepra invading a body tainted with constitutional syphilis, or having syphilis implanted upon it, assumes a more virulent character, it has been found impossible to make out a specific affinity between the two dyscrasies.

It is also a notable circumstance, that a considerable number of those affected, and some even of the worst cases, belong to the better class of natives, who are well off in every respect.

As to the mode of diffusion over the group, I have been able to gather a few important facts. The first leper seen by me in 1853 lived then in a thinly populated district of the island of Oahu, about twenty miles from Honolulu, in a small village near the sea. When, in 1861, I made inquiries about this man, I learnt from the most trustworthy source that he was now in a far advanced state of the disease; and that in his immediate neighbourhood six other persons had been taken with it. The same thing was observed in the district of Northkona Hawaii, where, towards the end of 1864, about seven cases became known, six of which were reported to have contracted the disease in the village of Kaslua, the tax-collector of which place had, for several years, been the only leper in the district. It must be observed here, that the natives are of very social disposition, much given to visiting each other, and that hospitality is considered as a sacred duty by them. Honolulu, the principal seaport and the capital of the kingdom, of course contributes the largest number to the official lists, while one or two of the remotest districts of Hawaii, which have but little intercourse with the rest of the group, were at the time that the census was taken, yet exempt from the disease. With the patients presenting themselves at the dispensary of the Queen's Hospital, I have made it a rule to ask to what cause they severally attributed the origin of their disease. About one-fourth avow contact with other lepers as the cause, a proportion which may be considered high, considering the shortness of time that the disease has been known, and the long term of incubation, during which the poison must lie dormant in the body, before it manifests itself.

In one family I heard of, a brother, a sister, and all individuals between 14 and 35 years suffered from an hereditary taint. It is well to remark that all these observations refer to tubercular leprosy, which, in an overwhelming majority of cases, has been found combined with anæsthesia, either in the extremities, or in the affected parts themselves, and generally associated with squamous eruptions of the skin—psoriasis. The tubercular affection does not confine itself to the cutis, but can be followed up the nares, producing ozæna, and to the palate and epiglottis, causing sometimes death by laryngeal phthisis. It appears on these mucous membranes under the form of small lenticular or pisiform knobs, which undergo a gradual ulcerative absorption, but never form large or deep corroding ulcers, as does syphilis, from which also the cicatrices are entirely distinct. Simple anæsthesia of particular nervous provinces in the forearm, particularly the ulnar, with contraction of one or more fingers, but without any ulceration of the affected skin, I have occasionally observed also, but their leprous character was not fully acknowledged. Since I visited China and India, however, all doubts on that point have disappeared from my mind.

Without indulging in loose speculations about the nature of the con-

tagion, supposing such to exist, I believe myself borne out by facts when I attribute to it the following characters:—

1. It must be of a fixed nature, either solid or liquid, not diffusible through the atmosphere. 2. It has an unusually long period of incubation. 3. It cannot take root in every constitution; or in other words, some men possess immunity from its attacks.

The very extensive prevalence of leprosy in the Hawaiian Islands can no longer be denied, and it constitutes a very serious problem for the Government of that kingdom. The mere expenditure in money annually reaches a very considerable sum, amounting probably to not much less than 5 per cent. of the total revenue. The evil has attained such large proportions that only by the most rigid enforcement of the law compelling the segregation of lepers can it be combated; this appears to be now fully recognised by the Board of Health, but it was not always so, and it is impossible to avoid the conclusion that the laxness at one time permitted has worked an amount of evil irreparable in this generation. In 1868 official returns gave the number of lepers as 274, and subsequent experience renders it almost certain that this enumeration was rather over than under the mark. This statement is grounded upon the fact that of 368 persons recently sent to the Medical Board of Examination as probably lepers, only 304 were declared to be undoubtedly leprous—that is to say, only 82 per cent. of the supposed lepers were without doubt suffering from the disease. Of the remainder, 22 were certainly not lepers, while as to 42 there was some doubt. At the present time, there are 749 lepers at Molokai, and over 600 still at large in the other islands, so that the Hawaiian kingdom cannot contain far short of 1400. Until quite recently the number at large was very much greater, for between July 1, 1887 and March 31, 1888, no less than 326 were, after careful examination by the Board above-mentioned, which consists of three physicians selected on account of their special experience, consigned to Molokai. The cases are not uniformly distributed, and in some localities the proportion is very large; thus in one valley of the island of Kauai over one-eighth of the population were lepers.

British India.—According to the Census of 1881, out of a population of 216,679,329 people under our rule, and that of feudatories, there were 128,089 persons affected with leprosy. These figures, however, so far as leprosy is concerned, are probably rather less reliable than those would be, if we employed our police constables in this country, on a certain day of the year, to ascertain how many people in England were suffering from eczema. The Indian police have no more definite idea of what leprosy is than our constables have of eczema, and have infinitely less intelligence and common sense. We may safely assert, however, that leprosy is common among the inhabitants of all parts of Hindostan; in some districts it is more prevalent than in others, but all are more or less infected. There are no legal restrictions in India against lepers associating with healthy people, and one sees all over the country, especially in the larger towns, lepers moving about among the people, or sitting by the roadside begging; some of those miserable creatures have suffered

from terrible mutilations of the face and other parts of the body, the result of this disease. The native customs were stricter in this respect before we obtained possession of India, and altered the laws and many of the customs of the country. How far these changes have tended to cause the extension of the disease in India we cannot now determine; but I concur in the opinion of many medical officers conversant with the subject, that leprosy has spread among the inhabitants of India slowly, but surely, of late years.

China.—Leprosy exists throughout the whole of the empire of China; the disease is considered to be contagious, but lepers are often found living with their families during its early stages.

In Havana a lazar-house was founded in 1861, and at the present time the average number of patients is about 90. During the last twelve years 216 lepers have been admitted into the hospital. The proportion of lepers to the population of the island is 3·75 per 10,000, excluding the Chinese; among this class of the population it is said that 94·34 per 10,000 are lepers; the proportion of men to women affected is as 5 to 1. Leprosy was diminishing in Cuba till the Chinese immigration began into that place.

In Russia, especially about Riga, leprosy has increased within the past few years; in 1887 Dr. Bergmann discovered 37 lepers in the town, and 21 in the neighbourhood. There are now over 100 lepers in this locality. In and around Dorpat there has been a marked increase of the disease within the past few years. Professor Münch collected the history of 800 lepers in Russia (European), and expressed the opinion that, by means of isolation, in thirty or forty years the country might be completely freed from the disease. In the Caucasus, up to 1841, there was a marked increase of the disease; but from that time, segregation of lepers having been enforced, the disease has much diminished.

Leprosy is met with on the west coast of Norway and in Iceland; in certain provinces of Spain and Portugal, and in the Riviera and Sicilly. It exists along the Caspian and Black Seas, in the delta of the Volga, and also in the islands of the Levant. It is common in Egypt and the North African States; in East, West, and Central Africa, Madagascar, Mauritius, Réunion, St. Helena, Madeira, Canaries, and Azores. It prevails also in Japan, Borneo, and Siam, and in Brazil, Central America, the West Indies, South America, Mexico, and a few cases are met with in New Brunswick. It has been observed among Chinese immigrants in the United States as far east as Chicago, and in Queensland.

Leper hospitals exist in several of the West Indian colonies, as

well as in various parts of Hindostan, in Mauritius, Singapore, Colombo, the Cape and Honolulu, in Canton, Java, and in various localities in Asia Minor.

Cape of Good Hope.—Leprosy has been on the increase for some years past among the inhabitants of South Africa; and also in New South Wales; and both these colonies have lately passed Acts for the isolation of lepers. The enforcement of legal enactments against lepers is of the greatest importance at the present time, not only from an historical, but also from a practical point of view. In the *British Medical Journal* for 25th June 1892, a copy of a *Gazette Extraordinary*, dated 18th May 1892, issued by the Government of the Cape, may be found. The editor of the journal remarks (p. 1427) that this *Gazette* contains the official promulgation of—

"The Leprosy Repression Act, 1884," and a statement of regulations made under the Act. The object of the Act is thus stated: "To check the spread of the disease known as leprosy." In the first paragraph of the Act there is a very important statement; and its importance is not lessened, but is rather increased, by the fact that it has recently been asserted that leprosy tends to diminish with the spread of civilisation, and, consequently, of sanitation, in countries prone to this disease. This is the statement: "Whereas the disease of leprosy is prevalent in this colony, and has lately been spreading, and continues to spread . . ." Here, then, is an official announcement of the fact that leprosy "continues to spread" at the Cape of Good Hope. The Act provides that when it is certified by any "duly qualified medical practitioner," and by a "field cornet," or justice of the peace, that any person is a leper, "and that the fact of such person being at large is likely to spread such disease, the Governor (of the colony) may, by warrant under the hand of the Colonial Secretary or Under Colonial Secretary, order that such person shall be removed to such asylum or hospital as he shall appoint, to be there detained during the Governor's pleasure, and kept apart from contact with all other inmates of such asylum or hospital who are not afflicted with the same disease. Provided always that every such person, while so detained, shall have the liberty and privilege of seeing his friends and legal advisers at all reasonable times under such regulations in force for the time being as the Governor may approve in that behalf." The Act also gives power to compel the keeping of male and female lepers in separate asylums; provides for the maintenance out of the colonial revenues of all pauper lepers, and the making by the "superintendent or keeper" of the hospital or asylum of a special agreement with any well-to-do leper which would require such a leper to pay his own expenses while he is detained in the asylum; prescribes the payment of fees to medical men employed by the proper authorities under this Act; and the giving to the local authority, or to any resident magistrate, by all district surgeons and medical officers, when they are requested so to do, of "any information which may be required in regard to the disease referred to in this Act. . . . Power is given to the Governor to at any time make, alter, and amend such regulations as he may deem to be advisable for the better and more effectually carrying out the provisions of this Act."

The Governor, with the advice of the Executive Council, has made,

within the last five weeks or so, some important "General Regulations" under the provisions of the "Leprosy Repression Act." It now is the duty of "every field cornet and police constable forthwith to report to the resident magistrate of the district the existence of any case of leprosy which may come to his knowledge." Europeans who are lepers are to be kept in wards into which "native or coloured" lepers are not allowed to go; the sufferers are to be classified "as far as possible" according to their condition of disease; notice of the serious illness or death of a leper is to be at once sent to his nearest relative by the medical officer in charge of the case. Visitors are not to be allowed "to enter any ward set apart for lepers or to visit a leper" without a permit from the medical officer in charge. The only person who has right of private access to a leper is his legal adviser.

To the very stringent legislation here set forth the leper in the colony of the Cape of Good Hope must now submit. As we read the Act, it is still legal in the Cape Colony for a leper to remain in his own home, provided the local authorities do not take action in his case and remove him to an asylum or to a hospital. It seems, however, very unlikely, judging from the tone of the Act, that the local authorities will allow lepers to remain in their own homes unless the authorities are convinced that while in his own home the leper will be carefully isolated and properly attended to in everything necessary to his own wellbeing and to the protection of his neighbours against the infection of leprosy. Here, we suspect, the authorities at the Cape will find some difficulty in carrying out the Act, both as to its letter and its spirit. But we must not forget that the colony is a community strong in the belief that leprosy, whether aided by insanitary surroundings or not, can only make new victims by virtue of the fact of its own infectiveness. Granted that the people have clearly apprehended the meaning of the inevitable consequences of such a belief as this, and have determined to carry their belief into action, then, in this event, it would be indeed venturesome to attempt to measure the amount of success which may crown their efforts. Much—practically everything—will depend upon how this Act is enforced at the Cape. In the interests of the healthy community, and of the lepers themselves, we hope the Act will be strictly carried out now that the people of the colony have decided to try the effect of compulsory segregation in their heretofore unsuccessful attempt to check the spread of leprosy among themselves.

We do not gather from the wording of the Act that the authorities at the Cape have reserved to themselves under it any power of inflicting definite penalties in the case of persons who break the law which it enacts, or even when its provisions are openly set at defiance. Perhaps the authorities have at command some means, which, however, do not seem to be mentioned in the Act itself, wherewith to enforce obedience to its requirements. Should this prove to be a weak point in the working of the Act, we should recommend the authorities at Cape Town to peruse the Act touching the question of the prevention of leprosy which is now in force in the colony of New South Wales. This Act is given in full in Dr. Thin's excellent work on "Leprosy." The New South Wales Act seems admirably suited to the purpose which its authors had in view,—namely, to enforce in the most thorough way the segregation of lepers, and to punish promptly all persons attempting to evade or to thwart the working of the Act.

In the Cape of Good Hope and in New South Wales we see the principle of the segregation of lepers being rigidly enforced. Among our fellow-subjects who follow this line of treatment in dealing with leprosy there is

no evidence of any tendency on their part to lose faith in what they are doing in that direction. They believe that leprosy spreads among healthy people because it is an infective disease, and they decline to postpone to an indefinite future the possible eradication of the disease by trusting to obtain this end by gradually improving their personal hygienic surroundings. The inhabitants of the Cape Colony have leprosy at their doors. We are told there are not wanting there certain sad examples of the truth that no person, in any grade of society, in a country where lepers are free to go about as they please, is safe from attack by this most horrible disease. It is not for us in England to judge harshly of people circumstanced as are the Cape colonists with regard to leprosy, or lightly to blame them for enacting severe laws against the leper. Many people are always eager to show with what calm, philosophical resignation they can regard the dangers and sorrows which, leaving them untouched, fall heavily upon their neighbours. Shall we be reproached for such an expression of opinion as this when to-day in British India lepers are allowed to act as salesmen in the bazaars, and to move about just as they please in the crowded thoroughfares of Indian cities?

Bacteriology.—As in Asiatic cholera, so also in leprosy, the question arises as to whether a specific micro-organism can be demonstrated in the affected tissues; and if so, does its presence there account for the characteristic symptoms of the disease. Professor Virchow, and Dr. Vandyke Carter, of the Indian Medical Service, though working independently, came to the conclusion in the year 1859, that in the enlarged nerves of persons suffering from leprosy, peculiar nucleated and granulated lepra-cells were always to be found. Professor Klebs determined, in 1873, that the tubercles of leprosy contained groups of specific bacteria. A few years later, Armaner demonstrated the fact that the granular lepra-cells of V. Carter enclosed heaps of bacilli. De Bary states that this bacillus possesses protoplasmic contents surrounded by a membrane, outside which is a material which swells up and assumes a gelatinous character so as to envelop the bacillus. It seems possible that it is by means of this gelatinous material that leper bacilli adhere to one another, and form the characteristic clumps so constantly found in the cells of tissues affected with leprosy.

The lepra bacillus resembles that of tubercle; in length it is about half to three-quarters the diameter of a human blood corpuscle. It is straight, but is sometimes slightly curved with rather rounded extremities. The protoplasmic interior of the bacillus may be demonstrated by means of certain reagents, such as iodine, or by staining agents, as hæmatoxylin; the protoplasm appears to break up into rows, with minute spaces intervening. The lepra bacillus multiplies by fission.

The micro-organism of leprosy may be stained by treating sections of affected tissues on a cover glass with a solution composed of the

following ingredients:—100 grammes of water, 5 grammes of carbolic acid, 10 of alcohol, and 1 of fuchsin (Ziehl's solution). The section is to be immersed in this solution for ten minutes, and then decoloured in a 25 per cent. solution of nitric acid; lastly, it must be washed in 60 per cent. of alcohol, and subsequently in distilled water, and mounted in a saturated solution of acetate of potash. Treated in this way the bacillus of leprosy, under the microscope, appears as a bright red rod. Gram's method stains the bacilli well, and demonstrates the divisions in its protoplasm. Fuchsin, gentian, methyl-violet, dahlia, in weak acid solutions stain the bacillus. An eosin-alum-hæmatoxylin solution (Ehrlich) stains the nuclei of the tissues blue, the cell protoplasm of a rose colour, and the bacilli orange.

With reference to the cultivation of the leprosy bacillus, the difficulty in demonstrating its specific character arises from the fact that, so far as we know, none of the lower animals are subject to leprosy, the bacillus having no pathogenic action on them.

In the Report of the Leprosy Commission in India (1890–91, p. 425), various cultivation experiments are described. Fresh "leper juice," that is, fluid drawn from living leprous tissue, was placed in glycerine-bouillon. In a mixture of this kind free leprosy bacilli were found about the tenth day. With fluid taken from a blister formed over a lepra tubercle, at the end of a month pure cultures of the bacillus were found. The Commissioners report that a tube containing such a culture transferred into glycerine-bouillon, at the end of three days the mixture had become slightly turbid; subsequently a pellicle or scum formed on the surface of the mixture; this was well developed about the twelfth day. As this scum was forming, minute greyish particles appeared on the surface of the liquid; these daily increased in number, joined together, so that the whole growth could be seen steadily advancing from the centre to the periphery. In some cases the scum sank to the bottom of the tube in a fortnight. The growth liquefied the gelatine, and it grew well on agar. The scum was found on microscopic examination to consist of a pure culture of leprosy bacilli. Sections made through fresh lepra tubercles, and treated as above, also produced pure cultures of bacilli.

No definite results have as yet been obtained by the inoculation of cultures of lepra bacilli into the lower animals. In man the disease has been communicated in this manner. Keanu, a Hawaiian criminal condemned to death, preferred to have a portion of a fresh leprous tubercle grafted beneath the skin of his left arm, rather than suffer death by hanging. Members of Keanu's family were leprous, but the man himself was free from any

symptoms of the disease, when a portion of fresh leprous tissue was inserted beneath the skin of his left arm. This was done on the 5th of November 1885. Within a month's time of this proceeding, Keanu complained of painful swellings along the course of the median and ulnar nerves of the arm into which the leprous tissue had been grafted; within six months these painful lumps had developed into unmistakable leprous tubercles. In September 1887 the following official statement was published regarding the condition of this man:—

OFFICE OF THE BOARD OF HEALTH, HONOLULU,
September 25, 1887.

This is to certify that we, the undersigned, have this day carefully examined one Keanu, a Hawaiian man, in confinement at the Oahu Gaol, who was inoculated with leprosy by Dr. Arning on November 5, 1885, and we find his condition to be as follows: Ears tubercular and considerably hypertrophied; forehead the same; face, nose, and chin show flattened tubercular infiltration; mouth clean, no tubercles; face generally presents a leonine aspect. Hands puffed, fingers swollen at proximal phalanges, tapering to distal phalanges; tips of forefinger and thumb of left hand are ulcerated from handling hot tin cups of tea or coffee, indicating anæsthesia. Body: black, thickly mottled with flattened tubercles, and the surface uneven to feel, colour of the same a yellowish-brown; front of the body, chest, and abdomen presents plaques of tubercular infiltration of larger size than back, separated from each other by wider intervals, and of a brighter colour, in some cases a ruddy pink, especially over upper part of sternum. Legs: the infiltration thins out as far down as the knees, there being one large bright patch on the inside of the left thigh; legs below knees quite clean, and skin smooth and even to touch. Feet, œdematous, have poor circulation; bluish colour; soles of feet clean. Seat of inoculation, outer aspect of left forearm, upper third, shows a dark purplish scar about $1\frac{1}{2}$ inches long by $1\frac{1}{2}$ inches wide, irregular in shape, keloid in aspect, dense, and inelastic. The tests for anæsthesia were not made. Eyes with sclerotitis, muddy, and injected. No signs of palsy about muscles of face, orbiculares palpebrarum, hands or forearms. It is our decided opinion that this man is a tubercular leper.

N. B. EMERSON, M.D.
J. H. KIMBALL,
Government Physician,
Honolulu.

Keanu died of leprosy within six years after the date of being inoculated with leprous tissue.

The Indian Leprosy Commission state that "sufficient time has now passed for it to be possible confidently to affirm that the presence of the bacillus lepræ in the new growths of leprosy is absolutely characteristic of the disease. Indeed, this bacillus has a specific relationship in the causation of leprosy. No leper is free from this organism, and in the bodies of those suffering from other diseases it never occurs."

Careful observations have been made with reference to the distribution of leprosy bacilli outside the body. But the evidence is entirely negative; neither in the earth constituting the floor of houses inhabited by lepers, nor in the tank water in which they bathe, could any lepræ bacilli be discovered. The same may be said of fish and of flies; there is no evidence showing that the leprosy bacillus exists in the bodies of the various species of these creatures which have been examined, either in India, or in other parts of the world.

Pathology.—Leprosy bacilli have been found within the cells of almost all the tissues of the body, in persons affected by leprosy; but it is in the cells of the diseased nerves and skin, that the bacillus will be discovered in the greatest abundance; in fact, the cells of such tissues are often crowded with these micro-organisms. It appears to be doubtful if the leprosy bacillus can live for any length of time outside the cell wall; at any rate it has not been found free, either in the blood or in any part of the human body.

Cells occupied by the leprosy bacillus vary much in size, some of them not being larger than white blood corpuscles, most of them are much larger, and the bacillus is not unfrequently found within compound cells. In the early states of its existence the bacillus does not interfere with the life of the nucleus of the cell which it invades; for the nucleus may frequently be seen of its normal size in cells crowded with bacilli. Subsequently, as the cell enlarges, its nucleus degenerates and ultimately disappears. As this disintegration of the living organic matter in the cell progresses, the formed material around it breaks down, and an ulcer in the part is the result of a process of this kind.

Nerve Leprosy.—In the early stages of this form of disease the bacilli first grow within the cells forming the sheath of one or more nerves, most frequently of the ulnar nerve. We find in this stage of the disease that an enlargement of the affected part of the nerve takes place, the swelling being due to an over-growth of its fibrous elements; among these fibres numerous cells may be found full of the leprosy bacilli. The secondary as well as the primary sheath of the nerve is affected in this way, and as the abnormal growth of the fibrous tissue increases, it presses upon the central axis of those nerve fibres which pass through it, and in consequence the functions of these fibres become impaired beyond the seat of the disease. Above the site of this over-growth of fibrous tissue the nerve may be perfectly healthy, and in passing through the diseased area a number of the nerve fibrillæ may escape destructive changes, and consequently perform their functions more or less perfectly.

Anæsthesia of patches of skin supplied by nerves which have been invaded by the leprosy bacillus is common, and is one of the most prominent features of this form of disease. Beyond atrophy of the subcutaneous fat and muscle fibres, no definite changes are observed in the tissues of anæsthetic portions of skin. But at a later stage of the disease, in consequence of the malnutrition following changes in the nerves, such as those I have referred to, ulceration of the skin occurs, and ultimately necrosis of the fingers, toes, and other parts of the body may follow. The loss of pigment in the skin, so constantly seen in well developed cases of leprosy, is attributable, like the anæsthesia, to defective innervation of the part.

Tubercular Leprosy, from a pathological point of view, differs in no way from nerve leprosy; in the one case the cells entering into the formation of the skin being invaded by the leprous bacillus, and in the other the connective tissue cells of the nerves are similarly affected. If an incision is made through a leprous swelling of the skin, or a tubercle as it is commonly called, a viscid fluid may be squeezed from the surface of the incision; and this tenacious matter under the microscope is found to contain a vast number of cells, more or less crowded with leprous bacilli. If the tubercle has existed for a considerable time, its cut surface presents a yellowish-white appearance, and a number of small spaces may be seen filled with the gelatinous substance above referred to. The abnormal growth is almost confined to the corium, the cutaneous structures in the first place becoming sclerosed; the sheaths of veins and vessels passing through this diseased area of skin also become thickened, and the circulation and innervation of the part is consequently impaired, so that degenerative changes occur, and extensive and deep ulcers form over the surface of the body. The skin of the face and ears, as well as of the hands and feet, in fact of the whole body, is thus often extensively diseased; cartilage and mucous membranes, especially of the larynx and epiglottis are frequently involved, as well as the lungs and other parts of the body.

Sections made through portions of skin affected with leprosy, and examined under the microscope, show a vast number of cells containing bacilli in the connective tissue. These cells are of all shapes and sizes, and unless in the advanced stage of the disease have well marked nuclei. The bacilli are often seen in sections made through the skin and hair follicles; their presence in the epidermis affords a means by which they may leave the body although the skin has not ulcerated. When ulcers have formed, a vast number of bacilli contained in cells must be thrown off the body, for the surface of

these ulcers is constituted of layers of cells, in almost every one of which colonies of the leprosy bacillus may be found. But the older colonies are dead, for in by far the majority of these cells the nucleus has disappeared; and without the nourishment which the connective tissue nucleus secretes, the leprous bacillus cannot live; this secretion appears to be the appropriate food of this micro-organism. The connective tissue cells of the iris, cornea, and conjunctivæ are not unfrequently in leprous patients invaded by the bacillus, which leads to sclerosis and chronic destruction of these tissues.

Etiology of Leprosy.—In the *Dublin Journal of Medical Science* for June 1877, Dr. Hawtrey Benson published the following case: "In 1872 Dr. Benson brought a man to a meeting of the Medical Society affected with tubercular leprosy, which commenced whilst the patient was in the West Indies, where he had resided for twenty-two years. After this individual had been under treatment in hospital for some weeks he was allowed to return to his home; he died there about eighteen months afterwards. After leaving the hospital, until shortly before his death, this leper's brother slept in the same bed with him, and wore the same clothes. This brother had never been out of Ireland, except on one occasion to England; there was no other case of leprosy in the family, but this man contracted the disease, and was shown to the members of the Dublin Medical Society on the 2nd of May 1877, suffering from tubercular leprosy."

Father Damien de Venster was born in Belgium with no trace of leprosy in his family; he left Europe in 1873 in order to devote himself to the relief of the lepers in the Hawaiian Islands. During the time he lived in the leper settlement of Molokai he ministered to the religious and material wants of the lepers; so far as he was concerned he treated them as he would have done any of his other fellow creatures.

In 1882 Father Damien first showed symptoms of leprosy; it commenced in his left foot, and in July 1889 he died of leprosy.

I have already referred to the case of the convict Keanu (p. 437), who developed fatal leprosy after having had a piece of fresh leprous tissue grafted into the skin of his arm.

I may here mention two cases of leprosy which have been under my care in London within the past few years; both patients were Englishmen, born and reared in this country, with no suspicion of leprosy in their families.

"A. after serving in India for some years was sent to Burma; he married a native woman, and in due time a son was born. The

child when 6 years of age was sent to England; when 11 years old, being still in this country, he developed tubercular leprosy, from which he died. His father in the meantime showed symptoms of leprosy. He left India and returned to England; the disease ran a slow course, and he died fifteen years after he became a leper. Before his death this patient's sight had been destroyed; his whole body and face was covered with leprous ulcers."

"B., a remarkably powerful man, served in the North-Western Provinces during the greater part of the time he was in India. He lived with a native woman for two years; some three years after this he became affected with tubercular leprosy. This man returned to England, and the change of climate appears to have arrested the progress of the disease. But the disease had destroyed his sight, and he presents marked tuberculous nodules over various parts of the body." These cases are known to other members of the profession in London, and there can be no question as to the nature of the disease or the circumstances of the patients as above given.

The three members of the Commission sent out from England in 1890 to inquire into the circumstances of leprosy in India, arrived at the conclusion that the disease "in the great majority of cases originated *de novo*," and that the extent to which leprosy is propagated by contagion "is exceedingly small." These Commissioners, before leaving England, were unacquainted with the language, habits, or prejudices of the natives of India, nevertheless it was from the evidence they collected from natives that the above opinion was formed; the Commissioners preferred their own judgment as to the matter at issue, rather than depend upon those who had resided among, and treated the diseases of the natives for many years. The Commissioners remark, at p. 259 of their Report, that, as a rule, our Indian civil surgeons believe that leprosy is a communicable disease, and that it spreads from diseased to healthy people in this way. But the Commissioners add that it is not "permissible to be too much guided by the opinion of medical observers in India—because most civil surgeons have not much opportunity of studying the etiology of this disease." Having been an Indian civil surgeon for some years, I beg to differ from the Commissioners in this opinion. I had frequent opportunities of watching cases of leprosy, often for several consecutive years, among the natives of India, and so of studying it in its various forms, and learning the family history of many such patients. The opportunities I enjoyed were not singular, and are within the province of most civil surgeons in India, for the disease is disseminated throughout the length and breadth of that vast and thickly populated

country. It seems to me that the opinion of our medical officers in India on this subject is deserving of every respect; and that as by far the majority of them believe from their own experience that the disease is communicable, their opinion is of great weight in a question of this kind.

The Rev. H. P. Wright, in his work on *Leprosy an Imperial Danger*, gives the following particulars of cases of leprosy (p. 33):—

(1) Betty MacCarthy, of Prince Edward's Island, native of Lancashire (England), married in 1836. In 1852 she became ill, and died of leprosy. She had five children.

(*a*) Richard, who died of leprosy, having suffered from the disease for twenty years.

(*b*) John, who died of leprosy, after suffering for twelve years.

(*c*) Mike, died of leprosy.

(*d*) William, died of leprosy. He was washed and buried by Joseph Brown.

(*e*) May, died of leprosy; she married John Doyle.

(2) John Doyle, died of leprosy.

(3) and (4) Two daughters of John and May Doyle died of leprosy.

(5) Joseph Brown, above referred to, died of leprosy.

(6) James Cameron, of Scotch origin, married, in 1866, Susan MacCarthy, one of the daughters of Betty, and had by the marriage two children, who, and the mother, are in good health. He often slept with Mike MacCarthy, and in 1870 presented undoubted symptoms of leprosy.

Dr. Thin, in his work on leprosy, has given numerous cases in which the communicability of leprosy from a diseased to a healthy person seems to have been established. I may also refer to the evidence afforded by Dr. Emerson, who was appointed by the Government of Honolulu in 1888 to "examine and authoritatively determine" the state of certain attendants on the lepers confined on the island of Molokai. Dr. Emerson reports that—

> Of the number examined sixty-six had at their own earnest importunity been granted permission by the Board of Health to enter the Settlement as non-leper *kokuas*, or helpers, to aid in the work of the Settlement, and in the care of their leper relatives and friends. Let me say that the duties of the *kokua* have a wide range, including nursing and care of the sick, fetching wood, water, rations, and other articles, cooking and washing for the disabled ones, and burying the dead. These *kokuas* are, as a rule, married to lepers, live in the same houses with them, and in every way conduct themselves as though they had neither fear nor care for the possibility of contagion. These people had been at the Settlement for periods varying in length from two or three to fifteen years, during which time they had been constantly exposed to the contagious influence of leprosy to such a degree and in such a variety of ways, that, short of actual inoculation, it would seem difficult for human ingenuity to devise conditions and methods more likely theoretically to communicate the disease than those which they had thoughtlessly employed and put into operation in the conduct of their daily life.

As a result of this examination, of the 66 *kokuas* that came before this Commission, 39 were declared to be lepers, 11 *suspects*, and 16 *not lepers*.

The conclusions to be derived from these facts is clear and direct. These people, while presumably not affected with the disease, enter upon such conditions of life as to expose them in as complete a manner as possible to the danger of its reception, with the result that 39 out of 66, or 59 out of 100—taking the latter as the whole number of *kokuas*—were actually infected by it.

There occur to me many other instances of lepers that I could mention by name were it proper so to do, which, after careful consideration, I have been led to regard as undoubted instances of contagion.

The force of the positive evidence in favour of contagion cannot be broken or weakened by the numerous instances of those who have escaped after seeming exposure to the disease.

I have already referred to Dr. Hillebrand's account of the introduction and dissemination of leprosy among the inhabitants of Honolulu (p. 429).

From a study of the history of this disease, its bacteriology and pathology, and from experience among a community where leprosy is frequently met with, I have come to the conclusion that it is caused by the action of a specific micro-organism. In fact, leprosy is a parasitic disease, and cannot therefore arise "*de novo*, or from any concurrent causes"; the only cause of leprosy is the bacillus of leprosy. Doubtless, this micro-organism presents great difficulties when we attempt to study its nature, its method of development, and the conditions of the soil or tissues in which it grows; in truth, these conditions are unknown to us at present; but, because this is the case, we should all the more firmly hold to the fact that the leprosy bacillus is the cause of the disease. If we act on this knowledge, all the rest will follow in the course of time; meanwhile we may do something towards mitigating the terrible evil which this disease inflicts upon a vast number of human beings; it is stated that in India alone there are 106,599 lepers, and this, I imagine, is far below the actual number of persons afflicted with leprosy in Hindostan.

Vaccination in relation to Leprosy.—The following case, given upon the authority of Professor Gairdner, of Glasgow, seems to point to the fact that it is possible the disease may be communicated from a diseased to a healthy person in vaccine lymph. I have taken the following details from Dr. Thin's work on leprosy, p. 193. A medical man residing in one of our colonies vaccinated his son with lymph taken from a child belonging to a family in which leprosy was known to exist. A Scotch ship captain's child was subsequently vacccinated from the child of the medical man above referred to, and Professor Gairdner saw both these children in

Scotland suffering from tubercular leprosy. "The child of the doctor living in a colony where leprosy is not uncommon may have acquired the disease independently of the vaccination. The child of the ship captain, on the other hand, visiting the colony only temporarily, had much less chance of acquiring the disease, and the presumption in favour of the vaccination is very much stronger."

Dr. Thin refers to other cases of a similar kind, which seem to render it probable that leprosy may be conveyed from an affected to a healthy person in vaccine lymph; and in localities where leprosy is endemic, we should be careful as to the source from which vaccine lymph is obtained.

Hereditary Transmission of Leprosy.—It is now held by all the best authorities on leprosy, that the disease is not transmitted through heredity from parent to child.

Symptoms.—In describing the symptoms of leprosy we may divide the cases into two classes, nerve, and tubercular leprosy; but we shall find that not less than 35 per cent. of the patients we meet with suffer from the mixed form of disease both nerves, and cutaneous structures having become affected with the leprosy bacillus.

Nerve Leprosy is more common among the natives of India than the tubercular form of the disease. In the early stages of this affection the patient suffers from feverish attacks, and more or less disturbance of his general health. But the first characteristic symptom which attracts attention is a burning, tingling sensation in patches of skin over various parts of the body, most commonly the back of the hands or forearms. Together with this uncomfortable sensation in the skin, the colour of the integument becomes altered; in some cases it grows darker, in others the skin pigment is diminished, and the part becomes whiter than the surrounding skin. At the same time, it frequently happens that an erythematous eruption appears over the affected patches of integument; this rash consists of pimples varying in size from a lentil, or less, to that of a pea; the spots last for a week or longer, and then disappear. The epidermis has a tendency to disquamate subsequently to the drying up of the eruption; after repeated attacks of this kind the surface of the skin becomes raised and thickened. This condition of things may continue for several years without the disease making any further progress; but in most cases, together with these changes in the skin, if the hand or forearm be affected, the muscles forming the ball of the thumb shrink, and there is a marked alteration in the conformation of the palm of the hand.

This wasting of the muscles also leads to loss of power, so that the patient's grip is weakened, and he has difficulty in writing and performing other delicate movements of his fingers and hands.

As the disease progresses the discoloured spots over the skin assume, from their centre outwards, a pale brown or dirty white colour; and at the same time the part becomes anæsthetic, with, it may be, hyperæsthesia in the surrounding skin. The patches enlarge and run into one another, so that considerable portions of the integument become anæsthetic and lose their natural colour. Over these patches the hairs turn white, and the cutaneous secretion ceases. Patches of this kind are, as a rule, rudely symmetrical, and appear over any, or it may be the greater part of the limbs, trunk, and face, but not over the scalp. These changes may occur without any material derangement in the state of the patient's health; and, as a rule, they progress very gradually for years, at one time with greater rapidity than at others, but always with remarkable slowness in their general advance. In some cases, however, the patient complains of severe neuralgic pains over various parts of his head, face, and limbs; this is particularly the case when the eyes are affected with leprosy. The supraorbital neuralgia is then at times excruciating, and is only to be relieved by the removal of the diseased eyeballs, supposing, of course, the sight has been completely destroyed, as it too often is in cases of leprosy when once the bacillus has become located in the inner structures of the eyeball.

In the more advanced cases of nerve leprosy, the ulnar and other nerves may be felt to be considerably thickened; and sooner or later bullæ form over the anæsthetic patches of skin. These bullæ vary in size from a hazel nut to that of a hen's egg, and contain a viscous yellowish fluid. They burst and leave an encicatrised red patch of skin, which in time is covered with a scab under which inflammation takes place, so that a depressed cicatrice forms over the site of the previously ulcerated surface. Bullæ of this kind seem to occur over anæsthetic portions of skin, in consequence of burns or injuries to the part; but they frequently form independently of any such exciting cause. On the sole of the foot, most commonly over the ball of the great toe, we sometimes meet with what are called perforating ulcers in cases of severe leprosy. These ulcers commence in a bleb over the affected part; but in place of superficial ulceration of the integument following, a circumscribed slough of the thick skin and subcutaneous structures of the sole of the foot occurs; the uncovered tissue in the course of time separates, and a deep painful ulcer remains, which is most difficult to heal.

In fully developed cases of nerve leprosy, necrosis of the fingers, and it may be of the toes and part of the foot, not unfrequently takes place. The part becomes painful and swollen, and the patient suffers from feverish attacks. The swollen skin gives way, and there is a constant flow of pus from the sore. The pain now diminishes, but the affected phalanx mortifies and separates from the living tissues, leaving a healthy granulating surface, which cicatrises in the course of time. One phalanx after another may separate in this way, until the fingers and toes disappear as well as the bones of the metatarsus and tarsus. In fact, the hands and feet of lepers may in the course of years be destroyed in this way.

During the later stages of this form of disease, the patient's health fails, he becomes anæmic, and is liable to visceral complications. Among these complications amyloid degeneration of the kidneys and liver, with chronic diarrhœa, terminate the patient's sufferings, which in many cases extend over a period of fifteen or twenty years.

Tubercular Leprosy commences with febrile symptoms, which in the course of time are followed in many cases by patches of leprous erythema, similar to that described in cases of nerve leprosy. After successive crops of erythema have appeared, or it may be there has only existed a burning, tingling sensation in the part, more or less continuously for months, the patch of skin affected becomes raised and thickened. These indurated patches of integument increase until they assume the form of nodules or tubercles; the hair over their surface disappears, the sebaceous follicles are dilated, and the skin looks smooth, greasy, and shining, a condition not inaptly described as resembling the rind of an orange when it is squeezed between the fingers. The colour of these tubercles varies in different races; in the light skinned they appear of a coppery colour, in darker races of a yellower hue than the surrounding integument. Tubercles most frequently commence over the skin of the forehead, the eyebrows disappear to a great extent; at the same time the lobes of the ears and the cheeks present the peculiar changes in the integument above referred to. It may be, however, that these changes commence in the skin of the arms, back of the hands, or some other part of the body. The mucous membrane of the larynx and mouth is commonly affected, and the patient's voice becomes husky. When fully developed, the skin of a patient's forehead is studded with tubercles, between which are horizontal furrows; his cheeks look greasy and tumid, and are covered with tubercles of various sizes. His lips are thickened and everted; his nose is flattened and widened, and the lobes of the ears are enlarged

and nodular. The conjunctivæ covering the globe of the eyes are yellowish and indurated, especially round the margin of the cornea; at a later stage of the disease the cornea becomes infiltrated, and a dense leucoma supervenes, with corresponding changes in the iris and deeper structures of the eye, leading to loss of sight. The skin over the back of the hands, arms, and, in fact, the whole body, may in the course of time become covered with elevated tubercular nodules, and as these increase in size, ulcers form on their surface; many of these sores last for years, others become covered over by a scab, and separation takes place beneath it, resulting in a depressed cicatrice. In many advanced cases the ulceration extends deeply into the fingers and toes, and these parts, as well as the nose and cheeks, are destroyed, producing the most hideous deformities. The various lymphatic glands of the body become enlarged, and ultimately break down; fistulæ form, the discharge from which, as well as that from the surface of the skin, is most offensive.

After years of suffering the patient's life is at length brought to an end by the extension of the leprosy to the mucous membrane of the larynx, lungs, intestines, and other parts. Lardaceous disease of the spleen, kidneys, and liver are not unfrequent complications of the latter stages of tubercular leprosy.

Treatment of Leprosy.—*Preventive.*—Leprosy being a parasitic and communicable disease, strict isolation of lepers must be the proper and only effective way of stamping it out. But in the present state of society, especially throughout the greater part of Asia, the complete segregation of lepers is impossible. I have already referred (p. 434) to the details of the Leprosy Repression Act of 1884, passed by the Legislative Council of the Cape of Good Hope. The provisions of this Act are excellent, as were the reasons given for its enactment, viz. that the "disease of leprosy is prevalent in the colony, and has lately been spreading, and continues to spread." The Government of the Cape have taken the right means to prevent the extension of the disease among the people committed to their charge, and doubtless under measures of this kind leprosy will in the course of time disappear from the colony. Our fellow countrymen in Australia have adopted precisely similar steps with reference to the treatment of lepers, segregation of the affected from the healthy community being the principle upon which they have founded their legislative enactments. There can be no question, if it were possible to carry out a similar system of isolation in India, leprosy would in the course of years disappear from among the inhabitants of that country; but, as I have said, this is impracticable. The religious feeling, customs, and habits of the

natives, as well as the number of lepers in India, all prevent the Government from attempting to introduce a system of compulsory segregation in that country. What I mean is that, so far as my knowledge goes, however desirable it may be to segregate all the lepers in India, it is impracticable, and consequently the best we can do for our fellow subjects in that country is, to make such provision for, and to draw such restrictions round, lepers as are possible in existing circumstances.

It would seem to be well within the power of the Government of India to enact certain measures for the protection of healthy persons against lepers. These measures may be divided into two classes: A. Permissive; B. Compulsory.

A.—I. For the isolation of lepers restrained in their homes at the express wish of friends; suitable separate lodgment would be necessary in such cases.

II. For founding leper colonies or villages, consisting principally of the affected, who, while allowed a considerable amount of liberty of movement, should be prevented from mingling with the healthy surrounding population.

B.—I. Adequate asylums to be provided for lepers in all the large towns and civil stations of India.

II. Authority to prevent the sale of articles of food and drink by lepers; to prevent such persons from practising prostitution; or from carrying on such occupations as that of barber, washerman or washerwoman; or any occupation which concerns the food, drink, and clothing of the people generally.

III. Authority to take up vagrant lepers; to succour the sorely diseased who may be insufficiently guarded, or cared for, at home; and at times to enforce continued isolation of lepers until medical sanction of liberty is granted to them.

The Government of India, as I am informed, had actually prepared a Bill as far back as 1889, which they were ready to have introduced into the Legislative Council regarding the preventive treatment of lepers. The action of Government was, however, stopped in consequence of the proposed despatch from England of a Commission to examine into the subject of leprosy. The provisions of the measure proposed by the Indian Government were to give power (1) for the expenditure of funds on the appropriation of property to retreats; (2) for the detention of lepers in retreats at their own request; (3) for the detention of lepers otherwise than at

their own request (district magistrates to have power to place suspected persons in retreats); (4) for the discharge of lepers from retreats; (5) for the recovery of lepers escaping from retreats; (6) for the segregation of the sexes; (7) for the protection of the religious belief of the lepers. Local governments would have been allowed to make rules regarding the inspection of retreats, and powers of inspectors, the management of retreats, the conduct of lepers in retreats, the exercise of authority to discharge lepers, the punishment of lepers escaped and brought back, and the carrying out of the Act generally.

Hygienic Treatment.—In cases of leprosy, especially in its early stages, it is advisable, if possible, to remove the patient from the locality in which he has contracted the disease, and to place him in a bracing and healthy district. At the same time every care should be taken that he is surrounded by favourable sanitary conditions, in fact, placed in circumstances conducive to the promotion of good health. In the case of many lepers it is impossible to remove them to a bracing climate; but, by proper restrictions and isolation, most of them might be provided with comparatively healthy homes. The crippled, and often hideously deformed lepers too often seen wandering about the bazaars of our large Indian towns, are not only a disgrace to humanity, but it is hopelessly beyond the power of any one to attempt to relieve them under existing laws.

Medical Treatment.—As regards local applications to the skin, the oil expressed from the choulmoogra nut has been extolled as being almost a curative remedy in cases of leprosy. The oil is to be rubbed into the skin twice a day, for fifteen or twenty minutes at a time. The oil may also be taken internally, in doses of twenty drops two or three times a day. I think there can be no question that patients treated in this way, provided they are also supplied with pure air, water, and wholesome food, do improve; the disease may probably be stayed in its progress by these means, but I question if it can be cured.

Gurgun oil and kowti have also been employed as external applications in cases of leprosy with unquestionable benefit in alleviating the symptoms. But, after long continued use of these drugs by friction over the skin of the affected parts, most practitioners have arrived at the conclusion that they are in no way superior in their action to that of choulmoogra oil.

Arsenic, administered internally for months together, has seemed to me more beneficial in the treatment of leprosy than any other drug. In cases of comparatively recent leprosy, especially when the crops of tubercles appear after feverish attacks, there can be no

doubt that a continued course of arsenic, together with careful hygienic treatment, will arrest, if not cure, the disease. In combination with arsenic taken internally, friction with choulmoogra oil should be used externally.

Ichthyol and salol, in full doses, are highly spoken of by some medical men as having a salutary influence in the treatment of leprosy. But our efforts must be directed to discovering some chemical substance which will kill the leprosy bacillus after it has invaded the cells, entering into the formation of the affected tissues. The difficulty is to find such a compound, which, while destroying the bacillus, will not otherwise harm the patient's general health.

It has been proposed to employ nerve-stretching for the relief of leprosy, and an operation of this kind may be useful in deadening the intolerable neuralgia which some lepers suffer from. With this object in view nerve-stretching may be practised, but beyond relieving pain the operation can do no good in cases of this disease. Excision of cutaneous tubercles has also been recommended, and should cases of tubercular leprosy come under our care in which one or more isolated tubercles exist which can be removed, no harm can result from this practice.

In instances of deep perforating ulcers relief may be given, and healthy action in the ulcer follow the excision of the diseased tissues surrounding the sore, or by their complete removal with a sharp spoon. The cavity left after such an operation, as soon as the bleeding stops, may with advantage be packed with powdered boracic acid.

With reference to the treatment of diseases of the eye, occurring in consequence of the growth of the leprosy bacillus in the part, little can be done. Atropine should be employed to stop, if possible, the formation of adhesions between the iris and lens. In some cases iridectomy is necessary; and, as I have before remarked, if the sight has been destroyed, we may have to remove the globe of the eye to relieve the neuralgia from which such patients sometimes suffer.

CHAPTER XII.

BERIBERI.

BY PATRICK MANSON, M.D., M.R.C.P., LL.D.

History.—Until recent years there has been much confusion in the medical mind with regard to the disease known as Beriberi, and it is only very lately that well defined and well grounded conceptions as to its nature are beginning to be entertained. Our knowledge of the subject presents the curious anomaly of having at one time, after attaining considerable precision, distinctly retrograded. When, in 1835, Malcomson wrote his essay on the *History and Treatment of Beriberi*, he gave us a powerfully drawn and very accurate description of the disease, bringing its symptomatology and pathology well abreast of the medical science of the time; but, strange to say, his teaching, although reiterated by such writers as Copland, Aitken, and others, seemed until lately to have made little impression, and was neglected and almost forgotten. Many systematic writers on tropical disease—led away by crude theories about beriberi, its nature and origin—so twisted and pared down Malcolmson's descriptions in order to fit them to some favourite theory, that the beriberi he and his contemporaries described is no longer recognisable in some modern works; and many of these writers have ended up by practically disregarding altogether the most striking and important element—the neurotic—in the symptomatology and pathology of the disease.

The first and most of the steps in the revival of our knowledge of this important disease were made in Brazil, in Japan, and in the Netherlands' Indies. Working in these countries da Silva Lima,[1] Hoffmann,[2] Wernich,[3] Anderson,[4] Sim-

[1] Da Silva Lima, *Edin. Med. Journ.* March 1873; *Arch. de méd. nav.* 2 Sem. 1873; and several papers in Portuguese and Brazilian journals.

[2] Hoffmann, *Trans. Germ. Asiat. Soc.* Tokio, 1872.

[3] Wernich, *Deut. Arch. f. klin. med.* Bd. 71; *Geog. med. stud.* 1875; *Virchow's Archiv*, vol. lxvii. 1876.

[4] Anderson, *Lectures on Kakké*, Yokohama, 1879, *St. Thomas's Hosp. Rep.* vols. vii. viii.

mons,[1] Eldridge,[2] Scheube,[3] Baelz,[4] Wallace Taylor,[5] Pekelharing and Winkler,[6] and others, have not only led us back to where Malcolmson left us, but have swept away many of the crude theories I refer to, and have gradually brought our knowledge of beriberi well in line with modern medical science, and paved the way for still further advance.

Little is known of what might be termed the history of beriberi. There is evidence in the writings of the physicians of China and Japan that it has existed in these countries from remotest antiquity. Native Indian writers appear to be silent on the subject; for the little we know of beriberi in that country we are indebted to our countrymen. Owing to the apparent exemption enjoyed by Europe, there is no mention made in our own literature of such an affection until some centuries after active intercourse with Eastern countries had commenced. Bontius, who wrote in Batavia in 1629, is the first European author who attempted a description of the disease. More than a century afterwards he was followed, in 1757, by Lind; later, in 1804, by Hunter; in 1822 by Marshall; and subsequently by many writers on Indian diseases. No mention is made of beriberi in America until da Silva Lima called attention to the subject in 1866; so little appears to have been known about the disease in Brazil before this date that he alluded to it as the "morbus innominatus." The early Dutch residents at Nagasaki appear to have had some suspicion as to the true nature of what is called "kakké" in Japan; but the distinct recognition of the identity of this with beriberi dates only from some time subsequent to the opening of the country to foreigners. Simmons claims to have been the first to really recognise the true nature of "kakké" in 1870; at all events, it is only since that very recent date that the investigation of beriberi in Japan and elsewhere by modern methods practically commenced.

Many diseases, especially those in which œdema is a prominent symptom, have from time to time been included under the term beriberi. This name has been applied to different forms of malarial

[1] Simmons, *Chinese Imp. Mar. Customs Gaz. Med. Rep.* 19th Is. 1880.

[2] Eldridge, *Pacific Med. and Surg. Journ.* 1880.

[3] Scheube, *Deut. Arch. f. klin. med.* 31 and 32; Zenker's *Arch. f. klin. med.* 1882; *Trans. Med Cong.* Amsterdam, 1883; *Virchow's Archiv*, vol. xcv. 1882.

[4] Baelz, *Deut. ges. f. nat. und volk.* Ostasien, Tokio, 1882; *Arch. de méd. nav.* April 1884.

[5] Wallace Taylor, *Sei-i-Kwei*, May 1886; *Studies in Japanese Kakké.* Osaka, 1886.

[6] Pekelharing and Winkler, *Recherches sur la nature et la cause du Beriberi.* The Hague, 1888.

Good bibliographies of beriberi are to be found in Hirsch's *Geog. and Hist. Path.* (Syd. Soc.); Corre, *Maladies des Pays Chauds*; Roux, *Maladies des Pays Chauds.*

cachexia, to scorbutus, to anæmia, and such causes of anæmia as anchylostomiasis, and to many other similar affections. At the present day the term beriberi (a word of uncertain etymology) is, or ought to be, restricted to the disease to which it was first applied in European medical literature by Bontius, and whose leading features were unmistakably defined by Malcolmson under that name. Local names—of which it has a crowd, including the best known of them "kakké," and certain somewhat pedantic appellations, for the most part expressing some theory about the nature of the disease which may or may not be correct—ought to be discarded, and the original name strictly adhered to, and its scope accurately defined.

Definition.—Beriberi may be defined as a specific, endemo-epidemic multiple neuritis of uncertain extent, duration, severity, and course. Mostly confined to tropical and subtropical countries, it is characterised by varying degrees of muscular paresis, muscular hyperæsthesia and atrophy, by anæsthesia and paræsthesiæ in certain skin areas, and by local or general œdema; and also by a marked liability to sudden death from paresis of the heart, or of the muscles of respiration, or from œdema of the lungs, or from hydro-pericardium, or from a combination of these conditions.

Geographical Distribution.—Beriberi is reported as having been recognised on the eastern side of Asia as far north as Saghalien. In Japan it is very prevalent; and there are accounts of it from Hakodate, Tokio, Yokohama, Kioto, Osaka, Nagasaki, and many other places. In China it appears not to be so prevalent as in Japan, although the limited acquaintance of foreigners with native diseases in the former country may, in some measure, account for this apparent rarity; it has been seen, however, in Shanghai, Tsuchou, Wenchow, Foochow, Formosa, Amoy, Swatow, and Fatshan. In 1847 fifty-two soldiers of the Madras 42nd Regiment were attacked with beriberi during their third year of service in China; and in 1857, and again in 1860, epidemics of the disease occurred in the jail at Hong-Kong. In 1882 and in 1884 I saw cases of it in Hong-Kong, and again in 1888 and 1889, when it was extensively epidemic among the native population there.[1] It is evident, therefore, that modern China is no more immune from beriberi than was ancient China, and that the reputation which this country for some time enjoyed in this respect was not deserved. There are no accounts of beriberi in the Phillipine Islands, but doubtless it exists there. It has been recognised in Tonkin, in Annam, in Cochin-

[1] Papers on the subject of the prevalence of beriberi in Hong-Kong. Report to Sanitary Board. Hong-Kong, 1889.

China, and in the neighbouring kingdom of Siam. It is very prevalent at times in Singapore, in Malacca, in Penang, and in the Malay peninsula generally. It is exceedingly prevalent at Atchin in Sumatra, and is found in Labuan, Borneo, Java, Celebes, the Moluccas, and throughout the Malay Archipelago. It is known in New Guinea, and at times is very prevalent among the crews of the pearling fleet in the neighbouring Torres Straits. In Burma beriberi attacked the British troops in 1824, and is probably endemic,—epidemics of it having been observed lately as far inland as Mandalay. In India it is chiefly met with in the Northern Circars, less frequently on the Coromandel Coast, in the Carnatic, and on the Malabar Coast; although the interior is reputed to be exempt, Fayrer and Moore seem to countenance the idea that it is more generally diffused throughout India than is usually supposed. Possibly it occurs in and around Calcutta and in Assam. Formerly, at all events, it was a common disease in Ceylon. It has been met with at Aden, Zanzibar, Réunion, Mauritius, Nosi-bé, Madagascar, on the Congo, and probably elsewhere on the West Coast of Africa. Certain cases of a form of peripheral neuritis have been reported as occurring in fishermen on the North American Coast, and are supposed to be beriberi.[1] It has been distinctly recognised in Cuba, in Guadaloupe, and in several others of the Antilles; it is known to occur on the Isthmus of Panama and at Cayenne, and it has been extensively epidemic in Brazil and in Paraguay. As regards the west coast of America there is no definite information, although it is not unknown among the crews of vessels trading there. Doubtful reports about it come from the Sandwich Islands, from the Gilbert Group, from Tahiti and others of the South Sea Islands.

Many epidemics of beriberi are reported as having broken out on board ship, and in this way cases have often been carried to ports altogether outside the strictly endemic area. Thus it has been carried to San Francisco by Japanese and Brazilian men-of war; to many places by labour and pilgrim ships; to England and, most probably, to continental ports by vessels from the East. Frequently beriberi cases are treated in the Seamen's Hospital, Greenwich;[2] and in 1887 several were landed at Newcastle from the Chinese transport *Too-nan*.[3] Formerly in the Indian and China Seas, when

[1] *Journ. Nerv. and Ment. Dis.* Aug. 1890; Seguin, *Philad. Med. News*, Dec. 1886.

[2] Between May 20 and October 31, 1892, I saw or heard of, through my connection with this hospital, 36 cases of beriberi, 5 of which were fatal. All of these cases came from ships in the port of London, and many of them developed in the Asiatic crews of vessels which had been "lying up" there for several months.

[3] *Lancet*, July 23, 1887.

the voyages were long and the sailing ships carried large native crews, who were badly fed, and badly housed in dirty, overcrowded, damp and steamy forecastles, beriberi frequently broke out on board ship, and was the cause of many sudden and apparently mysterious deaths and inexplicable dropsies and paralyses. Indeed, so common was it in ships, that Carter[1] attributed the derivation of the term "beriberi," to two Arabic words, "bhur"-dyspnœa, and "bhari"-marine. Even at the present day such epidemics are by no means rare, and many isolated cases occur on board even what are reputed to be well appointed steamers. Surgeons of vessels trading to the East and to the coast of Africa, and carrying native crews, would do well to bear this fact in mind, and regard complaints of muscular debility, degrees of leg paresis, numbness of limbs, slight or more extensive œdemas, and breathlessness among the crew, as being very probably attributable to beriberi. Malingering is only too common in native crews, but in presence of the symptoms I allude to, it would be well to pause before pronouncing such a diagnosis; too often in such cases the possibility of beriberi is overlooked, and the man who is pronounced a malingerer to-day is found dead in his bunk to-morrow morning.

Symptoms.—There are few diseases with so varied a clinical facies as beriberi; consequently it is exceedingly difficult to convey in words a clear conception of what it is like.

To the medical man encountering it for the first time, and with only European experience and the ordinary text-books to guide him, case after case may present itself in the out-patient room or in the wards of a native hospital, and yet he may for a long time entertain no suspicion as to the real nature of the disease he is dealing with. Such diagnoses as muscular rheumatism, locomotor ataxia, progressive muscular atrophy, heart disease, Bright's disease, œdema from anæmia, suggest themselves,—so varied and so apparently unconnected are the elements of the picture (Fig. 41). Such, as a matter of fact, are some of the diagnoses the novice in tropical medicine generally, on his first encountering beriberi, arrives at and registers. By and by he is agreeably astonished to find that many of his presumed locomotor ataxia cases recover rapidly, his progressive muscular atrophy cases cease to progress, his Bright's disease cases show no albumen in a fairly dense urine, and his heart cases lose their bruits. On the other hand, he is painfully surprised at the number of sudden deaths occurring among patients who did not seem to be very ill; he cannot account for the frightful attacks of dyspnœa he frequently has to treat, for the anæsthetic patches in the atrophic cases, for

[1] *Trans. of Bombay Med. and Phys. Soc.* No. 8, 1847.

the absence of ocular, bladder, and other characteristic symptoms in the apparently ataxic cases. After a time the idea of peripheral neuritis may occur to him, and then perhaps, on a hint from some one with more experience, or from a book, or by a sort of happy inspiration, he one day concludes that the diseases he is dealing with are but phases of the many-sided complaint, beriberi. Such I know

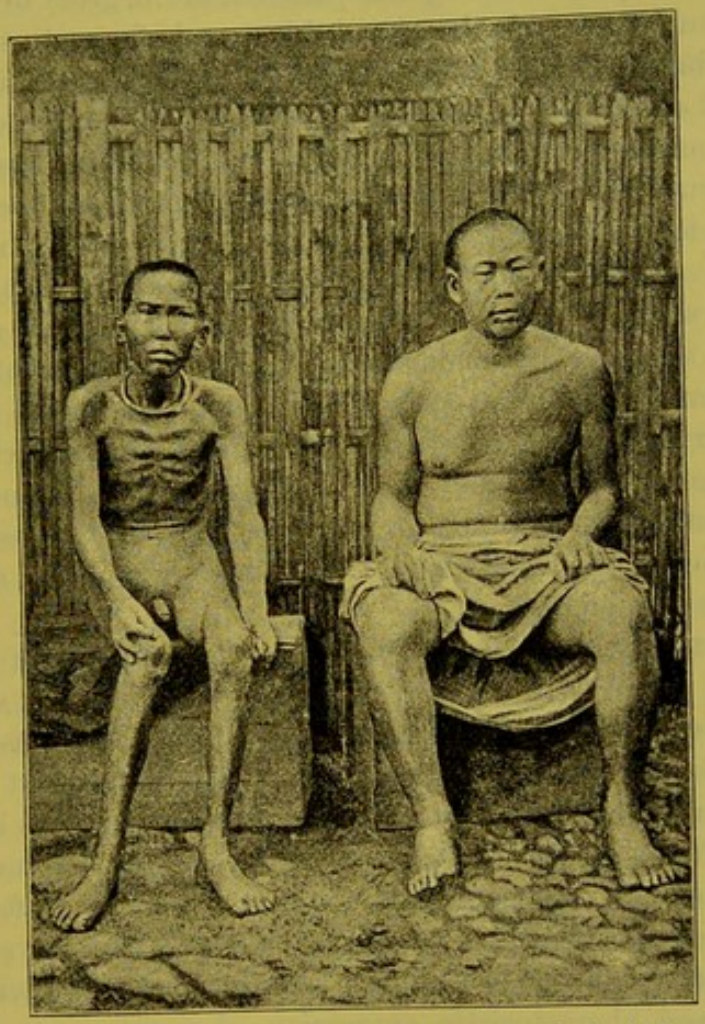

FIG. 41.—The "dry" and "wet" types of beriberi. (After Pekelharing and Winkler.)

to be the history of many a medical man's initiation to the diagnosis of this disease.

Until recently, either owing to the incomplete description of the disease as a whole, or to the disproportionate importance given in text-books to the dropsical, circulatory, and anæmic phases of beriberi, the student of medicine, until set right by experience, carried in his mind a picture of beriberi in which dropsy, anæmia, and cardiac bruits were the leading features. But such a picture is far from giving a correct and properly balanced conception of

what beriberi is really like; indeed, those features, thus dragged into undue prominence, are, in the vast majority of cases, far from being prominent ones; in many cases they do not obtrude themselves at all. Dropsical beriberi, in which the dropsy proceeds to an extreme degree is, in my experience, a rare disease. In the great majority of cases the most prominent features, and those principally complained of by the patients, are degrees of paresis of the legs, numbness of the feet, shins, and finger-tips, muscular weakness, breathlessness and palpitation; rarely is extreme dropsy complained of. In fact, beriberi is a disease of the nervous system, not what might be called a humoral or circulatory disease. The invariable underlying factor, that which determines all the important symptoms, is of a neurotic, not of a hæmic, character—in fact, it is a neuritis. Let the student thoroughly grasp this. Let him think out what would be the manifestations of a neuritis of varying degrees of intensity and suddenness of onset or exacerbation affecting the nerves of different parts of the body,—the crural nerves, the brachial nerves, the nerves of the trunk, the pneumogastric, phrenic, and laryngeal nerves, and the vasomotor nerves,—and he has thought out the symptoms of beriberi; he is in possession of the key to all its complex of symptoms, and their apparently inexplicable grouping.

Prodromata.—In most cases the development of the more pronounced features of beriberi is preceded by certain vague and ill-defined symptoms, which may be considered to constitute the prodromic stage. For several days or weeks the patient finds that he is not quite able for his work; he is languid, his legs and knees feel weak, he is easily put out of breath, he has vague pains in the calves of his legs, sometimes he has headache or feels chilly, and he is easily exhausted by mental as well as by physical effort. An ill-defined puffiness of the face is often observed, giving rise to a peculiar and characteristic expression of countenance by which those experienced in such cases are enabled to foretell the impending explosion. Now and again for a few days the patient may feel quite well, but presently he relapses, and he may keep getting better and worse in this way for a considerable time before the more characteristic symptoms of the disease definitely declare themselves.

Incidence and Progress of Symptoms.—These, when they come, may appear quite suddenly; more often they gradually supervene on the prodromic stage, seeming to grow out of it. Thus the rate of incidence of symptoms is subject to considerable variation. Not uncommonly a patient may go to bed fairly well, and on waking next morning discover that his shins are numb and œdematous; and,

on attempting to get out of bed, he finds that he has a difficulty in walking or even standing, and if he happen to squeeze his calf muscles he discovers that they are exquisitely tender. In another case, numbness and puffiness over the tibiæ may gradually and almost imperceptibly develop during several days.

In either case by degrees the numbness may extend to the thighs, to the arms, and to the hands, being especially remarked in the finger-tips. The patient finds he has trouble with his buttons or tools. The muscles underlying the areas of numb skin, such as the calf muscles, are found to be very sensitive when pinched or pressed; the hand grasp may be weakened, and the ability to walk and stand impaired.

Usually a characteristic walk is now developed, depending partly on imperfect perception of the ground, partly on weakening of the foot flexors, and partly on a certain amount of retraction of the heels from semi-spastic contraction of the calf muscles. Thus the patient when he attempts to walk has to raise the heel very high before he can clear his toes from the floor on advancing the foot; and on the foot being placed on the ground again, the toes are seen to be the first to touch it, the sole and heel falling with a flop. Should the flexor muscles of the leg and thigh become seriously involved, the foot cannot be raised, and then the gait becomes a sort of shuffle, the patient helping himself along by clinging to any available support.

Uncomfortable subjective phenomena may frequently distress the patient. The calf muscles feel full and stiff, and as if constricted; in some cases even passing from time to time into a condition of violent and agonising spasm. Probably a sensation of constriction is also felt around the epigastrium and chest, and there may be attacks of palpitation and breathlessness on slight exertion.

The œdema may from the first be confined to the shins and ankles, and continue quite insignificant, or even disappear; but in what is known as the mixed variety of beriberi it gradually extends up the thighs, and may appear in both arms and in the hands, or about the root of the neck and different parts of the trunk, especially over the sternum and about the flanks. Sometimes it is limited to one hand, or one arm, or to one side of the neck. It may come and go in a few hours, flitting from one place to another, though usually it is more persistent.

At any time the numbness, the œdema, and the paresis may cease to extend, and recovery set in. Should, however, the disease advance, the muscles of the thighs, of the arms, of the hands, the pectoral and abdominal muscles, suddenly or slowly, simultaneously

or one after another, may all or any of them become involved. In a severe case the patient may, in the course of a few days, be unable to move about, sometimes being hardly able even to turn himself in bed. If improvement does not soon set in, many of the muscles may begin to atrophy, and completely lose the power of contracting, whilst others remain swollen and hard. If many muscles are involved, the patient lies on his back, a more or less swollen or miserably atrophied and helpless log; his toes semi-flexed, and his feet dropped almost in a line with his legs; his hands lying passive beside him; his voice perhaps husky or almost abolished from implication of the laryngeal and thoracic nerves; his tendon reflexes, and sometimes even his superficial reflexes, in abeyance; and his limbs and trunk more or less swollen, or, perhaps, wasted to a skeleton (Fig. 42). In extreme cases of atrophy, if death does not

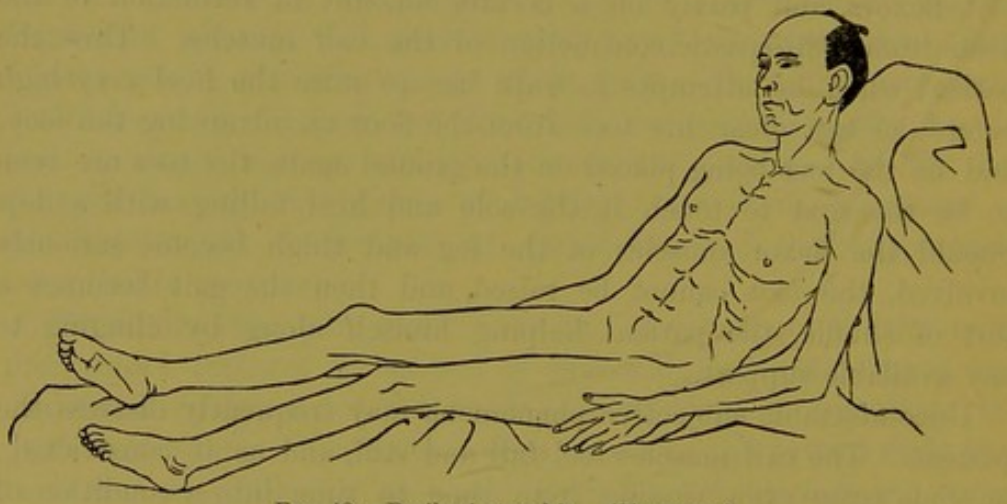

FIG. 42.—Beriberi atrophia. (Simmons.)

supervene, months will elapse before the wasted muscles are rehabilitated, and some of them may be permanently lost.

Notwithstanding all this amount of disease of muscle and nerve, the functions of organic life are usually efficiently performed; the appetite is good, the bowels are fairly regular, urine is sufficient in quantity, contains no albumen, and, apart from some little trouble from weakening of the abdominal muscles, is regularly and spontaneously voided. Cerebration, and the senses of sight, hearing, smell, and taste are generally unimpaired.

In one type of case the most striking symptom is the œdema (Fig. 43). This at times is very great. It may creep slowly up the legs and thighs, or it may appear suddenly, in the course of a few hours involving trunk, arms, head, and neck. Along with the œdema there is more or less paresis; and, in such an extreme case, the patient lies passively on his back, completely waterlogged and

distended, looking like a drowned body that has been for days in the water, and puffed out as much as the skin will stretch. Such a patient is quite unable to get up, or even to move much in bed; for, in addition to his weakness and unwieldiness, he is troubled with palpitation and attacks of dyspnœa. His urine is reduced to a few ounces a day, and his bowels are confined. Relief may come with a copious diuresis; but this form, although the actual amount of muscular paresis may not be very great, is a very dangerous type of beriberi, owing to the pronounced disposition to œdema, which, if it extend to the lungs, proves rapidly fatal.

Terminations.—Nothing is more striking about beriberi cases than the liability to sudden death. A case seen to-day, and considered by no means a serious one,—perhaps being able to move about a little, to take food with appetite, to converse with friends,

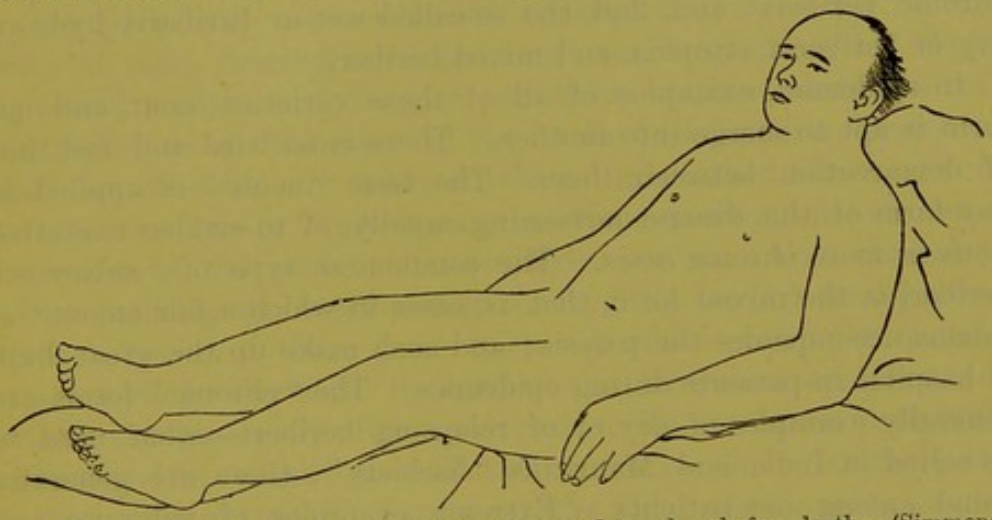

Fig 43.—Beriberia hydrops. Appearance presented two days before death. (Simmons.)

free from fever, and apparently doing well,—may be dead by to-morrow's visit. The paralysis may have been of very moderate extent and degree, the œdema trifling, the heart's action only slightly irregular and tumbling, and everything may seem to be going on favourably; when suddenly, with or without aggravation of the paresis and anasarca, distressing orthopnœa sets in, and in a short time, if not quickly relieved, after one or more struggles most distressing to witness, the patient dies.

Another striking feature about beriberi is the uncertainty, the irregularity, and the duration of its course. Some cases are well in a few weeks; others, dragging on for months, get better only when the cold season comes round, relapsing again perhaps with the advent of the next summer. This may occur for several years in succession, and to such cases the term "chronic" is sometimes applied. Most cases vary much from day to day, their condition

being influenced apparently by the state of the weather; heat and damp, or cold and damp aggravating the symptoms, mild or cool dry weather almost invariably improving them.

Classification of Cases.—Owing to the infinite variety in the combinations of paresis, atrophy, numbness, œdema, and rate of progress, it has been deemed advisable by several writers to attempt a classification of the different forms of beriberi; and such classifications have a certain use, provided it is thoroughly recognised that the different terms employed apply to only one disease.

To facilitate description, and to a certain extent for practical purposes, it is customary to divide the cases in two ways—1st, according to the intensity of the symptoms and the rate of progress of the disease; 2nd, according to the relative amounts of œdema and atrophy present. Thus we have: 1st, acute, subacute, and chronic beriberi; and, 2nd, the so-called wet or beriberia hydrops, dry or beriberia atrophia, and mixed beriberi.

In epidemics, examples of all of these varieties occur, and one form is apt to merge into another. There is no hard and fast line of demarcation between them. The term "acute" is applied to any form of the disease advancing rapidly, or to sudden exacerbations in more chronic cases. The commonest type of "subacute" beriberi is the mixed form, that is, cases in which a fair amount of œdema accompanies the paresis; and such make up the great bulk of hospital in-patients during epidemics. The "chronic" forms are generally examples of dry or of relapsing beriberi—what used to be called in India and Mauritius "barbiers"; these are generally found among out-patients. Extreme examples of subacute wet beriberi are comparatively rare anywhere, and so are pure examples of subacute dry beriberi. In more northern latitudes, as in Japan, the mixed and dry forms prevail, and the wet forms are rare; in equatorial latitudes, while the dry or mixed forms are common enough, the proportion of the wet variety is larger than in cooler latitudes.

Whatever may be the type of the disease, there are certain symptoms which all cases of developed and active beriberi have in common. These are—1st, numbness of the skin over the front of the legs; 2nd, more or less marked œdema in the same situation; 3rd, tenderness of the calf muscles on pressure. These are frequently the only symptoms present; but whatever the gravity of a case, whether it is one of extensive œdema with grave circulatory disturbance, or whether it is one associated with extensive muscular atrophy or paresis, or whether it is of the mildest possible type, these three symptoms are present at one time or another, and they

are the most important of the diagnostic marks. To these may be added as frequent, though not invariable, accompaniments, weakening or complete abolition of the patellar tendon reflexes, breathlessness, and palpitations. So that in beriberi endemic centres, or in individuals coming from such centres, if a case be encountered in which the skin of the leg is numb, the integuments puffy, and the calf muscles sensitive to pressure, and the patellar tendon reflex is absent, and complaint is made of shortness of breath and palpitation, the presumption is that the case is one of beriberi.

Analysis of Symptoms.—Although from this short description of the symptoms of beriberi it is evident that the disease is one primarily of the nerves, there is no very definite clinical evidence that the central nervous system, the brain or spinal cord, are seriously implicated; the brunt of the disease falls almost entirely on the peripheral nerves—motor, sensory, and vasomotor. The senses of sight, hearing, taste, and smell are rarely affected. Slight degrees of exaltation, dulling, or perversion of these senses, as well as some degree of obfuscation of the intellectual faculties, have been noted, but there is very rarely anything like marked implication of the higher centres. There is some evidence pointing to occasional implication of the cord or its meninges; but, as a rule, clinical as well as pathological observation give very little support for considering that disease of these parts is in any way a prominent feature. In attempting, therefore, to analyse the individual symptoms of beriberi, though we are always led back to disease in the peripheral nervous system, we can get no further; this seems to be the point of origin of the various affections of the skin, subcutaneous areolar tissue, muscles, and circulation,—the source to which they may always be traced, and from which they cannot be divorced.

Integuments.

The colour of the skin in beriberi depends very greatly on the presence or absence of œdema, and also, of course, on the natural colour of the patient's skin, whether white, yellow, or black. When œdema is considerable, and the skin more or less on the stretch, it has a pallid, pasty appearance. If much anæmia is present the mucous membranes show it; but very often these are of natural tint, or are bright red, or dusky from cyanosis. Similarly, the hands and face may have a bluish look. In the dry, atrophic forms the skin is often rough and harsh, perhaps scaly, and, in places, rather deeply pigmented. In the dark-skinned races it loses its natural glossy, velvety appearance, becoming grey, harsh, and coarse.

Different kinds of eruptions have been described as occurring in beriberi, but it cannot be said that there is anything like a

characteristic exanthem. In the epidemic of what was called "acute anæmic dropsy" which visited Calcutta[1] and Mauritius[2] in 1877–80, and which, on somewhat insufficient grounds, has been by some writers—Chevers,[3] for example—identified with beriberi, eruptions of various kinds — rubeolar, scarlatiniform, urticarial, petechial, and erythematous—were encountered. Careful observers of what were undoubtedly genuine epidemics of beriberi rarely make any allusion to similar skin lesions. I have only once seen anything like a peculiar eruption in beriberi. This was in a case of mixed beriberi, the paralytic element predominating. The eruption consisted of claret-coloured patches on the legs and thighs. At first the spots were about half an inch in diameter, slightly raised, disappearing on pressure, and shading off into sound skin. For some days they continued to enlarge, and as they enlarged the skin at the centre gradually assumed a natural tint while the ring of congestion faded, the spots finally disappearing in about ten or twelve days. Chevers alludes to a similar eruption. It was a sort of erythema multiforme, and can only be regarded as an accidental complication.

The œdema begins always under the skin over the crest of the tibia. In many cases it never extends further, and may soon disappear; but whether it subsequently disappears or not, at one time or another it is sure to have been present in this situation. As tested by pressure with the tip of the finger, the œdema seems to be of a firmer character than that met with in cardiac and renal disease; perhaps in this respect it might be said to be intermediate between the œdema of acute Bright's disease and the œdema of myxœdema. It may invade the subcutaneous tissues over the whole body, putting the skin everywhere on the stretch; or it may be confined to circumscribed spots, such as a hand, or the root of the neck, or the face, or the flanks, or the dorsum of one foot. Simmons says he has never seen this form of localised œdema; but, judging from my own experience, it cannot be so very rare as he represents. The swelling may be rapidly, or it may be more slowly developed; sometimes it may come in the course of a few hours, and it may disappear as suddenly, its disappearance being usually associated with profuse diuresis. The œdema in a mixed case may be set down as amounting to considerable swelling of the leg, foot, and ankle, puffiness of the face, some thickness about the root of the neck and flanks, and, perhaps, fulness about the arms, hands, thighs, and body generally; but there is no great swelling anywhere, and

[1] M'Leod, *Epidem. Soc. London*, 1893.
[2] Davidson, "Report on Acute Anæmic Dropsy," Mauritius, 1880, *Edin. Med. Journ.* 1880.
[3] Chevers, *Diseases of India*, 1886.

rarely any marked ascites. In a milder case the œdema may involve only the legs and feet.

Neuro-muscular System.

Anæsthesia of the skin.—Almost the first symptom of beriberi is the oft alluded to numbness of the skin (Fig. 44). It is developed symmetrically, commencing always over the front of the leg, where the initial œdema is to be found. It may not spread further; usually it extends considerably. Passing downwards it spreads to the dorsum of the foot, and upwards along the inner surface of the thigh. Should it continue to extend, the soles of the feet are next attacked, and, about the same time, the external surface of the thigh. Pekelharing says it always spares the groin and neighbouring surfaces. About the same time as the skin of the lower limbs is affected, or rather later, the anæsthesia appears on the inner surface of the wrist, spreading on to the back of the hand and along the fingers, attacking the finger-tips and palm last. Ascending the arm it first involves the inner surface and back of the forearm, the elbow, and, perhaps, the shoulder. More rarely is the skin over the chest affected, although that over the lower part of the abdomen is not infrequently markedly anæsthetic. Simmons found that numbness around the mouth was a common feature in Japanese beriberi; but Pekelharing did not remark this symptom in Java; and, though I frequently inquired for it among Chinese patients, I was seldom satisfied that it was actually present in any of them. Baelz records a case in which sensation was dulled over the entire cutaneous surface; such an extreme degree of anæsthesia is rare. The intensity of the symptom, as ascertained by the æsthesiometer, varies from slight numbness to almost if not complete anæsthesia. The perceptions of heat, cold, pain, and electrical stimuli diminish *pari passu* with the loss of common sensibility. Pekelharing mentions a case in which the patient could distinguish heat from cold in every situation except on the front of the legs; when the warm tube was passed over this surface he declared that it felt cold.

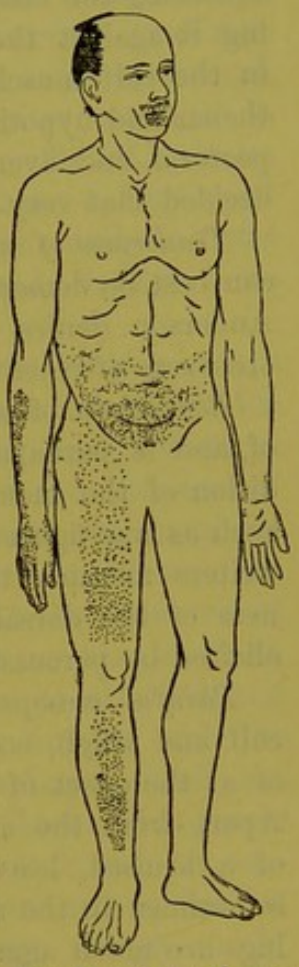

FIG. 44.—Areas of anæsthesia in both forms of beriberi, according to Simmons.

Paræsthesiæ of various kinds are common. Pricking, tingling,

and formication may precede the development of the anæsthesia, and also occur in the course of the disease. Burning of the soles of the feet is often referred to, especially by Indian writers; but there seems to be some doubt as to the connection of this symptom with beriberi.

Hyperæsthesia of the muscles, from the outset to the establishment of convalescence, is always, or nearly always, a well-marked symptom. It is present in all affected muscles, and can easily be elicited by squeezing the body of the muscle between the fingers, or compressing it against the underlying bone. It is most easily ascertained in the calf muscles, in those of the thigh and forearm, and in the thenar and hypothenar muscles; and it is often well marked in the pectoral, and even in the intercostal muscles. Sometimes it is so decided that very slight pressure causes exquisite suffering.

Tenderness of nerve trunks is also a fairly constant symptom, and can best be demonstrated in the crural, peroneal, and radial nerves. Anderson denies the presence of this symptom, but most other observers of recent times describe it.

Rachialgia of a very intense character is sometimes complained of, and is perhaps associated with an inflamed or congested condition of the meninges of the cord such as Bentley describes,[1] and such as was figured and described by Malcolmson. Both of these writers mention this symptom. Cousland[2] also alludes to a tenderness of the dorsal spine which in some of his cases was readily elicited by percussion.

Painful cramps in certain muscles, especially in those of the calf and thigh, occurring principally at the outset of the disease or at the onset of severe exacerbations, are of frequent occurrence. Apart from the existence of cramp, patients sometimes complain of a bruised, heavy, constricted feeling in the calf muscles, and sometimes in the soles of the feet and thighs; these painful feelings are much aggravated when the muscles are put in action, as in walking.

Precordial and epigastric pain is frequently an urgent symptom. The feeling is variously described as one of oppression, of distension, or of constriction. It is much aggravated by a full meal, and by pressure over the epigastrium.

Reflexes.—In a few cases, especially at the outset, the patellar tendon reflex is exaggerated; but, as a rule, it is quickly lost, and remains in abeyance throughout the disease. The skin reflexes, at all events the cremasteric, abdominal, and epigastric, are usually

[1] *Thesis.* Edinburgh, 1889.
[2] *China Med. Mis. Journ.* ii. 1888.

preserved; I have seen an instance, however, in which they disappeared one after the other. The case was one of extreme muscular atrophy. As improvement set in the reflexes gradually returned.

Ankle clonus is not developed.

The muscles are sometimes swollen, sometimes atrophied; and both atrophied muscles and swollen muscles may be found in the same patient, and even in the same physiological group. When swollen they feel hard and firm to the touch, and such muscles when they contract may be thrown into circumscribed swellings. Anderson remarks that indurations in the gastrocnemii, symmetrical, and apparently confined to the inner belly, are often met with in chronic cases. When examined electrically the affected muscles and nerves are found, after passing through perhaps a very short stage of increased irritability, to exhibit signs of diminished irritability for both galvanic and faradic currents, and, finally, those characteristic of partial or complete degeneration.

According to Pekelharing, the order in which the various groups of muscles are attacked is as follows:—First, and invariably, the muscles supplied by the peroneal nerve. Next, and almost as frequently, the muscles of the calf. Then follow the extensors of the knee and the glutei. After these the flexors of the knee, the adductors, and the flexors of the thigh. When the disease has attacked the thigh muscles it is fairly certain that the extensors of the hand and fingers, and the supinator longus are also involved, giving rise to wrist drop. The triceps may then be attacked, and the flexors of the hand, the muscles of the fingers, and the interossei. The abdominal muscles, the diaphragm, and the intercostals may now become implicated. Pekelharing has never seen the sterno-mastoid nor the trapezius attacked, and he considers that the biceps and the pectoral muscles are among the last to be affected. The facial muscles are sometimes, though rarely, involved; and the same remark applies to the extrinsic muscles of the eye. The muscles supplied by the hypoglossal nerves are, apparently, always spared; but the laryngeal muscles are very frequently implicated.

This is the usual order in which the muscles are attacked, but it is a rare thing to see anything like a general implication of the whole of the voluntary muscular system. Usually the legs and forearms only are affected; at the same time other muscles may be picked out in a more or less capricious way.

Pekelharing and Winkler's observations on the neuro-muscular symptoms in the very earliest stage of beriberi—in what might be

called the preberiberic stage—are of high pathological interest, and may possibly have in some circumstances a practical value. They say that in early beriberi, when neither medical man nor layman would venture on a diagnosis, when even the patient himself hardly feels indisposed, and long before he has any locomotor troubles, or even before he has the slightest sign of pretibial œdema, beriberi can be diagnosed with certainty by electricity. They assert that there is always to be discovered quantitative and sometimes qualitative changes in the reaction of the muscles to both forms of electricity, which, though not classifiable as "reaction of degeneration," are of great significance. Similar changes as regards sensation may be elicited by careful measurement of the tactile zones of the skin in front of the leg. These zones are always conspicuously enlarged in the preberiberic stage; and this even in those who, though they may not subsequently pass into a state of confirmed beriberi, are simply resident in a beriberi district.

The Circulation.

The blood.—Contrary to the opinions of many writers on beriberi, the carefully conducted observations in Brazil, Japan, and Java, particularly those by Magalhães,[1] Wallace Taylor,[2] and Eykman,[3] have shown that beriberi has nothing whatever to do with anæmia. In course of time, and after long illness, anæmia may develop; but in that case it follows the beriberi—is a consequence, not a cause. A large proportion of the inhabitants of hot climates, partly from poor living, partly from malaria, perhaps from anchylostomiasis, are anæmic, and these as well as the full-blooded may be attacked with beriberi; but in such cases the anæmia can be regarded only as an accidental concomitant. Strange to say, in Japan it has been remarked that it is the full-blooded, fresh complexioned, robust peasant or sailor who is most liable to the disease on entering an endemic area—not the pale-faced and limp townsman. Taylor gives figures showing that of 973 beriberi patients 916 were of strong constitution, 42 of average constitution, and only 15 are described as being weak. In 134 cases in which the corpuscular richness of the blood was carefully estimated, this amounted on the average to 94 per cent. of the normal standard, many cases being considerably above it. There seems, however, to be some diminution in the hæmoglobin value of the corpuscles; for his average, as regards this, in 101 cases was only 81 per cent. of the normal. Magalhães and Eykman's observations practically correspond with

[1] *Gazetta Medica da Bahia*, 1881.
[2] *Studies in Japanese Kakké.* Osaka, 1886.
[3] Pekelharing and Winkler, *loc. cit.*

these. There is no excess of white corpuscles, and no poikilocytosis. There is no indisposition on the part of the corpuscles to arrange themselves in rouleaux; no excess of crenated corpuscles, microcytes, or granules if the blood slides are properly prepared. Wernich's theory of pernicious anæmia, and similar theories by other writers, therefore do not stand the test of precise observation. The pallor of the face, usually so obvious in cases of wet or mixed beriberi, is often entirely attributable to the tension of the skin and consequent emptying of the superficial capillaries.

The pulse, as tested by finger and sphygmograph, shows that the dynamic condition of the circulation is one of relaxed arterial and vasomotor tension, with diminished cardiac power. Taylor, speaking from a large and carefully recorded experience, describes a beriberi pulse-tracing as being characterised by—(*a*) very sudden and high upstroke; (*b*) precipitous descent from the apex of the percussion wave; (*c*) dicrotism; the sudden and high upstroke indicating cardiac excitement, the precipitous descent diminished vasomotor tone. The relative amounts of cardiac weakness, irritability, and vasomotor paresis are subject to variations in the same and different cases, and the indications of the pulse are in harmony with this. In nearly every case of beriberi the pulse is very much accelerated by the erect position, or by any kind of effort. Irregularity of pulse, or a purple hue of the extremities, are of grave import.

Palpitation of the heart occurs in a large proportion of cases.

Bruits—generally systolic in character—over different valve areas of the heart, coming and going, are usually heard.

Increase of the precordial dulness, sometimes very marked towards the right side of the sternum, points to dilatation of the right side of the heart, and is a very common and sometimes rapidly developed condition. The heart's impulse is then diffused, and there may be much dilatation of and *pulsation in the veins of the neck*. The carotids, too, are often observed to throb violently, and pulsation is frequently visible in the thoracic and abdominal aorta.

Hydropericardium, to an extent sufficient to admit of positive diagnosis, is sometimes, though not very often, present.

The correct observation and interpretation of the symptoms connected with the circulation is a matter of prime practical importance in the management of a case of beriberi, and cannot be too carefully attended to. Extreme degrees of muscular atrophy, paresis, or even of œdema, provided the circulation is not seriously implicated, are of little importance as affecting life. But when signs of cardiac or vascular implication set in there is no telling

when, or how soon, death may supervene. Taylor, judging from his own experience, is inclined to consider relaxation of vasomotor tone a graver condition than cardiac weakness; therefore he regards a pulse-tracing showing a precipitous fall from the apex of the percussion wave and dicrotism as of graver import than weak pulse or feeble cardiac impulse.

Organs of Respiration.

Respiration may be affected in several ways, the principal of which are paresis of the diaphragm, paresis of the intercostal muscles, paresis of the muscles of forced respiration, paresis of the laryngeal muscles, œdema of the lungs, and hydrothorax.

Paresis of the diaphragm is indicated by falling in of the epigastrium during inspiration, and by a respiration entirely thoracic.

Paresis of the intercostal muscles is indicated by absence of thoracic breathing, exaggerated abdominal respiration, and feebleness of the expiratory effort on coughing being attempted.

Paresis of the laryngeal muscles is indicated by aphonia along with inability to produce an explosive cough. The inability to cough is very much increased if, along with the laryngeal paresis, there is also paralysis of the abdominal muscles. In such a case, at most, only a husky wheeze can be produced; when the attempt is made the walls of the abdomen, instead of being kept rigid and fixed, are suddenly shot out by the descent of the diaphragm forced down by the contraction of the intercostals and accessory muscles of expiration. The gravity of this condition, should œdema or other trouble in the lungs supervene, is very great.

Hydrothorax.—The frequency of extreme examples of this condition, as of hydropericardium, has been very much exaggerated. Anderson failed to note a single instance in upwards of 1000 cases of beriberi. Small collections of fluid are constantly found in pleuræ and pericardium in most post-mortem examinations of cases of wet beriberi; but the amount is insignificant as a rule, and could not have exercised any serious compression on the underlying viscera.

Œdema of the lungs is a complication of the gravest moment, and its occurrence is often the event which determines death. Like the œdema elsewhere, its advent may be gradual or sudden. Its development is signalised by great restlessness, dyspnœa, often a harassing cough, and, perhaps, vomiting, with the ejection of large quantities of frothy, blood-stained fluid from the air passages. Harsh breathing is heard on auscultation all over the lungs, and later—if death does not speedily ensue—fine moist crepitations. The distress is intense and terrible to witness. As signs of asphyxia

increase the patient loses consciousness, and usually dies within from twelve to seventy-two hours from the beginning of the attack (Anderson).

Abdominal Viscera.

Ascites is seldom a marked symptom, even in cases of extreme anasarca.

Diarrhœa.—I have met with cases in which the development of the symptoms of beriberi was preceded by an attack of diarrhœa; and this so frequently that it has occurred to me that the relationship may not be altogether one of coincidence merely.

Digestion.—In ordinary cases of beriberi the digestion is not seriously affected. As already remarked, appetite is usually unimpaired, and the bowels, as a rule, in the dry forms act fairly well. In the wet forms constipation is apt to occur. Dyspepsia at times is troublesome, the digestion of rice being said to be effected with difficulty—a full meal of this causing distension and much distress from pressure on the weakened and labouring heart.

Vomiting, unless attributable to ordinary indigestion, is always a grave sign, pointing probably to extending implication of the pneumogastric nerve. It is often the precursor of death. It is so regarded by the Japanese when associated with palpitation and dyspnœa. For a few hours before death the vomiting may subside; but as this is probably owing to exhaustion of the muscular walls of the stomach, abdomen, and diaphragm, the apparent improvement must not be looked on as necessarily a favourable sign.

The urinary organs are not affected, as far as known, in any specific way in beriberi. Albumen may be found in the urine, but its presence is attributable either to some accidental complication or concurrent disease, or to blood stasis during the death struggle. There is at times some difficulty in urinating from slight vesical paresis, or, more probably, from weakening of the abdominal muscles; it is seldom so urgent as to call for the employment of the catheter. In the wet forms of beriberi the urinary secretion may be reduced to a few ounces per diem, or it may be completely suppressed. Disappearance of the œdema is generally accompanied by profuse diuresis.

Temperature.—All observers are agreed that in advanced beriberi there is no febrile temperature, if anything, that the heat of the body is sub-normal; more particularly is this so towards the conclusion of fatal cases. But there is not the same unanimity with regard to the temperature in the earlier stages. In reports of cases we frequently read of the symptoms supervening on an attack of fever, or of fever declaring itself a few days after the commencement

of the premonitory numbness. That fever does occur at this time in many instances I do not think there can be the slightest doubt; but it would be rash to say that it is always a feature in every case, or to be positive as to its exact relationship to the other symptoms when it does occur. It has yet to be determined whether we are to regard it as simply an exciting or predisposing cause, or as a coincidence, or as an integral part of the disease, or as merely symptomatic of the neuritis. Cases rarely apply at the commencement of the disease, which is the time when fever occurs; as a rule, they only apply after paresis or œdema have become troublesome. Some years ago, in Hong-Kong, I had the opportunity of watching, in a small number of attacks, the development of beriberi from its commencement. The patients had been admitted to hospital for various non-febrile surgical diseases. As a matter of routine their temperatures were being regularly taken night and morning for some time before beriberi declared itself. They were quite free from fever on admission, and for a considerable period thereafter showed no signs of beriberi. I may cite one case as a type of what occurred in others. The patient was admitted with syphilitic fungus testis on the 30th July 1888, and was rapidly improving on iodide of potassium. About the 16th of August he began to complain of numbness of the legs and finger-tips, but continued otherwise in good general health. At 5 P.M. on the 20th of August he was seized with a rigor which persisted for three hours; at 9 P.M. his temperature was found to be 103°·6 F. Notwithstanding the fever he slept well; but on waking next morning he could neither walk nor stand, and his feet, hands, and face soon became swollen and puffy. He passed through a smart attack of beriberi of the mixed type. Altogether, in this little epidemic among my hospital patients, there were seven such cases of beriberi; and in each of these, soon after the appearance of the premonitory anæsthesia, pyrexia more or less marked, sometimes severe and preceded by rigors, occurred. It was likewise noted that exacerbations in the early stage were frequently attended with fever, generally slight, although in one instance the temperature rose to 104° F.

Mode of Death in Beriberi.—In a few instances death seems to be brought about, after a prolonged period of suffering and debility, by simple asthenia; occasionally it results from syncope dependent on sudden paresis and collapse of the weakened heart; but in a majority of instances the cause of death is a more or less rapidly developed asphyxia of a somewhat complicated mechanism.

Anderson gives a very graphic description of the closing scene in such cases. He says: "The grave symptoms may appear without

warning, nearly always developing in the course of a subacute or chronic attack. A patient comes under treatment for 'kakké' of apparently an ordinary character; he is usually strong and well nourished, has no sign of anæmia, and little or no œdema; the disease progresses in the usual manner, and no evil is anticipated, when suddenly rapid action of the heart, strong pulsation in the neck, and difficulty of breathing appear, with a distressing pain in the abdomen. Soon afterwards the patient vomits; and while an observer unaccustomed to see the disease still apprehends no danger, the Japanese doctor recognises the commencement of 'shiyôshin,' and predicts that the man 'will surely die.' During the next few hours the breathing becomes more embarrassed, the pulsations of the heart more and more accelerated, and vomiting occurs from time to time. The patient can lie down no longer; he sits up in bed or tosses restlessly from one position to another, and with wrinkled brow, staring anxious eyes, dusky skin, blue parted lips, dilated nostrils, throbbing neck, and labouring chest, presents a picture of the most terrible distress that the worst of diseases can inflict. There is no intermission even for a moment, and unless active treatment be at once resorted to the pulse fails, the temperature sinks, and at length the brain, paralysed by the carbonised blood, becomes insensible, leaving the dying man to pass his last moments in merciful unconsciousness."

The course of events leading up to this conclusion, which is practically an asphyxia, appears to depend primarily on paresis of the cardiac muscle. The left ventricle has nothing very much to contend against beyond its own imperfect innervation, and the vasomotor paresis in the arterioles. But it is otherwise with the paretic right side of the heart, as it may have, in addition to contending with its own weakness, to drive blood through lungs imperfectly expanded in consequence of paresis of diaphragm or intercostal muscles; besides having to struggle with the effects of vasomotor troubles, which may be present in the vessels of the lungs as elsewhere. As a consequence of this the right side, although like the left it generally undergoes a certain amount of compensatory hypertrophy, becomes overdistended, and fails to empty itself properly in systole. Gradually it becomes more dilated, and in proportion to the dilatation more weakened, passing into one of those hopeless vicious circles so common in pathology. It can readily be understood how in such circumstances a slight increase in the paresis of the heart itself, or of the intercostal muscles, or of the diaphragm, the occurrence of some pulmonary trouble which under other conditions would be of very trifling importance, or, above all, the

development of œdema of the lungs, by throwing extra work on the enfeebled organ, may cause further dilatation,—further stagnation of blood. Finally, the dilatation reaches such a point that the over-stretched heart muscle loses power altogether, and ceases to contract on the mass of blood which now distends it almost to bursting.

Such occurrences as hydropericardium, or hydrothorax, or over-distension of the stomach, may aggravate this condition; but they are seldom present in extreme degree, and are therefore seldom the actual cause of death.

Pathological Anatomy.—*Anasarca.*—In nearly every case of beriberi which comes to the post-mortem table, even although the external appearances of œdema may not be very marked, the subcutaneous cellular tissue, and the connective tissues of all the organs are found to be infiltrated with serous fluid, which is sometimes tinged red. An excess, sometimes considerable, of fluid is found in the serous cavities also. On the other hand, in a small proportion of extreme examples of the atrophic variety the tissues may appear abnormally dry.

Ecchymoses are very generally found under the pulmonary pleura, under the visceral layer of the pericardium, and about the serous coat of the abdominal viscera. Hæmorrhages, perhaps of considerable extent, are occasionally found in the substance of, as well as between the muscles. Smaller hæmorrhages are found at times in the sheaths of nerves, both in their continuity and at the point of emergence from the spinal cord. Sometimes very minute hæmorrhages are seen in the substance of the brain.

The blood is generally said to be acid in reaction, very fluid and very dark, unusual quantities escaping even from the small veins when cut across. Clots are few and not firm.

The muscles which were affected during life are either atrophied or swollen, and in colour brownish-red or pale red. They are dry and shining in the dry form, or infiltrated and macerated in the wet form. Both atrophied and swollen muscles may coexist in the same body. Under the microscope the individual fibres are seen to be wasted and in a state of granular or vitreous degeneration, and tending to break up into fibrillæ. Striation is feebly marked or absent; and, generally, most of the fibres are barely half the normal thickness, many being reduced to sarcolemma only. An increase of the nuclei and of the interfascicular tissue, especially along the course of the vessels, is apparent, particularly in chronic atrophic cases.

The lungs are generally gorged with blood, and may be œdematous. The amount of fluid in the pleura varies much, but it is seldom very great; sometimes it is sanguineous.

The pericardium nearly always contains an excess of fluid, but not usually to so great an extent as to seriously compress the heart. Pekelharing's measurements of pericardial fluid in sixty-four autopsies are as follows:—

Little or no serosity, ...	2 times.
From 20 to 50 grammes, . . .	20 „
„ 50 „ 100 „ . . .	13 „
„ 100 „ 250 „ . . .	17 „
Over 250 grammes, ...	12 „

The heart.—If death has been brought about by the slow asphyxia already described, the right side of the heart and large veins will be found to be enormously distended with dark fluid blood containing a few clots. If death has been by sudden syncope the appearances will correspond. Baelz says that the heart does not generally look degenerated or fatty to the naked eye; nevertheless, when examined microscopically, the muscular fibres may be found, though not invariably, to be much degenerated. Sometimes the striation is seen to be distinct, and little evidence of degeneration can be detected in the fibres; the nuclei of the muscle, however, are always degenerated and surrounded by fat granules and pigment, and, moreover, they are increased in number. At different points minute foci of inflammatory infiltration are visible under the microscope.

The cavities of the heart are always dilated; those of the right side most so, and often enormously. Both sides are also hypertrophied, especially the left. Muscular degeneration, according to Baelz, is generally most apparent in the right side.

Abdominal cavity.—Nothing characteristic of beriberi has been found in the abdominal viscera. Some writers refer to fatty degeneration of the liver and kidneys, and others to enlargement of the spleen; but these are by no means constant features, and are attributable to other causes. In Orientals, intestinal parasites are common in all complaints, consequently one must expect often to find them in beriberi; they have no etiological relationship to the disease.

Nervous system.—The condition of the nervous system in beriberi has been the subject of much careful and fruitful study of late years. To Baelz belongs the merit of having first distinctly described the specific alterations in the peripheral nerves, as well as those in the muscles. Pekelharing and Winkler, with more extensive opportunities, have confirmed and somewhat extended these researches in the pathological histology of beriberi, as they have, in the same way, confirmed and extended those of Scheube on the electrical phenomena.

Although Baelz's observations were founded on a very limited number of cases, they have been completely borne out by the detailed examinations of Pekelharing and Winkler on material from eighty-five autopsies, in none of which did they once fail to detect the characteristic signs of nerve degeneration.

According to Baelz, a degree of congestion, probably not pathological, is found in the cord and meninges.[1] In a proportion of cases —one in five—a certain amount of degeneration of the cells of the anterior cornua is present; and, according to Pekelharing and Winkler, there may be a slight diminution in the number of fibres in the prolongation of the posterior roots of the spinal nerves, but without swelling of the axis cylinders or augmentation of nuclei. Otherwise the great nerve centres are healthy. These trifling changes may be regarded as being merely secondary to the peripheral lesions, which are pronounced and indubitable.

In acute beriberi a degeneration of the peripheral nerves, analogous to that resulting from experimental section of nerves, is sometimes, though not invariably, seen. In this case there is a disaggregation of the medullary matter, and a proliferation of the nuclei in the sheath of Schwann. Pekelharing points out that these early changes are best studied in the phrenic and vagus.[2]

In the affected nerves, in every case of beriberi, there is seen to be a great increase in the number of the nuclei in the endoneurium, and, often, a proliferation of the nuclei of the blood vessels of the nerves. The principal evidences of disease are found in this and in the destruction of the medullary sheath, always most marked and advanced near the nodes of Ranvier. The myeline aggregates into small shining masses, like fat granules; and, at a more advanced stage, disappears altogether, leaving the sheath in contact with the axis cylinder. In such nerves the incisures of Schmidt cannot be made out; and, at a later stage, the axis cylinder itself may disappear. The fibres of the sympathetic, so numerous in the vagus, appear finely granular, and show multiplication of their nuclei.

In chronic cases of beriberi there is a hyperplasia of the endoneurium of a gelatinous character; it may be seen to surround as a ring some as yet undegenerated nerve fibres. This homogeneous,

[1] Malcolmson and Bentley describe appearances suggestive of actual meningitis at the lower part of the cord.

[2] The reason for this is obvious. Acute degeneration of other nerves is not necessarily or immediately fatal; acute degeneration of vagus or phrenic, if involving many fibres, is necessarily and rapidly fatal. Consequently, those nerves, when they come to be examined in suitable cases, not having had time for regeneration or secondary changes, exhibit those earlier phenomena in their purest and most typical form.

shining, easily stained hyperplasia is most marked near the blood vessels of the nerve. The sheath of the nerve is little affected; but most of the contained fibres are atrophied or considerably diminished in size.

These lesions are only seen in the peripheral nerves, and diminish in extent and intensity as the nerve is traced towards the spinal cord. They never extend into the anterior roots; in the posterior roots they may sometimes be traced, being always more distinct, however, on the peripheral than on the proximal side of the ganglion.

In the bodies of all beriberics signs of regeneration of nerves are found along with the signs of degeneration.

Etiology.—In attempting the discussion of this important matter, it may conduce to clearness of understanding if we divide the so-called "causes" of beriberi into the following categories:—

1st. The specific cause.

2nd. The conditions under which this is generated.

3rd. The circumstances under which it is absorbed by the human body.

4th. The circumstances influencing the susceptibility of the human body—the predisposing causes.

5th. The length of incubation.

6th. The circumstances determining the explosion of the disease.

7th. The question of infection.

In the discussion of obscure etiological problems these points are too frequently mixed up in inextricable confusion, and in no subject more so than in the present.

1*st. The Specific Cause.*

That beriberi is the result of the action of some specific agent on the human body does not admit of doubt. The well defined characteristics of the disease, and the circumstances under which it is acquired, all point to this. It cannot be the result, as has so often been vaguely asserted, simply of heat or cold, damp or drought, too much of one element of food or too little of another. Such conditions are met with everywhere and anywhere. But we know that beriberi can be acquired only in particular places; and, as far as known, is never met with unless in individuals who live in these places, or who have visited these places. When a healthy seaman visits a kakké port in Japan, or a healthy Dutch soldier is landed in Atcheen, or a healthy criminal is lodged in an Indian jail, and by and by becomes beriberic, it is not because of any unusual exposure

to heat or cold, dampness or dryness, nor of unphysiological feeding that he sickens; it is simply because he has had an opportunity in these places of absorbing a specific poison. Heat and cold, damp and drought, or an imperfect dietary, he might be subjected to in any place,—Madeira, for example,—but no amount of these would lead to beriberi there. Again, the inhabitants of a district, of a town, or the inmates of a building such as a jail or school, may for years have enjoyed perfect immunity from beriberi; yet, without any change of climate, dietary, character of work, or other manifest alteration of the conditions of life, beriberi suddenly breaks out among them. Such an occurrence must surely be the result of something new and specific which has been introduced from without. Lastly, a ship sails from a beriberi port. Weeks or months after she has left the land her crew, one after the other, is struck down with beriberi. In this case the timbers or cargo of the ship, or the provisions, must have become contaminated with a poison, or the sailors must have carried in their bodies or about them something brought from the beriberi port, which, though inoperative for a time, ultimately on certain conditions arising bursts into activity.

This "something," which is found only in particular localities, which can lie dormant for weeks or months, and which can multiply and spread, which can be transplanted to fresh localities and spread, and which can cling about men's bodies or their ships, must be particulate and living matter of some sort; not simply a gas, or fluid, or solid chemical substance, or an immaterial force. This living "something" is the germ or virus-producing germ of beriberi.

The conditions under which this germ becomes active are partly understood; but, unfortunately, very little is known about the thing itself, and it is still an open question if it has ever been separated, or even recognised.

There are two principal views entertained at the present time as to the nature of the beriberi agent, and which it is desirable to allude to. It is maintained by some that the beriberi agent is an animal parasite; by others, that it is a microbe.

(*a*) *Beriberi considered as a helminthiasis.*—The grounds on which this doctrine has been built are altogether inadequate; but, because it has led to considerable confusion and to some false hopes, it must not be ignored, and had better be discussed before we proceed to the more promising microbic hypothesis.

The principal advocates of the view that beriberi is a helminthiasis are Erni, a physician in Sumatra, and Kynsey.[1] The former, remarking the frequency with which *Anchylostomum duodenale*,

[1] *Report on Anæmia, or Beriberi of Ceylon.*

Trichocephalus dispar, and the larvæ of certain flies were met with in the bodies of the subjects of beriberi, arrived at the conclusion that the disease itself was caused by these parasites. Kynsey maintained that the disease called "beriberi" in Ceylon was really anchylostomiasis; and he ventured to generalise somewhat, and to express the opinion that the beriberi of other countries was of the same nature and origin, in fact, that beriberi is anchylostomiasis. Kynsey, unfortunately, gives no specific details of his cases. As far as the general description which he gives goes, it might apply either to beriberi or to anchylostomiasis. The nearest approach to precision in this respect in his paper is in an introductory note in which, referring to the epidemic of beriberi on board the Chinese transport *Toonan*, recorded in the *Lancet* of 23rd July 1887, he says that the cases he met with in Ceylon were exactly similar to these. There can be no doubt, from the description given of these Chinese cases by Drs. Slater and Oliver, of Newcastle, that they were examples of genuine beriberi. From this we are entitled to infer that at least some of the cases Kynsey met with in Ceylon were genuine beriberi likewise, and that therefore he attributed genuine beriberi to anchylostomiasis.

But, if the anchylostomum produces beriberi in Ceylon and Sumatra, why do we not hear of beriberi in Egypt, where the anchylostomum is so common; why is it not reported as one of the features of the many epidemics of anchylostomiasis occurring in Europe; and why is beriberi not found at the present time in Italy, where the anchylostomum is so frequently met with, as Sonsino, dealing with the neighbourhood of Pisa, has shown?

The symptoms of beriberi and anchylostomiasis are very different; in the former the symptoms are those of a neurosis, in the latter of an anæmia. The diseases ought not to be confounded. How a parasite lodged in the alimentary canal could produce anæmia directly by the abstraction of blood, or indirectly by interfering with nutrition, we can readily understand; but how it could produce a neuritis in the legs and arms we cannot understand. It is equally difficult, on the helminthiasis hypothesis, to explain the sudden deaths so common in beriberi in non-anæmic cases. Pekelharing and Winkler state that more than once they have seen cases of beriberi in which the patient, a soldier, was so well in the morning as to have drilled as usual, and even to have made centres at target shooting; and yet, before night, the same man was dead. Such cases of malignant beriberi are not to be explained by a few minute parasites in the intestine.

The fact is that in many tropical countries anchylostomum,

trichocephalus, and other intestinal parasites are exceedingly common in certain districts. Giles[1] has shown how prevalent anchylostomum is in Assam; Baker[2] and Leslie[3] have done the same for Burma; Scott,[4] Kerr and Rhodes[5] for Penang; Erni, Pekelharing and Winkler for the Dutch Indies; and Walker[6] for Borneo. It is also well known to be very common in the West Indies, and, doubtless, in many other places. Pekelharing and Winkler never failed to find it when they looked for it in a native, no matter what may have been the nature of the disease which caused death. Kerr and Rhodes found that it was so common in Penang that their difficulty was to come across cases without it; they did, however, find several cases of beriberi in which no anchylostomum could be detected; and, moreover, both they and others remark that the severity of an attack of beriberi bears no relation to the number of parasites present. Although Giles attributed the disease called "kala-azar" or "beriberi" in Assam to the anchylostomum, he was careful to point out that this disease—miscalled beriberi—was simply an anæmia, and a genuine anchylostomiasis; and not the peripheral neuritis described by Pekelharing and Winkler and others. Leslie says that in 11 post-mortems in fatal beriberi in Schwebo, Burma, although special search was made for the anchylostomum, it was not found in any instance; in 3 other post-mortems made by himself, a few were found in 1 case only; and he further states that in the stools of 11 cases of beriberi, to which thymol had been administered, the anchylostomum was not once discovered.

Doubtless, in consequence of faulty diagnosis, and led away by the teaching which attributed beriberi to anæmia, the earlier medical men and planters in Assam called and diagnosed anchylostomiasis "beriberi," and this name and diagnosis have been handed on from one set of medical men and planters to another; and, doubtless, a similar error in diagnosis has been made in Ceylon. But that all cases which go by the name of beriberi in Ceylon are only anchylostomiasis, as Kynsey maintains, I cannot believe. That beriberi exists in Ceylon now as formerly is probable, for we have Kynsey's own testimony that he has seen it there, or has seen cases exactly resembling it. Most probably there, as elsewhere where anchylostomiasis and beriberi are coendemic, there are to be seen cases of

[1] *Report of an Investigation into the causes of the Diseases known in Assam as Kála-azár and Beriberi.* Shillong, 1890.

[2] *Indian Medical Gazette*, December 1888.

[3] *On Beriberi*, G. C. Press. Simla, 1891.

[4] *Practitioner*, May 1892.

[5] "On Beriberi," Dr. Max Simon, *Brit. Med. Journ.* July 2, 1892.

[6] *Brit. Med. Journ.*, December 5, 1891.

uncomplicated beriberi, cases of uncomplicated anchylostomiasis, and cases in which both diseases, though entirely distinct in their respective natures, concur.

(*b*) *The doctrine of the microbic origin of beriberi* has long been a favourite one, and much work has been expended in the endeavour to establish it. The results of this work, however, have been so contradictory, and in several instances so extraordinary, that it is impossible to say in the present state of the question what to accept and what to reject.

On the assumption that the ultimate cause of beriberi is of the nature of a bacterium, two views suggest themselves: 1st. That a microbe is present in the diseased tissues, and by its presence there causes the disease. 2nd. That the disease is brought about by a toxic product generated by a microbe living either in (*a*) the blood or tissues, or (*b*) in the soil or elsewhere outside the body; in either case producing a substance which, on being absorbed, poisons the nerves, and so gives rise to the characteristic neuritis.

It would serve no useful purpose if I attempted to analyse the labours of the many observers in this field. Some of them have found micrococci, some of them have found bacilli, and some of them have found both micrococci and bacilli in the blood; and some of them have found mycelial growths as a result of their cultivations of the blood. But although there has been great discrepancy as to the morphological characters of the micro-organisms obtained, there is a suspicious general agreement that one and all of these very different bacteria, in the hands of their discoverers, produce paresis and nerve degeneration when injected into the lower animals. In these circumstances one is forced to conclude that all of these observers cannot be right. It would be invidious to suggest any one in particular as being in error; I shall therefore confine myself to a short description of the more recent investigations.

It is quite certain that bacteria cannot be demonstrated in the blood in beriberi in the same way and with the same ease as they can be demonstrated in the blood in anthrax or in relapsing fever; and also that they cannot be demonstrated in the nerves or other diseased tissues with the same ease and certainty as they can be in tubercular tissues. In common with many others, I have frequently examined the blood in beriberi for bacteria, both in fresh and in stained preparations, but I cannot say that I have ever satisfied myself of their presence there. Careful and competent observers, such as Baelz, Pekelharing and Winkler, have examined the tissues prepared in a variety of ways and according to modern methods, but they tell us they have always failed to recognise

pathogenic bacteria in these likewise. Others, on the contrary, such as Lacerda and Ogata, say they have seen both micrococci and bacilli in the diseased structures in beriberi. In this matter, just as in the matter of the examination of the blood, there is so much discrepancy in the results of different observers, that one cannot resist the inference that some of those who have attained positive results must have allowed their judgments to be influenced by preconceived theories, or that they have been misled by faulty experiment.

Possibly Pekelharing and Winkler have supplied the explanation of some of the apparently contradictory results in the case of the examination of the blood,—which ought to be a simple enough affair, and which ought always, if made under similar conditions, to afford uniform results, even to inexperienced observers. They say that in Atcheen (and inferentially in every beriberi centre), every one, both healthy and beriberic, after a few days' residence in the district has bacteria in the blood. When, however, the blood of either healthy or beriberic individuals is examined in a non-beriberic centre in the same country (Sumatra), it is found to be free from bacteria, unless the individual has come very recently from a beriberi centre, or has lately landed from a ship in which the disease had been contracted. That is to say, that no reinfection taking place from without, the bacteria taken in at a beriberi centre gradually die out, or pass into a resting state, and so can no longer be found in the blood.

According to Pekelharing and Winkler, these bacteria can be readily demonstrated in the blood in the usual way by staining dried cover-glass preparations with fuchsin or methylene blue; but they insist on this point, that to find them it is absolutely necessary that the patient should either be at the time in the beriberi centre, or only very recently have come from it.

The bacteria are seen to be of two kinds—granules and rods. They vary in size within wide limits, and also in shape and in reaction with staining agents. The rods seem as if in some instances they were dividing, and the cocci in some instances as if elongating; sometimes diplococci are seen. Frequently little groups, consisting of an agglomeration of all of those different forms, are encountered. The individual bacteria do not stain alike; some stain deeply at the ends of the rods, others irregularly, and some hardly stain at all as if degenerating. Some, too, are irregular in shape. As regards the numbers present in the blood, the Dutch savants say that this varies very much, even in the same individual. Sometimes every preparation is crowded with bacteria; at other

times it was only after a long search that unquestionable specimens of bacilli or cocci were found.

Pekelharing and Winkler remarked that in residents in Atcheen, even among the apparently healthy, feelings of weight and pains in the legs, palpitations, and sometimes shin œdema and anæsthesia, were very general. These symptoms they evidently wish to associate with the universal infection of the blood with micro-organisms; and they suggest the inference that declared beriberi in Atcheen is only a more pronounced form of a general but milder beriberic intoxication universally prevailing there.

With the view of ascertaining with certainty if any of the bacteria they describe were really etiologically related to beriberi, Pekelharing and Winkler attempted their separation and cultivation. They inoculated culture tubes with blood from twenty-four beriberics in whom the presence of bacteria had been ascertained. For the most part the tubes remained sterile. Out of many experiments in only 15 tubes was a culture obtained—in 12 of micrococci, in 3 of bacilli. In the 3 cultures of bacilli the organisms were all of different species. Of the 12 cultures of micrococci 2 failed on sowing them on solids, 2 produced yellow-coloured colonies, 10 white shining colonies. Of the 10 white cultures 1 was unlike the others in being composed of a different sized micrococcus which could not be subsequently cultivated, and 1 other culture was lost. Of the 8 remaining cultures of white cocci 6 only liquefied gelatine, 2 had not this property. With the 6 gelatine-liquefying cultures 9 experiments were made on 7 rabbits and 2 dogs, with, in most instances, a positive result. It was found that a single inoculation with beriberi blood, or a single coccus inoculation, had no effect. To produce an effect it was necessary to repeat the inoculations daily and for many days on end. In these experiments this repeated inoculation was carried out, with the result that a distinct degeneration of peripheral nerves, along with more or less paresis, was produced in 6 of the 7 rabbits, and in 1 of the 2 dogs. From these experiments Pekelharing and Winkler conclude that the white, gelatine-liquefying micrococcus, obtainable from beriberi blood, is the cause of beriberi.

They next attempted to ascertain whence these bacteria were derived, and through what channel they entered the human body. This they did by passing a large quantity of the air of beriberi infected places through neutral salt solution, thus collecting the various bacteria suspended in the atmosphere. Two rabbits and one dog were injected daily with salt solution thus charged with atmospheric microbes. Of the two rabbits one died on the seventh

day; on dissection, several of its nerves were manifestly degenerated —amongst others the superior laryngeal and the cardiac branches of the vagus. The other rabbit was killed after twenty-five injections; and in it, too, slight peripheral nerve degeneration was found in the nerves of the legs. The dog was killed after twelve days' treatment, but was found to be unaffected. From the blood of the first rabbit a culture of white, gelatine-liquefying cocci was raised, exactly like those procured from culture of beriberi blood. From this culture a dog and a rabbit were injected, which, on being killed after a time, were found to have suffered nerve degeneration. From this experiment the conclusion is drawn that the germ of beriberi is generated in the soil; and that, rising into the air as dust, it is inhaled, entering the human body by the lungs.

These elaborate investigations and experiments, made as they were by men of capacity, experience, and high standing, cannot be passed over. At the same time—and the distinguished investigators express themselves as aware of this—they cannot be said to settle the point. The difference in shape, size, and property of the cocci and bacilli invariably present in the blood in beriberi, requires explanation. Possibly the germ of beriberi is polymorphic, possibly some of the organisms met with are dead and degenerating forms. These and many other points require clearing up before definite views as to the relationship of Pekelharing and Winkler's micro-organisms to the disease can be considered as established.

If these views as to the *rôle* of this micrococcus are to be accepted, then the bacterium of beriberi occupies an unique place in pathology. In no other germ disease does the virus require to be constantly renewed from without; in no other germ disease does the germ, after thriving and multiplying for a time,—as we must, judging from the vast number of germs present in some cases, presume it thrives and multiplies for a time in beriberi,—die out slowly, and then be renewed from without again and again. In tubercle, syphilis, malarial fevers, and other chronic germ diseases, germs die out, but some are left which are capable of resuscitation, and which, on resuscitation, give rise to relapses. There are many other difficulties about accepting this theory, not the least important of which is the paucity of the experiments supporting it.

To some extent Pekelharing and Winkler's observations have been confirmed by Van Eecke,[1] who also obtained two species of coccus—a white and a yellow—from beriberi blood. Recently Musso and Morelli, of Monte Video, as reported by Leopold,[2]

[1] *Geneesk. Tijdschr. v. Ned. Ind.* vols. xxvii. and xxviii.
[2] *Berl. klin. Wochens.* Jan. 25, 1892.

obtained four species of micro-organisms from beriberi blood cultures, one of which, on being injected into thirty animals, produced in every instance œdema and multiple peripheral degenerative neuritis with muscular atrophy, and groups of cocci were found in the tissues of the heart.

If these observations are confirmed they should lead to a very great advance in our methods of dealing with beriberi epidemics, and give direction and precision to our efforts in contending with one of the deadliest and greatest pests of many warm countries. The matter is of such moment that it is astonishing our Indian, or some of our colonial Governments does not take it up. Apart from considerations of philanthropy and science, self interest of a pecuniary sort should stir them to action. The settlement of the question is beyond the resources of the ordinary medical pocket. In many of our colonies there are few diseases which so seriously tax Government hospitals, or which so seriously hinder the expansion of the agricultural, mining, and other industries on which the prosperity of these communities depends.

2nd. The conditions under which the beriberi germ generates.

These are damp, high temperature, and, possibly, contamination of the soil or surroundings by animal matter.

All observers are agreed as to the powerful influence of damp in conducing to the generation of the poison of beriberi. Dampness is a characteristic of all beriberi localities. Such is the condition of the low-lying beriberi towns of Japan. The same remark applies to the beriberi centres in Malaysia, Burma, and India; and such is the condition of the forecastles of beriberi ships. The epidemic in the Singapore jail, so carefully observed and recorded by Rowell,[1] is an illustration in point. This prison was built on what was originally a mangrove swamp. It was divided into two parts—a native and an European. The platform of earth on which the European portion stood was two feet higher than the foundations of the native portion, and, moreover, was kept from being flooded by a neighbouring creek by an effective retaining wall. At high tide the water was on a level with the floor of the native portion; and, on digging, water could be reached anywhere at from 18 to 36 inches from the surface. The two portions of the prison were contiguous, and the hygienic conditions, apart from the superior dryness of the European portion, appear to have been identical. Yet, whereas, between 1875 and 1880, 1274 cases of beriberi, of

[1] *Ann. Med. Rep. Straits Settlements Hospitals*, 1880-86.

which 176 died, occurred in the native prison, not one occurred in the European part of the jail in which, in addition to the European prisoners, the native female prisoners were lodged. Many similar instances proving the importance of damp in favouring the development of beriberi could be adduced.

The influence of high temperature is very manifest in countries like Japan in which, though the summer is hot, the winter is cold. In Japan beriberi cases begin to crop up about the end of April; they become more numerous in May, and still more numerous in June; in July, August, and September the epidemics attain their height; by October cases become fewer, and during the cold winter months fresh cases are rarely met with, and old cases, as a rule, gradually improve. In the tropics beriberi occurs all the year round, the principal meteorological agent influencing the rise and fall of the disease there being, apparently, the rainfall.

That the contamination of soil by animal matter has at times something to do with the generation of beriberi is suggested by the fact that beriberi is a house or town disease. Like diphtheria, typhus, typhoid, and yellow fever, it clings to inhabited places. In this respect it differs from malarial disease, which is always more rife and more deadly on the outskirts of towns and in the open country than in the large centres of population.

3rd. The medium in which the poison is generated, and the circumstances under which it is absorbed.

There is sufficient evidence to show that the poison of beriberi is not introduced into the human body by food or by water, and that it is a disease associated with locality, not with food or water supply. For example, in the case of the Singapore jail epidemic, above referred to, those affected drank of the same water supply as the other inhabitants of the town, and consumed the same provisions as the rest of the prisoners. But there was no general epidemic in Singapore, such as would certainly have been the case had the common water supply been the medium of infection; and the female and European prisoners would certainly have been attacked had the common food supply been the medium.

Experimental research, as well as common experience, point unmistakably to the soil and the immediate surroundings as the nidus of the germ. This being the case, the germ, or its products must first, before entering the human body, rise from the ground or walls into the atmosphere. As it appears that a certain intensity, as well as repetition of poisoning is necessary for the production of pronounced

disease, it is evident that such conditions of living as bring people for a considerable period close to the source of the poison, or which confine them in the poisoned atmosphere, or which prevent the poisoned atmosphere from being diluted by untainted air, must conduce to the development of beriberi. Hence the well recognised danger of sleeping on the ground in beriberi districts; hence the liability of prisoners and others accumulated, and constantly living in a beriberi-tainted atmosphere confined between high walls; and hence the value of ventilation and similar sanitary arrangement as preventives.

It is very commonly asserted that beriberi is caused by the agglomeration of large numbers of human beings in confined spaces; meaning thereby that the simple aggregation of people is sufficient of itself, without specific germ introduction, to produce the disease. I do not think that this is at all a satisfactory explanation of the acknowledged pernicious results of overcrowding. In the coolie houses, in the native hotels, in ships, in native schools and hospitals, and even in private houses in Eastern towns, the overcrowding is generally excessive. Cubic space per head has to be reckoned by tens of feet, not by hundreds or thousands, as every one practically acquainted with these towns very well knows. Overcrowding is the rule in these places; but beriberi is met with only in a small proportion of the instances, even of extreme overcrowding. It is manifest, therefore, that overcrowding is not in itself sufficient to produce beriberi. It is with beriberi in this respect as it is with infectious diseases; overcrowding favours their occurrence and spread, but it does not create them. No one can say that overcrowding can create a germ of any sort, a pediculus, an acarus, or a spirillum; neither can it create a beriberi germ. But if a disease germ is present in any particular building, all who live in this building are liable to be attacked by this germ. If there is only one individual, he may absorb it; if there are a thousand individuals, a thousand individuals may absorb it. In the former case, supposing the germ to be that of beriberi, beriberi would be said to be sporadic; in the latter, epidemic, and very probably the outbreak of disease would be described as an "epidemic of beriberi caused by overcrowding." The disease, however, was not produced *by* overcrowding, but *in* overcrowding.

Another reason why beriberi is so frequently met with in jails, in schools, and such like communities, is that the soil and surroundings are in such circumstances exceedingly liable to inoculation from without owing to the continual influx of fresh individuals. Some of these may bring in their clothes, in their food, or in their bodies, germs from other beriberi centres, which, falling on suitable media,

germinate and cause an outbreak of disease. The liability to the introduction of the beriberi germ in any given place is proportionate to the number of people coming into it, and to the number of different centres from which new-comers are derived; and the liability of any given individual, in any given place, to catch beriberi is in direct ratio to these circumstances; the greater the numbers, the greater the risk of some one carrying with him the germ of the disease, and the greater the risk to each individual.

Simmons points out an interesting feature with regard to the carrying by the prevailing winds of the beriberi poison from an infected centre to an uninfected spot; suggesting thereby another, among the many analogies existing between this disease and ordinary malaria.

4th. Predisposing causes.

Certain circumstances seem to have an influence in determining the susceptibility of individuals to the beriberi poison.

Age.—Beriberi is said to be very rare before the tenth or fifteenth year and after the seventieth; between 20 and 30 seems to be the most susceptible period.

Sex.—Females are said to be less liable than males. Corre[1] says that in Japan the proportion of females affected to males is only 1 to 27; Anderson says 1 to 3. On the other hand, Cousland records an epidemic in a girls' school in Swatow, China; and Corre refers to an epidemic in an Annamite convent, and two other epidemics among girls in the same neighbourhood. Da Silva Lima gives the proportion of the sexes in those attacked in Brazil as 28 males to 22 females. In estimating the proportion of the sexes affected by any disease in Eastern countries, large allowance must be made for the social prejudices which keep females from attending hospitals and dispensaries. This, together with the frequency with which large bodies of men, as compared to women, are crowded together in lodging-houses, factories, jails, ships, and camps, probably accounts for a good deal of the apparent relative immunity of the female sex. Exposed to the same conditions as men, doubtless they will contract the disease quite as readily.

Pregnancy and the puerperal state seem to be very provocative of attacks. Eldridge says that in Hakodate "beriberi is the curse of parturient women." Simmons records a case of recurring beriberi in a Japanese woman who was attacked in each of three successive pregnancies. Many other observers note this susceptibility of pregnant women.

[1] *Maladies des Pays Chauds.*

Race.—It is impossible to say from the data at our command that one race is more susceptible to beriberi than another. That one race is more *liable* to beriberi than another is certain; but the reason for this is, not that any particular race is more susceptible than another, but that it may be more *exposed*. Europeans do not acquire beriberi in Japan, because they do not live as the Japanese do, and where the Japanese live. The same remark applies to Europeans, as a rule, all over the East. But when, as in war time, the European approaches in habits his darker-skinned comrade, he is attacked in the same way and, apparently, as readily. In Brazil the disease attacked black, white, and half-caste alike. I have seen beriberi three times in Europeans in China.

Occupation.—In Japan, students and others leading a sedentary life are said to be more liable to beriberi than labourers, farmers, and others leading an active out-door life. This is probably because the former never or rarely leave their ill-ventilated, damp, infected rooms, where they inhale the poison by day as well as by night; whereas the latter pass only a small part of their time in-doors. The liability of sailors is well known; and this appears to be owing to the concentration of poison in the damp, hot forecastle.[1] Coolies working in the mines in the Malay peninsula are said to be more liable than the field workers near the same places;[2] probably because the former sleep in dirty, crowded, damp, germ-tainted quarters.

Defective diet is often mentioned, not only as a predisposing cause of beriberi,—which, possibly, it is,—but as the actual cause. At one time the ravages of this disease in the Japanese navy were very great. On the theory that there was a deficiency of nitrogen in the food of the sailors an improved dietary was issued. Straightway the mortality and invaliding from beriberi began to decrease, and at the present day are quite insignificant.

For this great boon Japan is indebted to Takaki, and to the enterprise and intelligence the Government displayed in carrying out this gentleman's suggestions. This experience is often quoted as a proof that beriberi is caused by nitrogen starvation. But it must be remembered that when attention is turned to sanitary

[1] When a ship carrying a native crew gets into bad weather or enters cold latitudes, the imperfectly clad, underfed, and shivering lascars huddle together for warmth in the forecastle, every crevice and port-hole of which they contrive to stop up most effectually. Often they light a fire, before which they dry their wet rags. In this way is created, even in the pure and cool breezes of the high seas, an artificial incubator in which abundant moisture, a high temperature, and much organic matter supply all the conditions required by the experimentalist in his laboratory for the propagation of germs. This is a principal reason for the occurrence of beriberi among lascar crews in cold latitudes.

[2] Scott, *loc. cit.*

matters, reforms are instituted in many directions. Doubtless when the Japanese improved the feeding of their sailors they also improved the ventilation of their ships, kept them drier and cleaner, and instituted other changes of a like nature. That a defective diet does not cause beriberi is certain; for if this were the case the disease would be more general than it is, and would be the constant companion of famine, and the chronic state of starvation in which so many Eastern peoples live. It would not be confined to a town or part of a town, to a jail or part of a jail, where, as a rule, the feeding is much superior to what can be procured by the majority of the surrounding non-criminal population. It would not attack the well fed and the rich, and would not be communicable.

TABLE SHOWING THE NUMBER OF MEN, THE NUMBER AND RATIO OF CASES OF BERIBERI, THE DEATHS AND INVALIDING FROM THE SAME IN THE JAPANESE NAVY FOR EACH YEAR FROM 1878–91.

Year.	Force.	Cases of Beriberi.	Ratio of Cases per 1000 of Force.	Deaths from Beriberi.	Invalided with Beriberi.
1878, . .	4,528	1,485	327·96	32	19
1879, . .	5,081	1,978	389·29	57	8
1880, . .	4,956	1,725	348·06	27	9
1881, . .	4,641	1,163	250·59	30	16
1882, . .	4,769	1,929	404·49	51	17
1883, . .	5,349	1,236	231·20	49	4
1884,[1] . .	5,638	718	127·35	8	1
1885, . .	6,918	41	5·93	0	1
1886, . .	8,475	3	0·35	0	0
1887, . .	9,106	0	0·0	0	0
1888, . .	9,184	0	0·0	0	0
1889, . .	8,954	3	0·34	1	0
1890, . .	9,112	4	0·44	0	0
1891, . .	12,223	1	0·10	0	0

[1] Improved dietary in force from February 2, 1884.

Another theory has been advanced to the effect that rice eating, or diseased-rice eating, is at the bottom of the trouble. But rice is eaten all over the East; no other grain is so generally consumed. No epidemic has been pointed out as corresponding in its distribution with the distribution of particular parcels of rice, as would surely have been discovered if diseased rice were the cause.

Scorbutus, too, and the causes of scorbutus, have also been spoken of as causes of beriberi; but there are no sufficient grounds for such an imputation. Like all the other circumstances just mentioned, scorbutus may have some influence as a predisposing agent, but it certainly cannot be set down as the cause.

State of nutrition.—It is a singular fact, and one contrary to what obtains in many other diseases, that the robust and well nourished seem to be more liable to beriberi than the feeble and half-starved. This fact is insisted on by the European physicians who have studied the disease in Japan and elsewhere. Da Silva Lima says the rich were attacked in Brazil more often sometimes than the poor. Rowell says that the bodies of beriberics, when brought to the dissecting table, were always found to be fat and well nourished. Others, again, say that mental and physical depression strongly predispose to beriberi; malarial fevers and dysentery likewise are said to be potent predisposing causes. My own impression is that beriberi attacks any one, irrespective of his physical condition—just as measles and smallpox do. There is a personal susceptibility to beriberi, just as there is to alcohol; but it is not developed by hygienic conditions, although it may be slightly influenced by them. Some men may swallow large doses of alcohol with impunity; in others, again, a much smaller dose will induce tremor, delirium, or a neuritis very like the beriberi neuritis; just so with the poison of that disease.

Idiosyncrasy, therefore, may be put down among predisposing causes.

Previous attacks are well known to render an individual liable to a recurrence after a very short re-exposure to the influences of a beriberi centre. Many suffer annually if they do not quit the endemic area, and relapse at once on entering it again.

Acclimatisation.—It would seem that tolerance of the poison may be established by long exposure, possibly by exposure from childhood. In this may lie the explanation of the inferior susceptibility of townsmen permanently domiciled in a beriberi quarter as compared with countrymen perhaps entering such a quarter for the first time, as has been so frequently remarked by observers.

5*th. The incubation period.*

It is very generally stated by the older writers that a residence in a beriberi district of several months, even so long as two years, is necessary before beriberi declare itself. Modern testimony is to the effect that the disease may show itself within five weeks of the time of entering a tainted district. In proof of this, I may refer to Pekelharing and Winkler's account of the disease as it attacked a troop of soldiers coming from the uninfected island of Madoura to Atcheen. They numbered 340. Five weeks after their arrival in Atcheen 12 were attacked with beriberi, and 1 died; during the

next week 59 more were attacked, of whom 14 died; the remainder were sent home.

The time elapsing from first exposure to the poison of the disease to its full development will depend on many things; most important of which are the degree of concentration of the poison, the continuousness with which it is absorbed, and the individual idiosyncrasy. Just as with alcohol chronic intoxication precedes for a variable period the manifestations of acute alcoholism, so with beriberi a slow poisoning precedes acute manifestations of the disease. I think the comparison is fair; it is borne out by the fact, already alluded to, that for a long time prior to declared beriberi there are evidences of nerve degeneration, as shown by the electrical reactions of the muscles, trifling anæsthesia, and pretibial puffiness. In the case of ship beriberi, if we accept the view that the disease germ is taken in by the patient at the last beriberi port of call, then the period of incubation may be variously estimated at weeks or months, for new cases may keep cropping up at intervals all through a long cruise; if, on the contrary, we adopt the theory of place or ship infection, ship epidemics show that individual idiosyncrasy has much to do with the length of the so-called incubation period.

6th. The circumstances determining the explosion of the disease.

Systematic writers are in the habit of enumerating a number of things as provocative of an attack of beriberi. They mention acute diseases such as malarial fevers, dysenteries, diarrhœas, excessive fatigue, sudden falls of temperature, sleeping in the moonlight, and so forth. I do not know on what ground they base their assertions; they are very probably correct, but well-authenticated evidence is desirable on all such points.[1] Simmons states, that "with a sudden fall of temperature after a few hot days, accompanied by rain, he has often been able, when having a number of beriberi cases under treatment, to predict an exaggeration of all their symptoms, and foretell an increase in the number of cases in the out-door service." Very likely any circumstance leading to physiological strain may act as an exciting cause of an attack, but for the most part beriberi declares itself without anything very unusual of this description preceding its manifestation.

[1] I have lately seen two well-marked examples of peripheral neuritis associated with ordinary phthisis in East Indians, who had acquired their diseases on board a class of ships in which beriberi is not an unfrequent occurrence. Paresis, muscular hyperæsthesia, and atrophy, and, at one time, pretibial œdema, were all, and in both cases, well marked; so that it is fairly safe to conclude that the neuro-muscular element in the cases was of beriberi origin. Query: Does phthisis render the subject of it specially sensitive to the beriberi poison?

7th. The question of infection.

The question of direct infection is one which, in the present state of our ignorance, it is impossible to settle definitely. Can the poison of beriberi pass from one individual directly to another and communicate the disease? There can be no reasonable doubt about the communicability of beriberi, that is, that it may be carried from one place to another by means of human intercourse, and spread when so carried; but whether this spreading depends on soil or place inoculation, or on personal infection, or on both, it is hard to say. My own belief is that it spreads by soil or place inoculation, rarely, if ever, by personal infection. Unintentional experiments have not unfrequently been made on a large scale which seem to support this belief. For example:—Rowell states that prisoners affected with beriberi were sent from Singapore to Penang jail which, at the time these prisoners arrived, was free from beriberi. Two months after the arrival of the Singapore prisoners beriberi broke out; 61 prisoners were attacked, and 2 died. This looks like the introduction of the disease into the Penang jail by the Singapore prisoners. Assuming that this was the case, the epidemic may be attributed either to personal infection or to soil inoculation. At the same time as these Singapore prisoners were sent to Penang, other prisoners, also affected with beriberi, were sent to the Malacca jail; but no beriberi ensued there. If personal infection could convey the disease to the Penang prisoners, it is strange that it did not do so to the Malacca prisoners. The explanation of the difference in the results of the introduction of bereberi in the two jails appears to me to be, that the soil and surroundings in the Penang jail were suitable for the propagation of the beriberi germ, whereas the soil and surroundings of the Malacca jail were not. The often quoted case, mentioned by Dos Santos,[1] of the French priests in Brazil, whose footsteps through the country were dogged by beriberi, is explainable in the same way.

In a recent number of the *Archives de médicine navale* (December 1891) there is a short account of the communication of beriberi by Tonkinese and Annamite immigrants to the inhabitants of New Caledonia. Eight hundred labourers from Tonkin and Annam were landed on the quarantine island, Freycinet. In a short time beriberi broke out among them, causing 28 deaths. The island was abandoned, and 400 of the immigrants were cantoned at Koutio Koueta, 15 kilomètres from Noumea. Beriberi continuing, 40 of

[1] In the *Gazetta Medica da Bahia*, 1883, Nos. 11 and 12, this writer has published a useful and very complete list of Brazilian writings on beriberi.

the 400 died, and the disease spread to places in the neighbourhood, causing 10 deaths among labourers who had been resident there before the arrival of the infected immigrants; and men from the Solomon Islands and the New Hebrides, as well as New Caledonians, were also attacked.

These, and similar observations might be quoted as evidence of the infectious nature of beriberi; but they are equally applicable as supporting the hypothesis of transmission and spread through inoculation of the soil. And the hypothesis of soil inoculation is more in consonance with the well ascertained fact, that beriberi does not spread in hospitals among the other patients when beriberi cases are admitted, and that it does not attack the attendants. I have never heard of a medical man being attacked. Surely it would be otherwise were beriberi infectious in the same sense as are scarlet fever, measles, and others of the exanthemata.

The habits of beriberi in this respect resemble those of cholera, typhoid fever, and malaria; and seem to be explicable only on the theory that the poison is capable of multiplying outside the body, and is not communicated directly from one person to another.

Prognosis.—It is an exceedingly risky thing to venture on a prognosis in a case of beriberi. Extreme degrees of paralysis, atrophy, and œdema need not end in death; on the other hand, death may supervene in what are apparently the least threatening cases. In all cases death is, so to speak, rather a matter of accident than of necessity; it depends partly, of course, on the intensity of the poisoning, but more on the particular set of nerves picked out by the poison. I have seen a case in which every voluntary muscle of the body except those of the face and of mastication was completely paralysed, and many of them atrophied to an extreme degree, so that the patient could not move even a finger, not to mention a limb, or to turn in his bed; and yet he gradually recovered. The force of the disease in this case fell on the voluntary muscles, and spared those of respiration and the heart. On the other hand, the force of the disease may fall on the heart and respiratory muscles, and but lightly on the voluntary muscles; in which case death will certainly ensue, and rapidly, even although to all appearance, merely judging by the state of the muscles of legs and arms, and the œdema, the attack is a light one. Hence the danger of venturing on a prognosis in beriberi.

Certain circumstances, however, are found to be prejudicial to recovery in beriberi; pregnancy is one of these, and so, I believe, are surgical operations and traumatism of all sorts. Vomiting—

not depending on indigestion—is always of grave import, seeing that, as already explained, it probably indicates implication of, or extension of disease in the vagus; it is of all minor symptoms the most ominous. Suppression of urine and œdema of the lungs are nearly as alarming in their import. Rapid extension of precordial dulness to the right, accompanied by precordial pain, dyspnœa, cyanosis, and fall of body temperature, is nearly a sure sign that the heart is failing, and that death is impending. In the matter of death and recovery beriberi is a disease of surprises.

Mortality.—The mortality is very difficult to estimate. If we include the minor forms of beriberic intoxication,—what we might designate beriberia ambulans,—such as slight degrees of leg paresis, pretibial anæsthesia and œdema, in our estimate, then the mortality is proportionately smaller than if we estimate it only on such cases as lay up or exhibit serious signs of heart and other thoracic complications.

Roughly speaking, wet beriberi is more deadly than dry beriberi; the mixed form seems to occupy an intermediate place as regards fatality. Another general statement might be made to the effect, that the more numerous the cases in a community, the more deadly are the individual cases.

Taking only cases of what might be called declared beriberi, that is, cases bad enough to be brought under the notice of the medical man, according to statistics the death-rate ranges between 1 in 40 attacked,[1] 1 in 20,[2] and 1 in 2.[3]

Corre, after examining a large number of statistics, arrives at an average mortality of 34·6 per cent., or about 1 in 3; but this is much too high an estimate if the milder cases be included.

The risk to life varies very much in different epidemics, and also according to the circumstances under which the patients are treated. If patients remain in the place in which the disease was contracted, in the same house, school, jail, or ship, with others constantly sickening around them, and with every evidence that the poison is continuing to be evolved by soil or surroundings, then the risk to life is very great indeed. But should the patient be removed at once from the endemic centre, and before the cardiac and respiratory nervous systems are very seriously implicated, the risk to life is very much lessened. It is with beriberi as with any other form of chronic poisoning in this respect.

Sequelæ.—These have not been studied with the care they

[1] Japanese Navy, 1878–84; of 1426 on an average attacked annually, 35¼ died. Takaki Sei-i-Kwai, April 1885.

[2] Naval Hospital, Tokio, 1878.

[3] Bahia, 1867.

deserve. Doubtless many deformities must result from permanent muscular atrophy. Usually the muscles become, after many months, completely rehabilitated and the cardiac dilatation is reduced or compensated; but this does not happen always. I have seen and operated on a typical case of talipes equinus (Fig. 45), the result of a long antecedent attack of beriberi. The frequency of cardiac murmurs among the Japanese has often been remarked, and is generally, probably correctly, attributed to former attacks of beriberi.

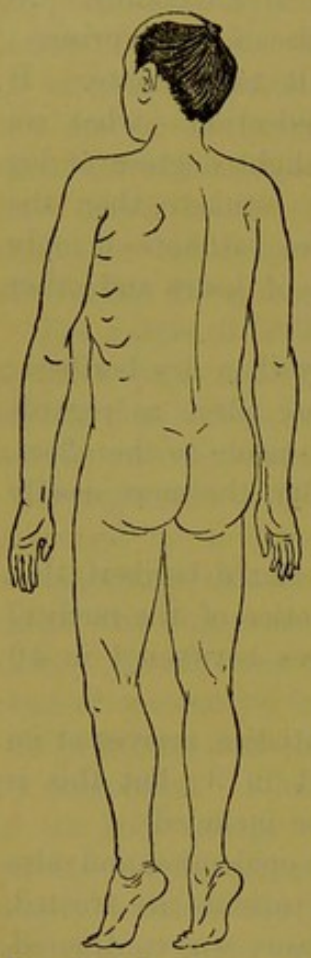

FIG. 45. — Position sometimes assumed in walking after partial recovery from beriberi atrophia—due to shortening of calf muscles. (Simmons.)

The sequelæ of this disease constitute a virgin field for the investigator.

Diagnosis.—The two requisites for the successful diagnosis of beriberi are, first, a knowledge of the symptoms of the disease; second, to suspect its presence.

In tropical and subtropical countries in which beriberi is known to occur, and in ships carrying coloured crews, whenever complaint is made of weakness of the legs, numbness of the shins, and puffiness over the tibiæ, the idea of beriberi ought always to present itself to the mind of the physician, and the patient ought to be examined in detail for other symptoms of peripheral neuritis. The companions of the patient, whether in house, school, ship, prison, barrack, or camp, ought also to be examined in the same way; and it is more than probable that it will be found that the individual who first complained is not the only one affected with latent beriberi. I have seen this on board ship. Considering the importance of early removal of the patient from the endemic centre, this general investigation of the pathological condition of the different members of a threatened community ought never to be neglected. If several cases be found presenting symptoms resembling those of the patient first brought under notice, there can be little doubt that the first case, as well as the others, is beriberi. In the fully developed disease there is no difficulty, as a rule, in diagnosis; but the earlier phases are difficult to pronounce on, unless it be found that several members of a community are similarly affected.

Carefully conducted electrical examination of the anterior tibial

muscles and tactile areas, as Pekelharing and Winkler have pointed out, would be of service in establishing a diagnosis; but as a galvanometer and other apparatus is necessary for this, and as these are not usually available, electrical examination is generally impracticable.

A difficulty sometimes arises in a beriberi district, or in patients from such a district, as to the nature of certain cases of peripheral neuritis. The question may come to be, are they malarial, alcoholic, or beriberic? The three poisons are frequently in operation simultaneously in the same individual, so that it is difficult, or impossible, to assess what weight we should apportion to each as a disease factor. Speaking generally, I would say that the presence of much œdema, and distinct implication of heart and respiration point to beriberi; severe general neuralgic pains and tremor to alcoholism; localised intermitting neuralgiæ with intermitting rises of temperature to malaria. Very little, however, is known about malarial neuritis; many of the cases of paraplegia, or leg paresis, attributed to this influence are, I feel convinced, attributable rather to beriberi or to alcohol.

Beriberi is readily distinguishable from locomotor ataxia by the difference of the electrical reactions, and by the absence in the former of true ataxia. In beriberi there is loss of power in the muscles, but what power remains can be correctly directed; in locomotor ataxia, at first, there is little or no loss of power, only loss of the faculty of directing it. Then there are many other points of distinction, such as the lightning pains, the ocular symptoms, the bladder symptoms, the trophic disorders, and the slow progress of locomotor ataxia.

It is possible that the paralytic symptoms of beriberi may be closely simulated by different affections of the cord, such as acute or chronic anterior poliomyelitis, diffuse myelitis, ascending paralysis, disseminated sclerosis, and so forth; but all of these diseases present their special symptoms, and the phenomena of central disease are never met with as epidemics, and are not usually associated with œdema. Pernicious anæmia is at once distinguishable by a microscopic examination of the blood, and by its non-association with paralytic symptoms and œdema. Scorbutus is distinguished by the hæmorrhages, the ulcerations and swelling of the gums, and the swelling of the spleen.

Horton[1] (quoted by Fayrer[2]) has unfortunately described the Sleeping Sickness of the Congo as a form of beriberi. A slight acquaintance with the symptoms of these two diseases will satisfy any one of their specific differences. Sleeping Sickness is a disease

[1] *Diseases of Tropical Climates*, 1874. [2] Quain's *Dictionary of Medicine*.

of the central nervous system; beriberi, of the peripheral. Sleeping sickness is slowly developed, the leading symptom for some being a gradually increasing torpor and somnolence; beriberi is more rapidly developed, the leading symptoms being paresis and œdema. There is no paresis in Sleeping Sickness, no marked œdema, no implication of heart and respiration, no liability to sudden death, no inability, only indisposition to move. Towards the end of Sleeping Sickness tremors, spastic affections of the muscles, and bedsores are general features; there is nothing of this kind in beriberi. Recovery from beriberi is the rule; Sleeping Sickness is always fatal. There are many other points of distinction, which are pointed out in the article on "Sleeping Sickness."

Prophylaxis.—The prophylaxis against beriberi is similar to the prophylaxis against malaria; that is, damp localities ought to be drained, and, if possible, avoided as sites for building or camping on; ventilation must be attended to; and overcrowding and the hot, steamy atmosphere it induces carefully shunned. Proper clothing, and a dietary sufficiently varied and nutritious must be provided.

Very great importance is attached to the dietary by those with most experience in the medical care of large bodies of men in beriberi districts. The endeavour has always been to get rid of rice as much as possible, and to substitute such cereals and other substances as contain a larger proportion of nitrogenous matter, due regard being had to economy of administration. The following is the dietary, according to Rowell, of the prisoners in the Singapore jail before and during the early part of the great epidemic there; and, in contrast with this, the improved dietary subsequently adopted, and on which, or a slight modification of which, prisoners in Singapore enjoy a practical immunity from the disease:—

Old Diet.

Articles.		
Rice,	24 ounces.	
Vegetables,	6 ,,	
Salt,	½ ,,	daily to all.
Salt fish,	2½ ,,	three times weekly to all.
Fresh fish,	4 ,,	three times weekly to Chinese, and four times to Malays.
Pork,	4 ,,	once a week to Chinese.

Improved Diet.

Rice,	14 ounces.	Daily to all.
Wheat flour,	4½ ,,	
Meat,	6 ,,	
Fish,	4 ,,	
Vegetables,	7 ,,	
Beans,	5 ,,	
Dhall,	2 ,,	

The following is the daily allowance of food in the Japanese Navy for one healthy person, as fixed on February 2, 1884, and after the introduction of which remarkable improvement in the health of the crews, as shown in the table at p. 490, commenced :—

(A *momme* is equal to 58 grains Troy. The numbers refer to *mommes*.)

Articles.	Weight in *mommes*.	
Rice,	180·0	Bread, 160 momme ; Biscuit, 130 momme.
Meat,	80·0	When eggs are substituted each egg to be reckoned as equivalent to 10 momme.
Fish,	40·0	When there is no fish 20 momme of meat may be substituted.
Miso, Sauces,	14·0	
Shoyu, Sauces,	16·0	
Vegetables,	120·0	
Beans,	12·0	
Wheat flour,	20·0	
Tea,	2·0	
Fat, or oil,	4·0	
Sugar,	20·0	
Milk,	12·0	If condensed, 1½ momme to be given.
Vinegar,	2·0	
Spices,	0·3	
Alcoholic liquors,	24·0	This is in the case of Japanese liquors. When other liquors are used, the quantity shall be fixed in proportion to the amount of alcohol contained.
Salt,	2·0	
Pickles,	20·0	
Fruit,	To be given with care.	

Sleeping on the ground, or even on the ground floor, ought, if possible, to be interdicted. Buildings in which several cases of beriberi have arisen ought to be closed for a time, and not reopened until the epidemic has passed away ; and before reoccupation they ought to be thoroughly disinfected, whitewashed, and ventilated. From time to time in schools, jails, and similar aggregations of humanity in beriberi centres, during an epidemic the individual inmates ought to be periodically examined for symptoms of incipient beriberi, and those who are found to be in the slightest degree affected at once sent away to a more salubrious locality.

In shipping native crews, the examining surgeon ought to be alive to the possibility of introducing beriberi into his ship ; he therefore ought never to pass a man with tender calf muscles, pre-tibial œdema and anæsthesia, and impaired knee-jerks.

In ships the periodical cleansing and disinfection of holds, and, above all, forecastles, ought to be attended to. I believe, if the crew's quarters were fumigated with sulphur or mercury in the same thorough way in which ships' holds are fumigated to kill rats

and cockroaches, and the crew forced to submit their clothes, chests, and other belongings to the same process, and be compelled to keep portholes and ventilators open, great benefit would ensue, and many deaths and much invaliding and inefficiency avoided. At the same time, wholesome and physiologically sufficient rations should be issued, and measures taken to insure that they are consumed. Suitable clothing must, if necessary, be provided for natives on entering cold weather.

Pekelharing and Winkler recommended the disinfection of buildings and the neighbouring soil with one per mil. solution of corrosive sublimate. To be of any use the process must be thoroughly carried out, and repeated at short intervals. This system of disinfection was extensively practised in Atcheen, and, apparently, with marked benefit.

Treatment.—The first, most important, and never to be neglected measure in the treatment of beriberi, is removal of the patient from the place in which he contracted the disease. If possible, he ought to be transferred to a high and dry locality where beriberi is not endemic. The necessity for this step I trust I have made sufficiently apparent. If removal is impossible, an endeavour ought to be made to place the patient in a well-ventilated room in an upper storey; at all events he must sleep well off the ground; and the ventilation ought to be such that there is no danger of stagnation of the air in his room. If he is well enough to go out, he ought to be encouraged to get into the fresh air as much as possible. Sailors attacked at sea ought to be removed from the forecastle and placed in some well-ventilated cabin or on deck; in any case they ought never—neither healthy nor sick—to sleep in the forecastle when beriberi has broken out in it. It is almost hopeless to treat beriberi, during a bad epidemic, on the spot where the disease was acquired, and where the atmosphere is still being poisoned; quite as hopeless as to treat a case of alcoholic neuritis while the patient is continuing to drink freely.

Food ought to be nutritious, varied, and digestible, and not of too bulky a character. Experience has shown that in native dietaries the substitution of wheat, barley, or beans, and a proportion of animal food for rice, has a beneficial effect in beriberi.

While there is evidence of heart implication, walking or active exertion of any kind ought to be avoided, as also everything else which may tend to throw unnecessary work on the labouring organ.

Many drugs have been tried in beriberi; but there is no definite evidence that any medicinal treatment, aspiring to be of a specific character, has been of the slightest service. It is customary to

mention certain Indian remedies as useful in beriberi; *treak ferook* and *oleum nigrum* were formerly in high esteem, but their use seems to be now quite abandoned.

In view of the tendency to accumulation of the blood in the right heart, the tendency to œdema, to constipation, and to cardiac asthenia, I have been in the habit of employing systematically small doses—sufficient to procure at least two stools a day—of some saline aperient—none better than Epsom salts—with or without small doses of digitalis. To this, after the case seems to have reached its acme, and muscular hyperæsthesia is subsiding, I add small doses of some preparation of iron, and, later, strychnia. When the threatening symptoms have declined the aperient is omitted. With the view of encouraging rehabilitation of the atrophied muscles, faradisation and massage ought to be practised as soon as the hyperæsthesia has sufficiently subsided. Arsenic and nitrate of silver are well spoken of as tonics at this stage.

Scott recommends the systematic administration of belladonna, on the supposition that the poison of beriberi is similar in its physiological effects to muscarin, the action of which is antagonised by atropine. The same writer recommends the systematic use of alkalis to counteract the acid reaction of the blood present in beriberi.

For the relief of hyperæsthesia and cramps, Anderson and others recommend tincture of aconite in 15 minim doses, carefully increased; they report favourably of its effects.

When symptoms of grave cardiac embarrassment—*shiyôshin*, as the Japanese call it—set in, the treatment which has been found of most service is a rapidly depletory one, directed to relieving the distension of the right side of the heart, either by diminishing the bulk of the blood, or by diverting it from the heart to the distal vessels. No time should be lost. Immediately threatening symptoms appear a brisk drastic cathartic should be administered—one or two drops of croton oil with 5 grains of calomel, or a quarter grain of elaterium with 5 grains of compound gamboge pill, are what Anderson recommends. Pending the action of these, and as a means of temporary relief, Simon[1] strongly recommends the administration of nitro-glycerine in full and repeated doses—5 to 10 minims of a 1 per cent. solution repeated in diminished dose every fifteen or thirty minutes, according to the indications supplied by the pulse, and until the paroxysm subsides. Bentley advocates the same practice. Von Tunzelman, in cases of cardiac paresis, recommends hypodermic injections of digitalis, along with external

[1] *Lancet*, March 4, 1893.

heat. If these measures fail to give relief quickly, and cyanosis and dilatation of the right side of the heart seem to be increasing, the only hope of saving the patient is to bleed him. If blood will not flow from the arm the external jugular vein should be at once opened, and sufficient blood taken to relieve the overburdened, dilated heart. A few ounces generally suffice. As soon as the patient breathes easily and expresses himself relieved, the flow of blood should be at once stopped, as beriberi patients do not stand bleeding well, and the venesection may have to be repeated within a very few hours should heart failure again threaten. Along with venesection or purgation, limitation of fluids ought to be enforced. Should it prove impossible to get blood from arm or neck, in these circumstances both Anderson and Eldridge recommend aspiration of blood from the right ventricle through a fine needle; this desperate measure is justifiable in the desperate circumstances.

Excessive accumulation of fluid in the pericardium or pleuræ indicates aspiration.

CHAPTER XIII.

NEGRO LETHARGY, OR THE SLEEPING SICKNESS OF THE CONGO.

BY PATRICK MANSON, M.D., M.R.C.P., LL.D.

THIS curious, very deadly, and, as regards the inhabitants of the countries in which it occurs, very important disease was first brought to the notice of the profession by Winterbottom, a physician who wrote about the natives of Sierra Leone at the beginning of the century.[1] Although since Winterbottom's short and very imperfect notice appeared a good deal has been written on the subject, and some new and important facts have been gleaned, we are still without assurance as to the cause and precise nature of this singular malady.

Geographical Distribution.—As an endemic disease, sleeping sickness is confined absolutely to the west of Africa. Its limits north, south, and east have not been accurately ascertained. Roughly speaking, Senegal and the Congo region may be set down as its northern and southern limits respectively; but how far it extends into the "hinterland" is not known. Of late years it has been very prevalent on the Lower Congo and in the neighbouring Portuguese territory to the south,—Banza Manteka and San Salvador, for example. As both of these places are quite 120 miles inland, this disease cannot be regarded as one which is confined to the coast merely.

There are some important facts with regard to the details of its distribution which require mention. 1st. It is not evenly distributed over all the vast region embraced in the limits indicated, but prevails, or rather originates in particular districts or villages only, neighbouring districts being free from it. Thus, while it decimates at times the villages in the cataract region of the Lower Congo, it is unknown at Stanley Pool a little further inland, and at Banana

[1] *An Account of Native Africans in the Neighbourhood of Sierra Leone*, etc., 1803.

Point on the neighbouring coast; and it is said to be unknown on the Upper Congo. 2nd. Although it originates in these limited districts, and is contracted in them, it may not show itself in the infected individual until many months, or even years, after he has quitted the endemic area. Thus it frequently happens that sleeping sickness develops in an individual when he is in a district in which the disease is not endemic, and in which, therefore, the germ (if I may use the expression) had not been acquired. The negroes are well aware of this fact; if one of them has visited a sleeping sickness district he does not consider himself safe from the disease until seven years have elapsed. This is a well-founded belief, and not a groundless negro fancy. The records of the slave trade are quite conclusive on the point, and their testimony is borne out by modern experience. Thus formerly, when negro slaves were imported into America, sleeping sickness not only frequently claimed its victims during the voyage across the Atlantic, but also after the slaves were landed; and even for several years subsequently cases would continue to crop up among them on the plantations from time to time. I am cognisant of an instance in point which occurred not very long ago in England. A Congo lad who had been in this country for three years, and apparently all this time in the enjoyment of perfect health, began to show the characteristic symptoms of the disease, and after a few months died of it. 3rd. The negro who has never been to Africa never becomes affected with sleeping sickness. 4th. It seems that the disease, after decimating or almost depopulating a village, may gradually die out, at the same time moving on to a neighbouring village or district. This last statement I make on the authority of Dr. Grattan Guinness, who lately visited one of the endemic districts on the Congo. Formerly, he tells me, the village of Banza Manteka was decimated by the disease; now, few cases are met with there. The wave of disease, he says, seems to have passed on coastwards, where villages which formerly enjoyed an immunity from sleeping sickness are now as much afflicted as Banza Manteka was a few years ago.

Age, Sex, and Occupation seem to have no bearing on the disease whatever. Whole families are struck down with it at a time; even young children and old people enjoy no immunity, and men and women are equally susceptible.

The Negro Race, it has been asserted, is alone attacked. This is much too sweeping a statement. Corre[1] states that he had heard of a European having died of the disease, and that he saw a Moorish woman whom he believed to be suffering from sleeping

[1] *Gaz. méd. de Paris*, 1876; *Arch. de méd. nav.* No. 27, 1877.

sickness at an early stage; and Chassaniol[1] refers to its occurrence in a mulatto. Negroes are certainly more liable to sleeping sickness than other races; but this is probably entirely owing to the fact that they are more exposed to its cause—whatever this may be—than others are, and not that they are the victims of some peculiar racial proclivity.

Symptoms.—The onset of sleeping sickness is very gradual in most cases. There are certain prodromata which, to the experienced, are significant of the impending disease. These are a peculiar expression of countenance, a fulness or puffiness about the face, a languid droop of the upper eyelids, perhaps occasional attacks of headache or vertigo, perhaps attacks of fever or diarrhœa. After these symptoms have shown themselves, and for long after it has become possible for the initiated to pronounce a diagnosis, the patient is still able for his work; it is observed, however, that he is apt to fall asleep at unusual times, even in the midst of his work or amusements. It is noticed that he has become very languid, that he is easily fatigued, and that he shirks his work. He complains of weakness, and likes to lie about in the broiling sun, as if he felt cold and enjoyed the warmth. By degrees these symptoms increase in intensity, and he no longer attempts to work. He begins to seclude himself, and after a time ceases to seek conversation, although when roused and his attention is loudly demanded he will still answer questions correctly enough, usually, however, only in monosyllables. He becomes taciturn, morose, and sad looking. His eyes are closed or half closed, and he sleeps, or appears to sleep, almost constantly. When he attempts to walk he is often seen to stagger like a man who is drunk or but half awake. At times he has irregular attacks of fever, and then his temperature is elevated and his pulse quickened; at other times his temperature seems to be subnormal and his pulse very slow. It is now observed that he has a good deal of muscular tremor, and that there is very marked muscular weakness. These symptoms increasing, he finally takes to bed or lies crouching in a corner of his hut or court, indifferent to everything going on around him, rousing up a little only when loudly and directly addressed, or when food is brought to him. This he does not ask for any longer; so great does the torpor become that it is difficult to rouse him even to eat, and it sometimes happens that he will fall asleep while in the act of conveying food to his mouth, or even whilst masticating. His lips may become swollen, and a sticky saliva dribble from his mouth. Meanwhile, all the functions of organic life are properly performed;

[1] *Arch. de méd. nav.* No. 3, 1865.

the food he can be got to swallow is well digested and assimilated, and nutrition does not suffer, and fæces and urine are regularly passed, though probably under him. A change, however, sooner or later sets in. He at length begins to waste; tremor of hands, arms, and tongue become more marked; there may be choreic movements of one limb; or convulsive seizures of a clonic, sometimes of a tonic character, followed by temporary paralysis, may attack a limb. Bedsores now form, and, strength gradually failing, he may fall into a comatose condition from which he can hardly be roused. Finally he dies either by slow asthenia, or suddenly in convulsions.

There is considerable variation in the symptoms and the rate with which this singular disease progresses. Sometimes it seems to be arrested, and the patient appears to be recovering; but sooner or later torpor recurs and the disease resumes its fatal course. In not a few instances a maniacal condition precedes or interrupts the usual evolution of symptoms, as if the intellectual centres in the cerebrum became involved in the pathological process already beginning to implicate the motor and trophic centres and tracts. In other cases some intercurrent inflammatory affection proves fatal before the torpor becomes complete.

In only one or two instances have we records of a thorough examination of the nervous symptoms by modern neurological methods. In two instances only has an ophthalmoscopic examination been recorded. Mackenzie,[1] in a case under his care in the London Hospital, found the fundus normal; and Corre states that Senés, in a similar case, also failed to find anything abnormal either in fundus or media. The conjunctivæ may be injected, the pupil normal or perhaps dilated and insensible to light, and there may be strabismus; but these are certainly not essential or constant symptoms. Sensation is not usually impaired, although in some cases there are patches of anæsthesia about the body or limbs. The muscular force is very much diminished, and tremors are often so marked that the patient cannot carry his food to his mouth. Mackenzie made a careful examination of the reflexes in his case, and found that the superficial reflexes were normal and the knee jerks present, perhaps rather too active, but there was no clonus. Neither albumen nor sugar are present in the urine, or, if they are found, their presence may be regarded as accidental. The thoracic and abdominal viscera are healthy. Although memory is impaired, and the intellectual faculties, as a whole, dulled, if the attention can be aroused the comprehension is found to be not very defective.

[1] *Clinical Soc. Trans.* vol. xxiv. 1890.

Torpor is a constant symptom, but there may be no excess in the amount of actual sleep indulged in, although it is apt to be taken at unusual times. Mackenzie had his patient carefully watched for a short time to determine this point; it was found that not more than eight hours in the twenty-four, in the aggregate, were passed in sleep.

Corre calls attention to the remarkable frequency with which a peculiar papulo-vesicular eruption is present in cases of sleeping sickness. This eruption is found over all the body, but is most marked over the chest; it is attended with excessive pruritus. The skin is harsh, dry, and furfuraceous when sleeping sickness is well developed. Sometimes the lymphatic glands of the neck are slightly enlarged; and it seems that the natives in certain districts attach some importance to this as a diagnostic symptom, and sometimes even excise the glands by way of treatment.

The Duration of the Disease is very variable. It may prove fatal in four or five months; in other instances it drags along for two or three years. A good deal must depend upon the care and attention bestowed on the patient. If left alone he would starve to death. Mackenzie's case from commencement to termination lasted about one year. He began to be ill early in 1890; he was then in Africa. In June he came to England, and he was then fairly well, moving about and doing a little work; the only thing wrong with him, apparently, being a tendency to fall asleep at unwonted times. In September tremor and muscular weakness were very marked. In October he was practically bedridden; and he died on the 3rd December.

Pathological Anatomy.—A considerable number of post-mortem examinations have been made in cases of sleeping sickness, but no characteristic lesion has been discovered. The brain and meninges are reported sometimes as being congested, sometimes as being anæmic; sometimes part or the whole of the brain is reported as indurated, sometimes it is said to be softened. In Mackenzie's case the post-mortem, which was most carefully and thoroughly made, yielded no evidence whatever in explanation of the symptoms during life; a *cysticercus cellulosæ* was found on the under surface of the left frontal cerebral lobe, but this was evidently in no way related to the symptoms. It is evident that the lesion of sleeping sickness is still to seek.

Cause.—Many speculations have been indulged in with regard to the cause of sleeping sickness. Thus it has been attributed to various articles of food and drink,—diseased grain, palm wine,

poisons, Indian hemp; to nervous influences, such as sunstroke, nostalgia, excessive venery; to such general causes of disease as scrofula, malaria, and beriberi. It is evident, however, that though one or other of these might possibly be operative in one group of cases, it will not apply to all. Such things as sunstroke, malaria, beriberi and so forth might be contracted in Africa; but exposure to such influences in Africa could not give rise to disease several years afterwards in England.

In seeking for the cause of sleeping sickness several important points must be borne in mind—1st, the cause must be sought for in circumstances which may antedate the development of the symptoms by several years; 2nd, it can only be acquired on the West Coast of Africa; 3rd, when acquired it may be carried away from the endemic area, and remain latent for several years; 4th, on being introduced into a new country it does not multiply and spread. Sleeping sickness bears some resemblance in the protracted nature of the incubation period to rabies, and it further resembles this disease in its being essentially one of the nervous system, and in its deadliness; but, unlike rabies, it is not directly communicable, and it is strictly endemic.

I have ventured to put forward a speculation[1] to the effect that possibly *filaria perstans* has something to do with sleeping sickness. This parasite is very common in the countries and districts in which sleeping sickness is endemic, and, as a matter of fact, has been found in the blood of several cases of the disease. Such an etiology would explain the singular persistency of liability to the disease which continues for years after the endemic area has been quitted. It would also very probably explain the frequent coincidence of the itching papulo-vesicular eruption described by Corre as being so common a feature in sleeping sickness. This eruption seems to be the same as O'Neil describes under the name "craw-craw" (see article in sequel), which is associated with a filaria-like organism in the papules; and there is some reason to think that this organism will turn out to be an advanced developmental form of *filaria perstans*. It would also explain the limitation of the endemic area of sleeping sickness; for, like this disease, as far as known at present, *filaria perstans* is limited to the west of Africa, where in certain districts it is to be found in nearly half or, in some villages, even in quite two-thirds of the population. Although its presence in the blood is not necessarily or by any means generally associated with disease, it does not follow from this that the parasite may not occasionally, and as the result of

[1] *Trans. Internat. Cong. of Hyg. and Demog.* 1891.

certain circumstances, be the cause of disease. In this respect it may resemble *filaria nocturna*, which becomes pathological only when misplaced or itself the subject of some abnormality or accident. In nine cases of sleeping sickness, samples of the blood of which were sent to me from Africa, and which I examined, *filaria perstans* was found in five, and it may have been present in the others, although I failed to find it in the very small quantity of blood submitted to examination.

As to the mechanism by which a parasite like *filaria perstans* could give rise to sleeping sickness, it is quite possible to imagine that if it happened to be located in the arteries leading to the brain, or in the veins or lymphatics coming from the brain, it might so interfere with nutrition as to give rise to the characteristic torpor, tremor, and convulsive seizures.

It is evident that there is room for much useful work in connection with the pathology and etiology of sleeping sickness, a disease which, as the African continent is opened up, is bound at no distant date to attract a good deal of attention.

Mortality.—The majority of those attacked by sleeping sickness die. It is even a question if any one ever recovers from the disease after it has thoroughly developed. In 148 cases, Guérin claims only one recovery. It may therefore be said to be almost as fatal as rabies.

Diagnosis.—Sleeping sickness has been described as a variety of beriberi; but a very limited acquaintance with the symptoms of the two diseases is sufficient to prove their non-identity, and to establish well-marked and specific differences. Sleeping sickness is a disease of the central nervous system, beriberi of the peripheral. Palpitations, well defined anæsthesia, hyperæsthesia of muscles, dyspnœa, œdema, and paresis are not present in the earlier stages or necessarily at any time in sleeping sickness; torpor and marked sleepiness, tremor, convulsive seizures, itching papulo-vesicular eruption, and liability to bedsores are not symptoms in beriberi. Knee jerks are usually absent in beriberi, present in sleeping sickness; the latter is a very slowly developed disease compared to beriberi, and it has a much more limited endemic area. There are many other points of difference, but those alluded to are sufficient to establish diagnosis.

Treatment.—Until more is known about the causation of sleeping sickness very little can be said on the subject of prophylaxis. Assuming that some such cause as *filaria perstans* is at the bottom of the matter, then careful attention to the water supply in the endemic regions is indicated, and would probably be rewarded by

immunity from the disease. When once established no treatment hitherto adopted has been found of the slightest avail. I have heard of cases in which quinine and arsenic did good. In one case, of what was believed to be incipient sleeping sickness, arsenic freely given removed entirely the threatening symptoms. Temporary improvement is said to follow free purging.

CHAPTER XIV.

FRAMBŒSIA OR YAWS.

WILLIAM THOMAS PROUT, M.B., C.M.Ed.

Definition.—A specific endemic disease, occurring in tropical countries, propagated by contagion, and characterised by the presence of an eruption, which commences as papules and eventually forms crust-covered tubercles. The disease runs a chronic course, tends to spontaneous cure, and is accompanied by more or less well-defined constitutional symptoms.

Synonyms.—The disease has now been described in different parts of the world under different names. The scientific name Frambœsia was given to it by Sauvages in 1759.[1] Yaws is the name by which it is generally known in the English colonies, and is probably of African origin. Pian in the French Antilles, Buba in Brazil, Coko or Dthoko in Fiji, Tonga in New Caledonia, Bouton d'Amboine in the Moluccas, Parangi in Ceylon, are the principal synonyms. On the Gold Coast, where I have seen the disease, the Fanti name is Dūbé or Dubea, the Accra name Ajortor, and the Hausa, Tongara.

Geographical distribution.—This has been already indicated to some extent in the preceding paragraph. The disease, which was at first supposed to be entirely confined to the negroes, is now found to affect many entirely different races. It occurs in the following places:—

Africa.—On the west coast from Senegambia to Angola.

On the east coast at Mozambique, the Coromo Islands, and Madagascar. At Mauritius I cannot recall having seen a typical case of yaws, but there was an affection of the feet which was known as crab-yaws.

In Northern Africa it is met with in Algiers and the Nile basin; in Central Africa, at Bornu, Timbuctoo, etc.

Asia.—It is found in India (Pondicherry), but is rare. It occurs in Ceylon; also in the Moluccas, Java, Sumatra, and Macassar.

[1] Sauvages, *Nosologia Methodica*, 1768.

Oceania.—It is widely diffused in Fiji, New Caledonia, Loyalty Islands, and Samoa.

America.—It is endemic in most of the West India Islands, San Domingo, Guadeloupe, Dominica, Jamaica, etc.; also in Brazil, Guiana, and Venezuela.

History.—The disease is mentioned by Oviedo,[1] but the first authentic description of it appears to be that of Pison in 1648,[2] though it is supposed that the disease described by the Arabian physician Ali Abbas as *Sahafati*, as far back as the tenth century, was really yaws and not syphilis. Following these, Bontius described the disease in the Moluccas in 1718;[3] Dazille in the French Antilles in 1742;[4] Winterbottom in 1762 in Sierra Leone;[5] Hillary in 1759 in Barbadoes;[6] De Rochas in 1860 in New Caledonia;[7] Macgregor in Fiji in 1879,[8] and Kynsey in Ceylon in 1881.[9] De Rochas[7] endeavours to trace the origin of this disease through the Foulahs of the Central Soudan to Arabia; but the universal opinion hitherto has been that West Africa was the home of this disease, and that its spread over the world was due to the exportation of negro slaves. It is possible that this may account for its introduction into the West Indies, but it must be remembered that it exists and rages in other parts of the world, such as Oceania, where no importation of negroes has taken place. In the West Indies, however, it would seem to have found very favourable conditions for its spread, for the disease appears to assume a severer form there than on the Gold Coast. During the period of slavery the disease was a recognised one on all sugar estates, and means were adopted for the isolation and treatment of affected persons. In Dominica, after the emancipation, the disease gradually increased, until in 1871 it assumed the dimensions of an epidemic, and segregation in special yaws hospitals was made compulsory. This appears to have been beneficial in checking its spread. In Jamaica a diminution of the disease took place after the emancipation, which Dr. Bowerbank[10] attributed to the cessation of the practice of inoculation which had been prevalent among negroes. Later on, however, in 1863 a recrudescence of the disease took place.

[1] Oviedo, *Hist. general y natural de las Indias*, lib. ii. caps. 13, 14.

[2] Pison, *De Medicina Brasiliensi*, lib. ii. cap. 19.

[3] Bontius, *Medicina Indorum*, cap. 19.

[4] Dazille, "Observations sur les Maladies des Nègres."

[5] Winterbottom, "An account of the Native Africans in the neighbourhood of Sierra Leone."

[6] Hillary, "Observations on the changes of the air and the concomitant epidemical diseases in the island of Barbadoes."

[7] De Rochas, "Essai sur la topographie hyg. et. méd. de la Nouvelle Calédonie."

[8] Macgregor, *Transactions of the Epidemiological Society*, 1880, p. 53.

[9] Kynsey, "Report on the Parangi disease of Ceylon."

[10] *Med. Times and Gaz.* April 3, 1880, p. 369.

In Barbadoes and Antigua the disease is said to have almost completely disappeared.

Clinical history.—The period of incubation may be taken to be from three weeks to six months. Paulet[1] states that in some cases where he inoculated healthy negroes with yaws fluid the incubation period was from twelve to twenty days. During this stage there may be no premonitory symptoms, but occasionally there is slight feverishness, pains in the limbs, gastric disturbances, palpitation, and sometimes a certain degree of anæmia. In the negro the skin loses its shiny black colour and becomes dull, sometimes scaly and lighter in tint. Dr. Macgregor points out that in Coko the incubation stage is sometimes very prolonged and severe, and that these cases are generally fatal.

According to Dr. Rat,[2] the stage of invasion begins at the seat of inoculation with what be describes as a primary sore, "as characteristic of the disease as the chancre is of syphilis," and points out that it is generally overlooked. This initial lesion consists of a papule which at the end of about seven days develops a pale yellow fluid at its apex. After about seven days more the fluid dries and an ulcer forms at the seat of the papule, which heals by contraction, leaving a slight superficial cicatrix. Sometimes this local lesion only takes the form of a slight growth of granulation tissue. Bowerbank[3] states that the abrasion or small sore to which the yaws virus has been accidentally applied may heal up without any change, or a small ulcer may form, and also that when the yaws poison happens to be inoculated through an existing ulcer, this changes its character and becomes unhealthy looking. Moreover, Paulet, in the inoculation experiments already referred to, states that four out of fourteen cases showed *no change* at the point of inoculation, while in the remaining ten the lesion at this point differed in no way from ordinary yaws. It will be seen, therefore, that any lesion at the point of entrance of the poison may be entirely absent, and even if it does occur, it can hardly be regarded as a characteristic primary sore.

The eruption begins as a papule or papules, about the size of a pin's head, single or scattered here and there over the body, and these gradually increase in size until they form tubercles varying from a quarter of an inch to two inches in diameter. As the papule increases the epidermis becomes thinner, until it bursts, leaving a raw papillary surface from which a whitish-yellow, sero-purulent fluid exudes. This forms a yellowish crust on the surface of the yaw,

[1] Paulet, *Arch. gen. de médecine*, Aug. 1848.

[2] Rat, *Essay on Frambœsia*. London, 1891.

[3] Quoted in *Report on Leprosy and Yaws in the West Indies*, by Dr. Milroy. London, 1873.

which can be removed like a cap. In healthy individuals, the surface left after the removal of the crust is bright red, and not unlike a raspberry; but in old and weakly cases the colour is of a dull, dirty yellow or white. Dr. Imray, of Dominica,[1] describes the yaw excrescence as being "not unlike a piece of coarse cotton wick, a quarter of an inch more or less in diameter, dipped in a dirty yellow fluid, and stuck on the skin in a dirty scabby brownish setting, and

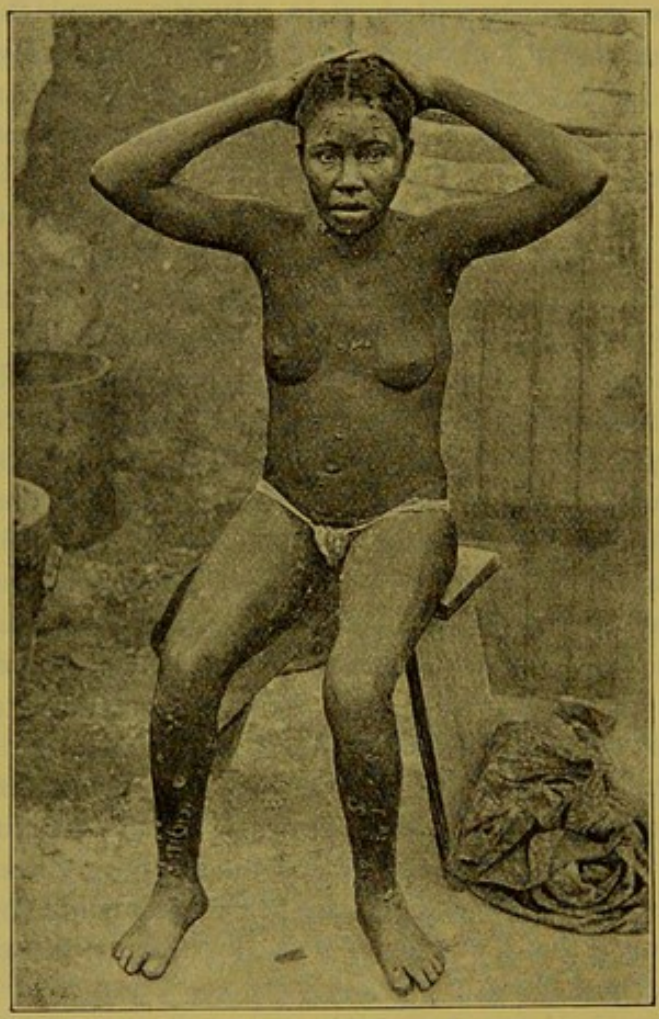

FIG. 46.—The eruption of yaws. (From a Photograph by Prout.)

projecting to a greater or less extent." In shape they are generally round or oval, but may be irregular, from the coalescence of several neighbouring tubercles. Where there is pressure, as between the thighs or in the armpits, they tend to become flattened out. They are somewhat softish tumours, and, unless on the foot or where ulceration has taken place, are not sensitive; in fact, a rough method

[1] Memoir of Dr. Imray in Dr. Milroy's Report.

among negroes of diagnosing a yaw from any other similar sore is to squeeze the juice of a lime on it, very little pain being present if it is a yaw. A peculiar musty offensive smell accompanies the eruption, and there is always itching. The eruption, which mainly affects the face, corners of lips and nostrils, the neck, arms, axillæ, legs, thighs, gluteal region, and vulva, is less frequently seen on trunk and hairy scalp. The tubercles vary greatly both in number and size. There may be only a single yaw, characterised by its size and persistence, which is known as the "mother-yaw" or "mama-pian." Many of the papules recede, and never form tubercles. Exposure to cold may cause them to disappear partly or wholly. The appearance of the eruption is shown in Fig. 46.

The tendency of the yaws tubercles is towards cure; the tubercle contracts, gradually dries up, and eventually a spot, which may last for years, is left, darker than the surrounding skin, but with no cicatrix or depression indicating a loss of tissue. Should there be white blood in the patient, the spot is rather lighter than the surrounding skin. With the disappearance of the eruption, the general health recovers, and in ordinary cases no evil results follow. All cases, however, do not terminate in such a favourable manner, and this is particularly the case in individuals who are debilitated from any cause, such as improper food and bad treatment, or in strumous constitutions. In such circumstances the tubercles, instead of disappearing, increase in size, form large irregular ulcers which eventually involve the deeper tissues, and result in irregular, radiating cicatrices. Contractures and stiffness of joints may follow these, and patients may become helpless cripples. Gangrene of the foot and loss of toes may even result. Occasionally a serpiginous form of ulceration takes place, usually below the knee, long after the tubercles have ceased to exist. Destructive ulceration of the throat and nasal bones may occur, but is rare, and then only in long-standing cases. Sometimes the bones become affected, but this is generally secondary to ulceration. A fatal termination may take place from extensive ulceration and subsequent exhaustion.

During this stage there may be no noticeable symptoms beyond the eruption, the patient's appetite remaining good; he is able to move about, and suffers little inconvenience except from the presence of the yaws. On the other hand, the eruption may be accompanied by fever, shooting pains over body, occasional cramps, want of energy, and loss of appetite.

The stage of eruption may last from two months to several years, successive crops of yaws coming out at intervals. The duration is affected greatly by treatment, food, and hygienic conditions.

Occasionally one or two yaws—"memba yaws" (*i.e.* remember yaws) —may appear at a considerable period of time after every symptom has disappeared; but whether this is due to a recrudescence of the previous attack, or to a reinoculation, it is difficult to say. It is not impossible that it may be comparable to the mild attack of smallpox which is sometimes seen in persons who have had a previous attack, and who have been exposed to reinfection.

There are various modifications of the eruption which I have just described. "*Ringworm*" *yaws* is characterised by the presence of tubercles, arranged wholly or partly round a portion of healthy skin. This ringworm appearance may be the result either of several tubercles growing together, or of the manner in which a large one heals up.

Another form is that known as *pian dartre*. Instead of tubercles, of which, however, there may also be one or two, there are spots or patches of a yellowish colour, in which are seen minute pin-point elevations covered with dirty white scales. This form is very persistent, and very rebellious to treatment.

In *pian gratelle* we find a number of small slightly prominent vesicles distributed over the body. It may sometimes follow an ordinary eruption of yaws, and is also very intractable.

When yaws affects the soles of the feet or palms, the name *crab yaws* is applied. The thickness and hardness of the epidermis hinder the development of the tubercles, and cause much suffering and local irritation. When they do break through, they are found to be smaller, and produce a more abundant exudation of serous fluid. The *running crab yaws* is a variety of this, where the exudation takes place through a series of sieve-like openings or fissures. The cuticle is often greatly thickened around these openings.

A form of yaws is met with in fowls, known as *fowl yaws*. The eruption is most plentiful about the head and beak, but also affects parts of the body which are not well protected by feathers. The tubercles resemble exactly those seen in man. Whether the two diseases are identical is a moot point. I am not aware that the disease has ever been successfully transmitted from the human subject to animals. I inoculated the ear of a small monkey with serum from a yaws tubercle, after having produced a raw surface by blistering; but though a certain amount of local inflammatory action took place, no manifestation of the disease followed. Inoculation of a fowl on the comb was equally unsuccessful.

Etiology.—*Age.*—No age is exempt from this disease, but it chiefly attacks children between the ages of 6 and 15.[1] In the Fiji

[1] Rat, *op. cit.* Table B, p. 54.

Islands the prevalent idea is, that unless a child suffers from coko, he will not grow up a healthy adult; hence, children are sent to inhabit the same house with those suffering from the disease, or are inoculated.[1] *Sex.*—It affects both sexes indiscriminately. *Race.*—No race appears to be exempt, but the white race and even those with mixed blood seem to be less susceptible than negroes. Their comparative immunity may, however, be due to the fact that they are less exposed to contagion. *Climate.*—Yaws is essentially a disease of tropical climates. Hillier and others identify it with the Sibbens of Scotland and Ireland, but considerable diversity of opinion obtains respecting the nature of this disease.[2] *Diet.*—If insufficient in quantity or quality, may have a predisposing effect, by weakening the constitution. It is extremely doubtful whether any particular form of diet has any special effect on the causation of this disease.

Contagion.—It is almost universally admitted that yaws spreads by immediate contact. I myself have never seen a case where another case of yaws could not be traced in the house or neighbourhood. In all cases an abrasion or some solution of continuity of the skin is necessary—the disease will not be produced by the application of the virus to the unbroken cuticle. One of the commonest modes of entrance of the poison is through an ulcer on the foot or leg, round which the yaws begins. It may be transmitted from a mother to a sucking child, and *vice versâ*, and may also be contracted by sexual intercourse. Wearing the clothes of, or sleeping on the mat which has been used by, an affected person, is said to transmit the disease, while the common house fly is believed to be frequently the agent of contagion. Infection through the medium of the atmosphere does not appear to take place. Nurses in yaws hospitals, for example, do not contract the disease so long as they are cleanly and careful to avoid abrasion of the skin. Vaccination is frequently credited with spreading the disease, but Dr. Keelan[3] believes that vaccination mitigates or prevents it. He found (1) that out of several hundred cases of yaws, very few were vaccinated; (2) that the few who had been vaccinated were attacked mildly, and easily cured; and (3) that when some of those suffering from the disease were vaccinated, the lymph took effect in very few, but even in those the disease appeared to be benefited.

Heredity.—The weight of evidence seems to be against this, but it is extremely difficult to prove whether the disease is hereditary or not. Against it is the fact that it does not occur so often among

[1] Macgregor, *op. cit.*, and Skottowe, *Glasgow Medical Journal*, March 1880, p. 211.
[2] Hillier, *Handbook of Skin Diseases*. London, 1865.
[3] *Lancet*, August 5, 1876, p. 201.

newly-born children as during the ages of 6 to 15. Children are never born suffering from yaws as in syphilis—the disease is always developed afterwards, when there have been opportunities for contact.[1]

Yaws, as a rule, cannot be contracted twice, but cases are recorded where one or even two attacks have followed the first. This is simply what is observed in diseases such as smallpox or scarlet fever, where immunity takes place to a greater or less extent. Among natives it is universally recognised that one attack protects against subsequent attacks.

Pathology.—The evidence as to the condition of the internal organs is meagre and unsatisfactory. Van Leent[2] states that the lymphatic vessels are dilated, the lymphatic glands enlarged, and that in some cases they have undergone caseous degeneration. Caseous tubercles are seen in the liver, spleen, kidneys, and lungs.

Hirsch[3] regards the changes in the skin as "a chronic dermatitis proceeding from the papillary layer, and extending deeper into the corium in successive stages of the malady."

On examining the crust of a tubercle microscopically, we find it to consist of cast off epidermis cells, pus corpuscles, and various micro-organisms evidently derived from the external air. A section through a tubercle shows the horny layer of the skin thinned off, and projecting through it a mass of what is practically granulation tissue. It consists of numerous rounded and fusiform cells lying between a network of delicate fibrils and numerous blood vessels. According to Pierez,[4] who was the first to describe any definite micro-organism in this disease, along the surface of the growth, and stretching below it, are seen masses of micrococci with leucocytes in their midst. They are plentiful at the junction of the rete Malpighii and the horny layer. From here they penetrate into the deeper layers of the growth, being found in the lymph spaces and generally through the tubercle. He also detected the same micrococcus in the blood. Beef-broth inoculated from the blood showed cloudiness in three days, and micrococci similar to that described in the tubercle were seen. A tube of nutrient gelatine inoculated from this showed a growth which rendered the jelly diffluent on the surface, and eventually formed a greyish film. He further states that in *pian dartre* and *fowl yaws* a similar micrococcus exists. He concludes from these observations that the cause of frambœsia is a micrococcus, which he proposes to call the "micrococcus frambœsia." To complete this

[1] Rat, *op. cit.* Tables C, D, E, F, pp. 55–57.
[2] Van Leent, *Archiv de méd. navale*, 1880, No. 425.
[3] Hirsch, *Geog. and Hist. Path.* vol. ii. (Eng. trans.).
[4] Pierez, *Thesis for Degree of M.D.* Edinburgh, 1890.

chain of evidence there is, however, one important link wanting, viz. that he has not proved that in an animal inoculated from a pure cultivation of this organism a disease similar to yaws is produced. Should his observations be confirmed, a flood of light will be thrown upon the nature, contagiousness, and manifestations of this disease.

Diagnosis.—The principal disease with which yaws may be confounded is syphilis. Almost every observer who has had an opportunity of observing both diseases is of opinion that yaws is a disease *sui generis*. On the other hand, such distinguished physicians in Europe as Sydenham, Copland, Hebra, and Alibert, etc., are inclined to class it with syphilis. The proof that the micrococcus which has been described is the cause of yaws, and the absence of a similar micrococcus in syphilis, will settle the question as to the identity of the two diseases; but, in the meantime, the following points of difference may be noted. In syphilis there is a characteristic primary sore; in yaws there is often nothing at the point of inoculation.

Syphilis is distinctly hereditary, yaws probably not so. Syphilis is known to affect the fœtus *in utero*, yaws never. In syphilis there is no itching, in yaws this is present, and sometimes very severe. Syphilis generally affects adults, yaws children. Yaws is practically confined to the tropics, syphilis is found in all climates. Exanthematous eruptions and febrile diseases exercise a retarding influence on yaws; they do not do so in syphilis. Finally, we may ask, if yaws is syphilis modified by climate, race, etc., why is it that we find the two diseases independently side by side in the same place, and never find the hard chancre of syphilis followed by an eruption of yaws, or a case of yaws in one member of a family producing syphilis in another? Why is it that where yaws has ceased to exist syphilis still remains? It must be remembered that in the negro race we find the same manifestations of syphilis as in the European. Would we not expect to find some interchange still taking place if they were the same disease?

Prophylaxis.—Persons exposed to contagion should, of course, be very careful as to abrasions of the skin, should pay great regard to cleanliness, and should live under as healthy conditions as possible. So far as communities are concerned, the only preventive treatment is isolation, which to be effective must be compulsory. As I have already indicated, in the island of Dominica it has been found necessary to pass a local ordinance compelling affected individuals to resort to yaws hospitals, or, in the event of refusal, providing for imprisonment as a penalty.

Treatment.—During the attack the greatest attention should be given to personal cleanliness. A warm bath with plenty of soap

should be taken every day. The patient should be warmly clothed, and especially while the eruption is coming out, in order to guard against its disappearance; and he should live under as good hygienic surroundings as are attainable. Exposure to sudden changes of temperature should be avoided. Food should be nourishing, and should consist of fresh meat, fish, rice, yams, etc.

While the eruption is coming out, remedies which will keep the skin and bowels acting are indicated. Sulphur and bitartrate of potash may be given in daily doses. Dr. Rat recommends ammonium carbonate in frequent doses of 2 grains with tincture of guiacum. Warm diluent drinks should be given freely, with light, easily-digested food. If there is much weakness and anæmia, iron and quinine may be given with advantage.

When the tubercles are well out, I have found the most useful drug to be the liquor arsenici et hydrarg. iodidi in 5 to 10 minim doses, thrice daily and gradually increased. Iodide of potassium may be given along with this in 10 to 30 grain doses. Some of the other preparations of mercury are also recommended, *e.g.*, liquor hydrargyri perchlor. ʒi. to ʒii. three times a day, with decoction of sarsaparilla; or pil. hydrarg. subchlor. co. in 5 grain doses. Dr. Rat recommends calomel fumigation. I have had no experience of this, and in the majority of cases it is inapplicable. Arsenic is more especially indicated when the eruption is badly developed or scaly, as in *pian dartre.*

Locally, I have found the sulphate of copper most efficacious. The tubercles are regularly touched with a crystal, and gradually disappear under its use. A corrosive sublimate lotion is also very beneficial, but Dr. Imray prefers a carbolic acid wash. When ulcers are forming, the sores may be well washed with corrosive sublimate lotion, dusted with iodoform, and covered with lint. Ung. hydrarg. nitratis is often a useful application to large tubercles or ulcers. Where *crab yaws* occur, the feet should be well soaked in hot water or poulticed, the softened skin pared away until the yaw is exposed, and touched freely with a crystal of sulphate of copper.

During convalescence, tonics—particularly iron and arsenic—should be persisted in for some considerable time.

CHAPTER XV.

TROPICAL DIARRHŒA.

BY SIR JOSEPH FAYRER, K.C.S.I., LL.D., M.D., F.R.S.

Etiology and Natural History.

Tropical diarrhœa, diarrhœa alba, chronic tropical diarrhœa, hill diarrhœa, white flux, sprue, psilosis, alphœo-gastro-enteritis-tropica, gastro-enteritis-aphthosa-indica, are synonyms applied to a form of disease which frequently comes under observation as the result of residence in China, Cochin-China, Batavia, Java, the Indian and Malayan peninsulas, Ceylon, and probably in other tropical and subtropical countries, and occasionally in extra-tropical regions.

It is a peculiar form of disease, which is the result most frequently, if not always, of tropical and climatic influences, and the debility and cachexia induced thereby. Our rapidly and widely extending relations with foreign countries, by bringing us into closer and more frequent communication with them, are also making us more familiar with this and other varieties of exotic disease, so much so, that this form of diarrhœa may frequently be seen in Europe, especially in localities resorted to by former tropical residents.

The disease is apparently confined to adults; at all events its occurrence among persons below that period of life is rare. It is insidious in its onset, generally slow in progress, wasting in its effects, and when continued beyond a certain stage, fatal, owing to irreparable degenerative changes.

It has been described by several observers; among them may be mentioned Annesley, Twining, Martin, Grant, E. Goodeve, and Fayrer in India; by Manson in China, under the name of Sprue; by Vander Burg in Batavia, by Bertrand and Fontan, and by Thin in a most valuable paper, in which he proposes to give it the name of psilosis, this term (from ψιλος = bare) being expressive of rawness or bareness of the tongue and of the intestinal mucous membrane, which is a prominent feature of the disease.

Most of the cases seen in this country come from China, India, or Ceylon. In India it is frequently met with, where a form of it is known as hill diarrhœa, being so called from its proneness to affect dwellers in the hill stations, especially those who have been subjected previously to the debilitating influences of the plains; atmospheric changes, vicissitudes of temperature, greater altitude, rarefied atmosphere, and possibly water being concerned in its causation. It is not, however, restricted to the hills, for the hill diarrhœa so called, and the while flux, which may be seen in any part of the country, are so much alike as to justify the belief in their identity, at all events for practical purposes, though some high authorities, including Scriven, consider that so-called "sprue" is a distinct disease; as also Crombie, who looks upon hill diarrhœa as distinct from tropical diarrhœa; occasionally there is a resemblance to certain forms of chronic dysentery; indeed, the two conditions may be associated, or the one may merge into the other.

It is to be regarded as a climatic disease; but its relation with malaria seems to be remote rather than immediate. It is frequently seen in this country, for the reason that those who suffer from it are generally sent home for change of climate,—often unfortunately too late,—and it occasionally makes its appearance some time, even years, after the subject of it has returned from the tropics, and may apparently have been in fairly good health on leaving the country where the conditions which gave rise to the disease originated. It not unfrequently begins spontaneously and insidiously without any previous apparent derangement of health; though in other cases it is preceded by dysentery, ordinary diarrhœa, or some indication of malarial infection, or functional derangement of the liver and other abdominal viscera; and so little does it disturb the general health and wellbeing, that it is often not noticed until wasting, loss of strength, and soreness or excessive tenderness of the tongue compel the patient to recognise the gravity of his condition. Such persons have probably become unusually susceptible to the cold, damp, and other vicissitudes of climate.

Here I may note that persons in this country suffering from recent climatic malarial cachexia, attended, it may be, with hepatic and splenic derangement and portal congestion, are liable to be troubled with diarrhœa of irregular character, generally occurring in the morning after eating, or after exposure to chill and mental or physical fatigue, and errors in diet. This may, if not properly dealt with, be the precursor of the more serious, chronic form of diarrhœa, but is often itself obstinate enough, especially if, as remarked, there be hepatic or splenic complications, or any vestige

of previous dysentery. This, however, is not so grave in character as the white diarrhœa, whilst recovery is often rapid enough to show that it was due rather to functional derangement than to structural degeneration. Here it may be added, that all these forms of the complaint, if promptly and carefully treated at the outset, are comparatively amenable to control if unattended by structural changes in the viscera, after the portal system has been relieved by appropriate remedies, very careful attention to diet, warmth, rest, and sound hygienic conditions.

Semeiology.

Tropical diarrhœa often begins with simple looseness of the bowels, attended with little or no pain, though it may supervene on chronic dysentery or ordinary diarrhœa. The action produces a sense of relief rather than of suffering. The dejecta at the first may be natural and bilious, but gradually become light coloured, frothy, pultaceous, and copious. The patient at first feels no inconvenience beyond that of frequent calls to stool and a peculiar tenderness of the tongue; but in some cases the action of the bowels is attended by tenesmus and tormina. He sooner or later finds that he is losing flesh, strength, and energy, and begins to appreciate the serious character of the complaint. He may still retain a good appetite and spirits, and is reluctant to admit that the disease is gradually gaining ground.

As the disease advances, the pale, frothy, copious, semi-fluid character of the motions becomes more marked. They are occasionally tinged with blood, the sufferer becomes more attenuated and exhausted, incapable of any prolonged exertion, and very sensible of loss of strength and energy. The appetite may still continue good, but the rawness and tenderness of the mouth and tongue, which are frequently accompanied by aphthous spots and ulceration, interfere with its gratification, as anything salt or pungent, wine or alcohol, are dreaded from the smarting they cause. The appearance of the dejecta is suggestive of deficiency of bile, whilst the microscope or even the naked eye detects the débris of vegetable, muscular and fibrous tissue, or other portions of the ingesta, passing unaltered. There are also certain forms of micro-organisms. The light colour is not, however, due to any specific disease of the liver. It is remarkable that the fæces should present this appearance—and they do so irrespective of the nature of the diet, though it is more conspicuous when the food is chiefly milk. At the same time, there is absence of bile pigment in the urine and skin. This condition

may continue for months; but with returning health the normal coloration returns, whilst at the same time there is no evidence of liver disease beyond the shrinking which that viscus shares with other organs in the general wasting, and which disappears as recovery is assured. This shrinking of the liver is frequently, probably erroneously, attributed to atrophy of a more serious nature.

Diarrhœa, though a general characteristic of this disease, is not always a prominent one. Cases occur—it has been observed by the writer of this article more frequently in patients from Ceylon than other parts of the tropics—in which the soreness of the mouth and tongue, and wasting from want of assimilation, are the chief characteristics; the excreta are not normal, though neither copious nor fluid, whilst the wasting and exhaustion are scarcely less severe than in the typical form of the disease.

The subjects of tropical diarrhœa are pale and emaciated, with the skin dry, flaccid, flabby, sallow, and sometimes pigmented in patches. The abdomen is soft and flaccid, and the intestinal coils can often be felt through the wasted abdominal parietes.

The fat disappears everywhere, the eyes become pearly and sunken, the gums pale and shrunken, the lips and conjunctivæ are blanched and anæmic. The tongue, at first pallid and flabby, becomes shrunken, red, and glazed; the papillæ are obliterated, and in the advanced stages in many cases the organ is much contracted, whilst it is raw, smooth, and tender, often affected, as is the buccal mucous membrane, by aphthous ulceration, and in some cases covered with a thick coating of white epithelial scales. The state of the tongue is generally significant of the progress and the stage of the disease; a return to the normal condition, the renewal of papillæ, is a hopeful sign; whilst increasing glazing, redness, and tenderness with aphthæ, are of evil augury. Needless to say that a profound condition of anæmia is induced in the advanced stages.

In the outset, or even in the course of the disease, evidences of malarial action are occasionally manifested in recurrences of fever, which is in some instances severe; or there may be other vague conditions of malaise or disorder, myalgic or neuralgic, such as are common to those who have been subjected to malarial influences, or dyspepsia, flatulence, and excessive irritability of the bowels, which are provoked to action by the use of any food, however simple. Languor, physical and mental inertia, go on increasing until exertion becomes as difficult as distasteful. These symptoms may be mitigated by treatment, but the improvement is often more apparent than real. The disease slowly and insidiously progresses, till at last the patient is compelled to give up work and seek

recovery in change of climate. Should this not prove effective, especially when too long postponed, the symptoms increase in gravity, the patient rapidly emaciates, and is easily fatigued or exhausted. The breathing becomes feeble and accelerated, temperature sub-normal, extremities œdematous, whilst anæmia is profound; the urine is often albuminous, the alvine evacuations and the tongue present the appearances already described; catamenia are suspended or diminished.

If this evil condition be not arrested, and it seldom is when it has gone so far, especially if it have been of long duration and not a first attack, and if age be advanced, still more if there be other visceral complications, fatal exhaustion or pulmonary embolism or thrombosis soon closes the scene.

Here it may be noted that the absence of bile pigment in the motions, urine, and skin, or the presence of a certain amount of albumen in the urine, are not necessarily indications of organic disease, either of liver or kidneys. The former is generally if not always observed, the latter not unfrequently, and yet perfect recovery may follow.

This disease appears to be the consequence of general degeneration rather than specific disease of any one organ, though the intestinal tract seems to be the seat of the most marked change. Anæmia, atrophy, and exhaustion are prominent indications during life, and after death the wasting which is so marked in the body, is found to be even more pronounced in the viscera.

Morbid Anatomy.

When death has occurred at an early period, the intestines have been found contracted, with the mucous lining thickened, congested, and even ulcerated; but when at a later period the coats of the bowel are found to be attenuated, diaphanous, and the seat of fatty and lardaceous degeneration, with occasionally ulceration in the ileum or colon. The intestinal and mesenteric glands are atrophied and degenerated, the latter sometimes enlarged; the mesentery itself wasted; the liver shrunken, pale, and contracted, so as in life to have given rise to the belief that it was specially atrophied; the spleen, pancreas, and kidneys in a similar condition; occasionally, though not in ordinary cases, there may be chronic enlargement of liver or spleen, or of both.

It has often been thought that the complaint is necessarily associated with chronic disease, or functional derangement of the liver. No doubt it may sometimes be so, but the so-called atrophy of

ordinary cases, so often noticed, disappears when the diarrhœa ceases, and health is re-established.

E. Goodeve showed that the mucous membranes and follicles were sometimes found in a state of atrophy and amyloid degeneration. The writer of this article pointed out, many years ago, that in the chronic form there is not so much surface change as thinning of all the coats of the small intestines, so that they become translucent, whilst there is atrophy of the glandular structures. When dysenteric symptoms have appeared in the course and towards the close of the disease, ulceration of portions of the colon have been found, the liver being flaccid, anæmic, and small, but free from other morbid change.

With reference to amyloid or lardaceous degeneration, Dr. McConnell, Professor of Pathology in Calcutta, says, whilst amyloid degeneration of the bowel, as far as his experience goes, is rare among the natives of India, yet, of all parts, the intestines are least frequently affected; but he confirms the accuracy of the account given by Dr. D. D. Cunningham of the morbid anatomy of the intestines of the sufferer from chronic diarrhœa and dysentery in famine seasons; and the condition is probably like that now under consideration.

Dr. Cunningham's valuable and elaborate report on the famine diarrhœa shows how the degenerative changes involve the intestines as well as other viscera, and the bearing of his observations on the pathology of chronic tropical diarrhœa is interesting. The following is an abstract of Dr. Cunningham's observations :—

The mucous membrane of the stomach, jejunum, and duodenum was white, pulpy, soft, and bloodless ; there were some traces of congestion, marks of disintegration in the jejunum, disappearances of the epithelial coat, pigmentation and atrophy of the sub-epithelial areolar tissue. The mucous membrane of the ileum presented more evident signs of change ; it was bloodless generally, but here and there showed patches of congestion ; the epithelial coat had become disorganised or was absent, and the sub-epithelial tissues were affected. The solitary glands were either very few or almost entirely absent. Peyer's patches were an empty network of elevations surrounded by slightly elevated ridges ; the adenoid tissue was virtually absent ; the surface was smooth ; the villi were almost unrecognisable ; the muscular coat, covered only by a thin membranous investment, seemed to form the inner lining of the tube ; the change was most pronounced over the lower half or two-thirds of the ileum.

Microscopic examination showed that the epithelium, where present, contained much fat, the cells including large quantities of

oil granules. In the vicinity of denuded areas, vestiges of cells and free granular matter were present. The sub-epithelial tissue showed evidence of similar changes; the nuclei were granular and oily. This may, to some extent, have caused the appearance of pigmentation that was observed, but there was a certain proportion of pigment granules.

It is impossible to say whether this deposit of pigment in the mucous membrane is essentially connected with the morbid processes. It is a phenomenon of other wasting diseases, and is generally ascribed to antecedent minute extravasations of blood; but it may be due to degenerative processes. In the more advanced stages of atrophy of the mucous membrane, the nuclear elements in the villi appeared to be almost entirely absent, and the villi were represented by abortive processes containing granular matter.

The mucous membrane of the large intestine was also generally anæmic, white, but mottled pink here and there, and its texture was very soft. In some cases there was thickening. Besides the general softening, there was evidence of loss of substance in some places. These patches, in certain instances, might be called ulcers, in others they seemed to be merely the result of disintegration and atrophy. In such instances there was no evidence of congestion, and the colic lymphatic glands were pale and inconspicuous. The appearances presented by the mucous membrane were sometimes more like those of dysentery. In one case this was decidedly so. There was great thickening of the gut; the mucous membrane was broken up into hard rough masses of purple and green colour; distinct ulceration had occurred, and there was considerable congestion, whilst the lymphatic glands were turgid and of a deep purple colour.

Fatal cases of the so-called famine dysentery may occur in which no true dysenteric processes, as ordinarily understood, are present. The essential process appears to be the same as that affecting the small intestines, but it may be complicated in various degrees by the supervention of true dysenteric changes. Does it not show how nearly the two diseases are allied; perhaps only different manifestations of the same? The liver was small, pale, yellowish, fatty-looking; in some, the cells were healthy; in others, full of fat granules. The gall bladder was half full of bile; it was healthy in those where the cells were healthy; in the fatty, it was pale, yellow, and very thick. The spleen was small and firm, in most cases conspicuously so. In some cases it appeared to present little save a dense contracted mass of stroma in a white shrivelled capsule. The kidneys, as a rule, were pale and anæmic; in some there was fatty degeneration of the epithelium. The pancreas was normal in some, in

others its substance seemed as though it were opened out, the lobules widely separated. The brain was slightly anæmic. The general result of the entire series of observations was to show that the diseased conditions were specially characterised by extreme general anæmia, and destructive processes affecting the mucous membrane of the intestinal canal.

Dr. Thin remarks that with regard to the pancreas it was found to be healthy in the majority of cases examined.[1]

Dr. Manson says that in several cases the pancreas was found to contain centres of degeneration and even suppuration.

Professor Aitken[2] says: "Lardaceous disease of the intestines comes next in frequency to that of liver, spleen, and kidney, and involves the internal capillaries of the villi and surrounding network of mucous and sub-mucous tissues; it progressively involves the villi, mucous, and sub-mucous capillaries; there is infiltration round the solitary glands, and degeneration of the vessels surrounding Peyer's glands." He has repeatedly met with it in the cases of soldiers at Netley, when it involved the whole tract from mouth to anus. Anæmia of the mucous membrane, with a peculiarly glistening or shiny aspect of the surface is the most characteristic sign of the lesion, "otherwise there are no outward signs to attract attention"; the application of the iodine test is necessary to detect it.

Aitken refers to Virchow's account of the condition: "Virchow has known the villi to drop off and the intestine to be bare of them." Such, there is little doubt, would be found generally to be the case in fatal examples of chronic tropical diarrhœa, though "the villi are sometimes changed into lardaceous or albuminoid material."

The absence of epithelium and obliteration of villi are very remarkable in the tongue.

Besides these characteristics, an absence of fat and a wasting of the tissues generally are conspicuous.

The opportunities of studying the morbid changes that take place in ordinary examples of the disease are not frequent, as where death results it is often under circumstances where post-mortem examination is not readily obtained. The pathological changes above described are such as have been observed where examination has been permitted.

With reference to micro-organisms connected with this disease, Dr. Thin[3] states that cultivation of organisms from a case of this kind were made, the medium being neutral, meat-peptone gelatine.

[1] *Lancet*, April 23, 1892.
[2] Aitken, *Science and Practice of Medicine.*
[3] Psilosis or "Sprue," its nature and treatment.

Thirteen distinct forms were isolated, seven being micrococci, six rod-shaped bacteria, and there were others. These thirteen organisms grew in the gelatine, and produced an appearance perfectly distinctive. The cultivations were watched during three months, and it was observed that they were distinguishable by their effects on the gelatine as well as their naked-eye appearances. They presented various shades of colouring. One of the bacilli, a very small one, was characterised by the great rapidity with which it liquefied the gelatine; and he satisfied himself that the worse the stools were, the greater was the proportion in which this bacillus was present.

In a more recent communication to the Medical and Chirurgical Society on April 23, 1892, Dr. Thin refers to a microscopic examination by Dr. Wethered of soft masses passed with the stools after the motions had become solid. They were found to consist of almost pure cultivations of bacteria, a rod-shaped, moderate-sized bacterium being most prominent. Reference was also made to a paper by Macfadyen, Nencki, and Nieber, in which, in the case of fistula near the ileo-cæcal valve, it was shown that the food mass that passed into the colon was acid, but the reaction of the mucosa of the ileum alkaline; both the mucosa and contents of large bowel were alkaline. But reasons were given to show that in the so-called sprue the alkaline secretion of the mucosa of the ileum was wanting, that the food mass that passed into the large bowel was more than usually acid, and that the fæces were acid. It was argued that the abnormal reactions of the intestine must produce abnormalities in the development of the bacteria normally present in the bowel; and it was suggested that the normal colouring matter of the stools, under the influence of abnormal bacterial products, either was not formed or was destroyed. The same condition, but to a less marked extent, obtained in some cases of ordinary tropical diarrhœa. Clinically the fact was established that persistent white stools might exist, whilst both the liver and the pancreas were healthy.

The subjoined notes by Dr. S. Martin and Professor Macfadyen are a most interesting contribution to the pathology of this complaint.

Tropical Diarrhœa.

From Sir Joseph Fayrer, a sample of typical stools from a case of tropical diarrhœa. The bacteriological examination is still in progress, and any observations I now make must necessarily be of a preliminary nature. A description of the organisms isolated, and their pathogenic action, will be given when the examination of this and samples from other cases is finished.

By way of introduction a few words may be said concerning the bacteria present in the normal and abnormal digestive tract.

The digestive tract normally contains a large number of bacterial species. The mouth is particularly rich in organisms. There are many also present in the stomach. The small and large intestine contain a great variety of bacteria. Microscopically, it is almost impossible to recognise with certainty each individual species present, and to differentiate the one from the other. That must be done by endeavouring to cultivate the bacteria on nutrient soils outside the body. Till that is done, one is not in a position to accurately determine their morphology and pathogenic action.

The writer had the opportunity of examining bacteriologically during a period of six months the discharge from a fistula at the lower end of the small intestine. The patient was a woman in Berne Hospital. The examination made enabled one to determine the nature of the bacteria present in the *normal* small intestine of man. It was found that whilst a large number of bacterial forms were present in the normal small intestine, *no* one kind predominated. The bacteria present consisted of a mixture of many different species. Further, the contents of the normal small intestine had a faintly *acid* reaction, due to organic acids, such as acetic acid, etc. This acid reaction had an inimical effect on the growth of the organisms, and would seriously check the development of any pathogenic organisms which happened to be present. The bacteria normally present in the small intestine were those which ferment carbohydrates, and not those which decompose albumens.

In a fatal case of acute intestinal catarrh the conditions were found to be different. The contents of the small intestine had a distinctly alkaline reaction. The multitude of bacterial forms found in the normal small intestine were no longer present, or were so in greatly diminished numbers. Certain micro-organisms *predominated*, and the ordinary harmless saprophytes of the digestive tract were, so to speak, driven into the background; only two bacteria could be isolated under these abnormal conditions. They were bacilli, and one of them had an active disintegrating action on albumens. It will be seen that the results were different to those obtained on examining the normal digestive tract.

It is therefore important to be acquainted with the bacteriology of the normal digestive tract before drawing conclusions as to the appearances presented in the abnormal.

The stools from the case of tropical diarrhœa were in the first instance microscopically examined.

The fresh unstained specimens were crowded with micro-organisms. The first point noted was the predominance over the others of one special form, viz. a bacillus.

Stained specimens likewise showed that one form of bacillus predominated. It was about the size of the anthrax bacillus, though somewhat thicker. Its ends were flattened, and it was motile.

The *Bacterium coli commune*, which is well represented in every normal stool, was only present in small numbers. A search was also made for amœboid organisms, but the writer did not succeed in finding any. It would, of course, be rash to say that they are therefore not present in cases of tropical diarrhœa. One would have to examine a number of cases before coming to a definite conclusion on this point. It might also be that in this particular instance the amœboid organisms had disappeared from the stools before the examination was made.

Cultures were made from the stools in nutrient gelatine and agar.

The number of colonies of the *Bacterium coli commune* present on the gelatine plates was much below the average found in normal conditions.

The bacillus, with flattened ends, already described, was isolated. It grows well in gelatine, and produces large gas bubbles in the gelatine. The writer has not yet fully studied its morphology or determined its pathogenic action. A full description of it, and the other organisms isolated from the diarrhœic stools, must therefore be reserved for a second communication. By that time I hope to be able to determine their significance in relation to the disease.

Further bacteriological notes, by Dr. Allan Macfadyen.

It has undoubtedly been proved that in certain diseases of the digestive tract occurring in hot climates, large numbers of amœboid organisms are to be found in the large intestine. We are not yet in a position to ascribe to these parasites a definite *rôle* in the causation of intestinal disease. The subject is one that demands careful and exhaustive study. The interesting researches already published will no doubt stimulate investigation generally in this direction, and furnish fuller data than at present exist. It may then be found that more than one group of micro-organisms is concerned in the production of such diseases as tropical diarrhœa and dysentery. It is only when the causes are discovered that we will be able to explain the various phases these diseases are capable of assuming.

Massintin[1] found amœboid parasites in five cases of intestinal disease, viz.—

1. Chronic dysentery.
2. Chronic intestinal catarrh.
3. Typhoid fever, with late diarrhœa.

4 and 5. Diarrhœa, with fluid mucoid stools.

He supposes that the parasites gain access to the intestine through water, and does not ascribe any special significance to their presence.

Dr. Kartulis, as already stated, discovered similar parasites in the large intestine in cases of dysentery. His observations were made on more than 500 cases.[2]

When the dysentery was complicated by liver abscess, the amœbæ were also found in the abscesses. The primary lesions in the liver he thinks may be due to the amœbæ, and that the ensuing suppuration is caused by micrococci carried there with the protozoa by means of the portal vein.

Professor Osler, of Baltimore, while confirming Kartulis' observation, quotes the following criticism by Baumgarten: "We will not contradict this view, as many old and recent observations show very similar amœboid forms occur in other intestinal affections, and even in normal fæces. We regard it, however, as unlikely that the amœbæ could induce all of the conditions in the dysenteric process. We have no analogy to show that amœboid parasites can induce ulceration, and we rather believe that the pyogenic organisms, well known as exciters of ulcerative processes, are concerned with the amœbæ in the causation of tropical dysentery."[3]

The causal relation of amœbæ to intestinal disease is very fully discussed in a recent monograph by Drs. Councilman and Lafleur on "Amœbic Dysentery."[4] This admirable paper contains an account of the authors'

[1] *Centralblatt für Bakteriologie*, Bd. vi. p. 451.
[2] *Virchow's Archiv*, Bd. cviii.
[3] *Lehrbuch der Pathologischen Mykologie*, Bd. ii. p. 937.
[4] *Johns Hopkins Hospital Reports* for 1891.

researches on this subject. Its value is also greatly enhanced by an exhaustive bibliography. They thus state the object of their investigations: "To study the diseased conditions in which the amœbæ are present both from a clinical and anatomical point of view, to determine whether the amœbæ are always associated with definite anatomical lesions which can be separated as a unit from a number of different anatomical conditions which have been described under the term dysentery, and whether these are distinctive clinical symptoms which accompany the anatomical lesions or not."

They find that the lesions produced by the amœbæ differ in mode of production and kind from those due to the action of bacteria. The ulcers differ from those found in any other form of dysentery. They are produced by infiltration of the sub-mucous tissue and necrosis of the overlying mucous membrane, and have in consequence the undermined form. In the lesions, unless complicated by the action of bacteria, there is an absence of the products of purulent inflammation. They conclude that what they term "amœbic dysentery" is separated, not only by its distinctive pathological anatomy, but also by its etiology and clinical history, from other affections of the intestines with which it has hitherto been classed under the general name of dysentery.

Abscess of the liver is a more frequent complication in this than in any other form of dysentery. The important statement is also made that in these liver abscesses the amœbæ are *not* associated with any other organisms. In the abscesses of the liver they found no pus organisms. It would appear from this, then, that they are *not* true abscesses. It will be observed that the investigations of Councilman and Lafleur were made on cases where there were dysenteric lesions of the intestine. With regard to the etiology of tropical abscess of the liver they remark: "Whether or not the amœbæ are capable of producing the abscesses of the liver so often met with in the tropics without any ulceration of the intestine, must still remain *sub judice*."

In the one case of tropical abscess of the liver investigated by the writer there was no history of dysentery. The pus, bacteriologically examined, yielded a pure culture of the *Staphylococcus pyogenes aureus*. Amœboid organisms were not detected. It seemed to be a simple infection of the liver by pyogenic micrococci.

As regards tropical dysentery, the different phases that disease may assume may be due to the action of different groups of organisms. The liver abscesses that complicate the disease may also result from the action of more than one form of parasite. Whilst the primary lesion is due to a non-pyogenic organism, its development or not into a true abscess may depend on the presence or absence of pyogenic bacteria. In the case of the small multiple pyæmic abscesses of the liver, there may also be a mixed infection.

The writer has not yet been able to detect amœboid organisms in the cases of diarrhœa of tropical origin he has hitherto had an opportunity of examining. Here also it is probable that more than one organism is concerned in the production of the disease.

The bacteriology of such affections has still to be thoroughly worked out. And an important feature in such investigations will be the study of the chemistry of the organisms present in the diseased digestive tract, with a view to explaining their mode of action.

ANALYSIS OF THE FÆCES OF A TYPICAL CASE OF TROPICAL DIARRHŒA (BY DR. SIDNEY MARTIN). DR. MCCONNELL.

1. *Microscopically.*
   - *a.* Granular débris with shreds of vegetable cellular tissue and yellow fragments of undigested meat (muscle) fibre—few in number.
   - *b.* Crystals of triple phosphates—few.
   - *c.* Quantities of needle-shaped crystals soluble in ether and alcohol but not in water.
   - „ Crystallised fats (stearine and palmitin) combined with lime. No starch granules.

2. *Watery extract.*
   - *a.* Distinctly alkaline.
   - *b.* Contains no sugar and no starch.
   - *c.* Contains alkali albumen (? casein); a form of albumose and coagulable proteid (globulin).
   - „ The proteids are present in moderate amount.
   - „ Only a trace of alkali albumen, and about equal quantities of albumose and globulin.

3. *Percentage composition.*

	100 parts.
Water,	84·25
Total solids,	15·75
Bile,	0·212
Fat,	4·13
Leucin, tyrosin, etc.,	0·146
Soluble proteids and insoluble matter,	8·752
Ash,	2·55

REMARKS.—1. The absence of starch and sugar shows that these bodies are digested and absorbed in the alimentary canal.

2. The presence of albumose shows that the digestive juices have the power of acting on the proteids taken. The presence of globulin shows either that when taken in the food it is not digested, or that there is a discharge of globulin into the gut (as from an ulcer).

3. The chief point to be noticed is the occurrence of a *large proportion of fat* (in relation to the total solids) and a *small quantity of bile.* Bilirubin is present in an unaltered form—only traces of the bile acids could be obtained. The crystallised fats may be palmitin, stearine, and the lime compounds. The fats, therefore, are not in an emulsified form; they have been partly split up into the fatty acids and glycerine.

4. The presence of leucin and tyrosin shows decomposition.

N.B.—The bile is diminished more than one-half.

TREATMENT.

From the insidious character of the earlier stages of the disease, tropical diarrhœa, wherever originating, not unfrequently gains ground before radical measures for its relief are resorted to. The homeward voyage is sometimes productive of improvement, though much may still remain to be done before recovery is completed. Successful issue depends much on the patient's resolution and

perseverance in carrying out the instructions he receives. Diet is the most important consideration, and must be very strictly regulated and adhered to; and scarcely less important is the question of clothing, habits, and mode of life. Alternations of temperature, errors of diet, fatigue, excitement, exertion, mental or physical, should be avoided; physiological rest should be insisted on. There is a tendency in chronic diarrhœa, in its earlier stages at all events, to get well. The object is to favour this tendency, and not to thwart it by neglect of simple precautions already described.

All irritating ingesta must be absolutely avoided, even the simplest and, under other circumstances, most appropriate forms of nourishment are here unsuitable. Milk alone (cow's milk) should be the only diet, and it must be given in small quantities often repeated, say from four to six ounces every hour, day and night. Larger quantities at longer intervals will not do; even at night, if possible,—and it is possible with the aid of a good nurse or attendant,—this should be insisted on. When, in the twenty-four hours, an adult is able to take three or four quarts of cow's milk in this way, ample nourishment is afforded to support his strength and to enable him to recover from his disease.

The writer of this article began this method of treatment in India, before returning to England in 1872; after trying all other forms of food and remedy, and after prolonged experience, he has found it more effective than anything. The milk should be given pure and simple, without, as a general rule, any addition; and if strictly adhered to it seldom, except in very advanced and chronic cases, or in very aged persons, fails. Under its influence the patient may at first lose weight, but soon regains and even increases it.

In ordinary cases it must be adhered to for three weeks, a month, or six weeks, after which a varied diet may be given, though any change or addition must be tentatively and gradually made. It is seldom necessary to dilute the milk, though occasionally it may be expedient to add a little water, lime or soda water. Boiling or peptonising are unnecessary. The milk should be fresh and sound, and in the hot weather supplied at least twice a day fresh from the cow.

Beef-tea, raw beef juice, or other animal broth, finely minced meat which has not been previously cooked, raw eggs beaten up with milk, may be tried; but in the early stages seldom if ever—it may be said never—agree. Farinaceous food is equally uncertain, and experience has shown that milk *alone* is the best. Tea, coffee, cocoa also, as a general rule, disagree, and should be avoided. Alcoholic stimulants for those who have long been accustomed to them may be necessary, and should be cautiously administered in

limited quantities (from 2 to 6 oz. a day). The best are old brandy or whisky given with the milk, or with some alkaline water such as that of Vichy or Vals. As a general rule all wines are unsuitable.

Dr. Crombie, in an interesting paper in the *Indian Medical Gazette* of May 1892, remarks of the hill diarrhœa as a distinct form of the disease, that it is liable to occur at elevations of 6000 feet or more, in India, Europe, or elsewhere; that it is peculiar to the early hours of the day, beginning between 3 and 5 A.M. and ceasing after 11 A.M.; that it is a form of dyspepsia, and is often attended by flatulence of stomach and intestinal canal; that in many people in Simla, on first arrival, the stools become white, without diarrhœa, but with flatulence.

The influence of the monsoon is potent; when, or even before, the rains set in, the cases of diarrhœa become frequent. In July and August it becomes epidemic; with the cessation of the rains in September it ceases.

Elevation is not the only factor, there is some other condition connected with the monsoon; but he is unable to say whether it is increased moisture, or diminished barometric pressure, or some combination of causes. It seems to be independent of the purity of the water supply. The exemption of children under twelve seems to negative a water theory of the disease.

He points out that the liver function is seriously affected; that the stomach, pancreas, and intestinal canal are also functionally disturbed. The disease is not a true diarrhœa, but an indigestion due to imperfect secretions of the digestive ferments generally, including those of the bowel.

Removal from the hills to a lower elevation in early cases stops it; and especially should the hills be left during the rains.

Dr. Crombie speaks highly of the value of ingluvine, and pepsine, and intestinal antiseptics to check abnormal fermentation. Also of perchloride of mercury; and he points out that to Surgeons Macpherson and Barry is due the credit of first indicating the value of the latter. This drug should be administered, says Dr. Crombie, in doses of 10 to 15 drops of liq. hydrag. perchlor. before each meal; pepsine or ingluvine in doses of 10 to 12 grains after meals is also recommended.

The mercurial preparation Dr. Crombie has found useful in the "very similar condition which is known as tropical diarrhœa."

Dr. Crombie's view of the nature, etiology, and treatment of this variety of the disease is interesting, and probably correct as far as it goes; and his suggestions as to the use of pepsine, ingluvine, and perchloride of mercury should receive careful consideration and trial, for any method of treatment that offers the prospect of amelioration of this troublesome, often dangerous complaint, would be a welcome addition to our therapeutical knowledge.

So far, after many years' experience both in India and at home, of the disease and its treatment, the writer has found milk, carefully and gradually administered, combined with attention to warmth, rest, and other hygienic precautions, the most effective, if not the only remedy that can be relied on. Drugs as a general rule are of no avail.

The return to ordinary diet must be cautiously managed, and very gradual. When any addition to the milk is made, it must be at once discontinued if it seem to disagree. Regularity in the times of administration and in the quantity given is essential. More than six ounces of milk can very seldom be taken at a time; and it is

generally well to begin with two or three ounces, gradually increasing to six for a full-grown adult; in very severe and aggravated cases it may be expedient to give a smaller quantity, say an ounce to two ounces every half-hour. If after three weeks or a month the diarrhœa have entirely ceased, and for some time, and if the tongue be less red or sensitive, and if the papillæ have reappeared, a slight addition may be made; but until this be the case, no change can be attempted with advantage.

After taking the milk for some days, the tongue becomes coated with white fur, but it is less red at the tip and edges; the aphthæ which are often present, as well as the abnormal sensitiveness, disappear. The diarrhœa gives place probably to constipation; the patient feels in all respects better; the malaise and feeling of insecurity about the bowels disappears, and the dry and shrunken condition of the skin is replaced by a plumper and more healthy appearance. The appetite increases, and nocturnal wakefulness gives place to a quiet, restful state, attended by refreshing sleep. When this condition has been attained, a change in diet may begin.

At first bread not recently baked may be added in small quantities, gradually increasing, and thoroughly incorporated as crumbs with the milk. If this agree, after a week or so a further addition of a little finely minced mutton or chicken, which has not previously been cooked, may be made. Should no irritation of the bowels or diarrhœa result, there may be a further addition of a little light pudding or other farinaceous food, such as arrowroot, the milk at the same time being diminished in quantity. Gradually and tentatively a return to meat and vegetables and other light food may be effected; but whenever any sign of gastric or intestinal irritation occurs, the recent addition should be discontinued, and the milk resumed and continued until all disturbance has ceased, and digestion and assimilation are perfectly performed. This is the trying and difficult stage of the treatment; the appetite is good, and the feeling of improvement and self-confidence is so marked, that it is difficult for the patient to realise the danger of indiscretion, and the importance of continued care and caution. Happily these difficulties are often surmounted, and perfect recovery takes place; but even then it is necessary that continued caution should be observed, for relapses are liable to occur.

In addition to the above precautions as to diet, it is essential that others should be observed. The patient must be kept warm, *i.e.* in an equable, moderate temperature, day and night. The body should be covered with flannel or woollen material, and a flannel bandage should surround the abdomen. Chills and damp are most

prejudicial, for exposure to them may seriously aggravate the mischief. During cold and damp weather the patient should remain indoors. Rest also is most imperatively required, and at first in the recumbent posture, if not in bed, at all events on a couch; the greater part of the twenty-four hours should be spent in this posture in the early stages. Exercise must only gradually be taken, and never, even when improvement has considerably advanced, to the extent of causing fatigue.

The patient should reside in some mild and sheltered locality, such as the south coast of England; but the care, comfort, attention, and nursing of a well-ordered home in any part of the United Kingdom outweigh the benefit of any change of climate or locality where these might be absent.

The improved state of the tongue as well as the diminution of the diarrhœa is the best indication of progress. When this has occurred, the discipline may be relaxed, and the patient be permitted, especially if the weather be mild and genial, to take moderate exercise, either on foot or in a carriage; but all shaking and fatigue should be carefully avoided.

As the diarrhœa decreases, the excreta improve in character and diminish in frequency, though under milk diet they are always large, light-coloured, occurring once or twice a day, and gradually, sometimes quickly, increasing in consistency, till constipation may render enemata of soap-water and oil, or laxatives necessary. It is then that the food, as already indicated, may be varied, but very cautiously.

For long after recovery is apparently complete, the greatest care must be taken to avoid errors of diet, over-fatigue, or vicissitudes of temperature. It is desirable that the stay in Europe be prolonged; and it may often be necessary to defer return to a tropical climate till another year, if not indefinitely.

Under the impression that the liver is at fault, chologogues are sometimes given; this as a general rule is unnecessary, but not unfrequently the continued and careful use of saline laxatives, such as the sulphates of soda, or magnesia, combined with some bitter infusion, given early in the morning to relieve abdominal plethora and portal congestion, are most beneficial, the greatest care being taken not to produce overaction. For this purpose the saline waters of Carlsbad or other similar mineral springs, judiciously administered, may prove useful. The chief aim is to restore healthy action by giving the intestines rest, by promoting absorption of nourishment, and delaying the expulsion of their contents; and this is best effected by giving only the blandest and least irritating form of aliment. None have hitherto proved so efficient as simple or

slightly diluted cow's milk. The occasional use of medicines to allay irritability, and, subsequently, of such remedies as may give tone and tend to improve the general health, is desirable. For this purpose small doses—3 to 5 drops—of liquor arsenicalis twice a day, continued over a period of weeks, with occasional breaks of three or four days, are beneficial.

As before said, in the earlier stages, if there be hepatic and, as so frequently happens, portal congestion, salines or even ipecacuanha may be needed. These, however, are quite inappropriate in the advanced stages. When the bowel is very irritable, with pain, and a tendency to watery discharges, small doses of from 5 to 10 minims of tr. opii, or a few grains of Dover's powder, are useful. Bismuth is frequently prescribed, but apparently without any benefit; indeed, it often does more harm than good. The fresh Bael fruit or other demulcent remedies sometimes appear to do good; but experience gradually leads to the conclusion that they are of little use, at all events in the early stages.

As recovery progresses, mild preparations of iron, especially the ferri potas. tart., are beneficial; but they must be laid aside for a while if they appear to disagree. Change to a milder climate, or a visit to some of the appropriate health resorts, may be of benefit, not so much for the water—which, however, when there is portal or hepatic derangement may be useful—as for the important element in insuring recovery, change of locality, with a well-regulated and physiologically correct life, with the tone imparted by the mental attitude of determination to do all that can be done to promote restoration to health. But in the early stages, especially if the disease be severe, nothing can surpass the comforts of an English home, and the soothing influence of a well-managed milk diet.

Experience has shown that in the treatment of chronic tropical diarrhœa, drugs are of little use, with the exceptions already alluded to.

The hygienic and dietetic precautions already described, if resorted to within a reasonable time and strictly adhered to, and where visceral complications of a structural nature have not occurred, and when the patient is not exhausted by old age or debility, are generally successful. Unhappily a considerable number fall within the latter category, and gradually succumb despite treatment, whilst a certain proportion suffer relapses from neglect of sustained observance of the rules laid down; and these after repeated recurrences become at last the subject of irretrievable degeneration of the intestinal tract, and finally die from inanition and exhaustion. Hence the urgent necessity for early interference, and the importance of rigid adherence to the very

simple though irksome regimen which experience has shown to be the most effective mode of dealing with the disease.

Prognosis.

The prognosis depends on the stage which the disease has reached, the age of the patient, and the extent to which degenerative changes have advanced. The writer has frequently seen cases of persons between 20 and 50 years of age, even older, in whom the disease had continued for a year or more, with extreme emaciation, albuminuria, œdema, red, dry, and glazed tongue, with continuous white diarrhœa, recover satisfactorily under the regimen just described. Other cases, occurring at more advanced age, of longer duration, and after frequent relapses, or when complicated with visceral disease, have often obstinately resisted all treatment, though even then a certain measure of relief has been obtained, occasionally recovery.

It has been frequently found that when milk has failed, it was either because it had been mixed with other food, or had been taken irregularly and in too large quantities at a time.

The conclusion arrived at is that, whilst drugs are in most cases unavailing, if milk, given as described, does not succeed, there is little prospect that any other mode of treatment will be more successful. This untoward result, however, may be looked for only in instances where advanced age, long delay in treatment, obstinate neglect of the discipline proposed, or structural changes, have rendered recovery hopeless.

Illustrative Cases.

A few cases, chiefly from the writer's notes, illustrating the symptoms of the milder and more severe forms of the disease, and the results of treatment, are subjoined

No. 1.—Case of ordinary Tropical Diarrhœa. Recovery.

An officer, aged 32, who had been in India ten years, had enjoyed good health for the first six years; after that it failed somewhat, and he had suffered from diarrhœa for the last year. He returned to England in March 1882; was emaciated, though he looked fairly well; motions loose and light-coloured; papillæ of tongue not obliterated; had lost more than 4 st. in weight. Had tried various drugs and diets. On 24th May he began to take milk alone in small quantities every hour, up to 3 or 4 quarts a day. By 11th June the diarrhœa had ceased, and castor oil had to be taken to produce action. The diet then began to be varied.

By 27th June he was quite well; went to the South of Europe, and returned after nine months, having remained in good health.

This was a simple case.

No. 2.—Case of ordinary Tropical Diarrhœa. Recovery.

A gentleman, aged 31, who had been in India for seven years, had for the last two years suffered from constant white diarrhœa. A variety of drugs and diets had been tried, but without avail. He was much emaciated, with tongue red and glazed. On 17th May 1882 he was put on a diet of milk, to be taken in small quantities every hour, until the amount taken daily reached 3 or 4 quarts in the twenty-four hours. On 31st May the diarrhœa had ceased. His diet was then modified by the addition of simple food; his weight increased, and he soon completely recovered.

No. 3.—Case of ordinary Tropical Diarrhœa. Recovery.

A gentleman, aged 45, had been in Mauritius for twenty-one years, the last time for fifteen years without coming home. Quite well until Christmas 1881. Returned to England in July 1882, suffering from tropical diarrhœa; two motions on an average daily, frothy, light-coloured, no pain. Frequently has aphthæ in the mouth; tongue not smooth. Put on milk diet and Bael sherbet on 30th October 1882, and remained on it with nothing else for thirty days. By 15th January 1883 was much better. Could take almost anything with care. Was ordered to take moderate exercise, but to avoid over-fatigue. He ultimately recovered.

No. 4.—Case of Tropical Diarrhœa. Recovery.

A lady who had suffered from white diarrhœa for upwards of four years in India returned to England on 28th May 1882. When she came under observation, on 20th March 1883, the tongue was glazed, the mouth aphthous and ulcerated, the appetite was impaired; there were anæmia and constant white diarrhœa, generally painless, most troublesome in the morning. The urine, though scanty and high-coloured, was free from albumen.

Many drugs and forms of diet had been tried without success. She was put on a diet of milk alone, and complete rest was enjoined. She began with about 4 oz. every hour, increasing the quantity gradually until by the end of the first week she was taking 3 quarts in the twenty-four hours. Improvement began at once, and by the end of the third week the diarrhœa was checked. She remained free from it, and in May 1884 was in excellent health.

No. 5.—Case of ordinary Tropical Diarrhœa. Recovery.

A gentleman, aged 31, had been in China for twelve years; suffered for the last two years from diarrhœa. Returned to England in September 1883. On 13th December, when he came under observation, the tongue was smooth; motions frequent and light-coloured, little pain; much flatulence, eyes yellow, skin flaccid. Put on milk diet and saline laxatives in the morning, to relieve portal congestion. He improved at once, but continued the milk diet, with occasional biscuits, for five weeks, in which time he gained 5 lbs., and the diarrhœa all but disappeared. The diet was then modified; but towards the end of February 1884 there was a slight return of diarrhœa, and the milk diet was resumed. After this he gradually returned to normal diet, and ultimately recovered.

No. 6.—Case of Tropical Diarrhœa, following Hepatic Congestion, and complicated with Dysentery. Recovery.

An officer, aged 28½, went to India first in January 1876. Kept well till 1878, when he came home on account of hepatic congestion, etc. Went out again in January 1880 to Bangalore, and remained there till March 1881, when he went to South Africa. Got back to India in February 1882. Had several short attacks of diarrhœa, the last at Christmas 1883, from which he never quite recovered, and for which he was sent home in February 1884, and arrived in England in May. Diarrhœa was dysenteric, a little blood with the motions, five or six a day; very pallid, lost weight. On 14th May put on milk diet and saline laxatives for a few days, to relieve portal congestion. By 9th June he was much better, and had gained a great deal in weight. Diet varied with bread, lightly-boiled eggs, rice pudding, bacon. Papillæ of tongue well-developed; one motion a day; felt much stronger. Weight, which had fallen to 7 st. 10 lbs., rose to 8 st. 8 lbs., almost the normal.

He also recovered.

No. 7.—Case of Tropical Diarrhœa, following Fever. Recovery. Death long afterwards from Pneumonia.

An officer, aged 43, had resided in India and Burmah for several years. He had suffered from malarial fever, and subsequent relapses; became debilitated, and lost flesh. After a severe recurrence of fever in August, during which he suffered from gastralgia and hæmatemesis, diarrhœa set in. He continued to lose flesh; the tongue became sore, and affected together with the mouth with aphthous ulceration. During the ensuing months he suffered from continual diarrhœa, with frequently recurring attacks of eczema. In March of the following year, 1883, he was sent home in an extreme state of emaciation, the diarrhœa still continuing, there being four or five motions of a light colour daily. On his way home was detained at Malta by weakness and fever, pronounced to be typhoid, and so-called atrophy of the liver. Arrived in England in the end of May 1883.

He had taken a variety of drugs and nourishment, but had not improved. Diet had been chiefly milk and lime water, farinaceous substances, light puddings, etc. Bismuth and the liq. ferri pernitrate had been freely administered. Until January 1884 this method of treatment was continued, many variations being made in his food, but all simple, and supposed to be of an unirritating character. No further improvement took place; the white diarrhœa, with the soreness and glazed condition of the tongue, continued. He was then extremely emaciated, lips and gums white and bloodless, skin dry and harsh; temperature, 96°; tongue red, and tender at tip and edges; voice reduced to a whisper; feet and ankles œdematous; traces of albumen in urine. Discharges of four to six copious, light-coloured stools daily; a bedsore as large as a 5s. piece on the sacral region; pulse feeble, exhaustion great, weight so much reduced that he was easily lifted in the nurse's arms from his couch to his bed.

On 29th January cow's milk was prescribed in quantities of one to two tablespoonfuls every half-hour, and nothing else of any description.

January 30th.—Bowels moved twice in the twenty-four hours, but not watery.

31*st.*—No action of bowels.

February 1*st*.—Bowels acted once; motion large, white, and pultaceous.

2*nd*.—Bowels acted once. Two ounces of milk every half-hour; it agrees well.

5*th*.—A little tea given with the milk.

13*th*.—Milk increased to 4 oz. every hour. No recurrence of watery motion up to this date. Bowels acted twice in twenty-four hours.

23*rd*.—Same treatment continued. Conditions of patient greatly improved; bedsore healed. Weight, 6 st. Temperature, 96°.

March 1*st*.—Temperature, 97°·4; weight, 6 st. 2 lbs. One motion in twenty-four hours.

8*th*.—Temperature, 99°; weight, 6 st. 3 lbs. One action in twenty-four hours. One ounce of stale bread, pulped with milk, added.

11*th*.—Bread increased to 1½ oz. three times daily. A little bread with butter added.

15*th*.—Continued improvement. Temperature, 98°·6; weight, 6 st. 6 lbs. 8 oz. Milk increased to 6 oz. every hour.

29*th*.—A further change of diet was made; an over-boiled egg was taken, but it disagreed; diarrhœa began to return. A small dose of oil given to expel the peccant matter. Returned to pure milk diet.

April 23*rd*.—The evil effects of the egg did not pass off for some days, portions of undigested egg continuing to appear in the motions, accompanied with mucus. His weight fell again to 6 st. 4 lbs. 12 oz. Temperature, 98°·3. Kept on pure milk diet.

May 3*rd*.—Bowels now acting once in twenty-four hours, healthy in character, but light-coloured; milk continued every hour, with the addition of a little cream; improving steadily, tongue better.

10*th*.—Allowed to go out in a bath chair. Weight, 6 st. 8 lbs.

31*st*.—Continued to improve; no diarrhœa. Progress was again slightly retarded by a chill caught when out, and from eating biscuit; but he soon regained lost ground. His health and spirits improved, and his weight increasing. The only medicine was an occasional dose of 5 drops of tinct. opii. He steadily improved, and at the end of June was resuming an ordinary plain diet, which he was digesting and assimilating satisfactorily. He ultimately recovered, though not without an occasional slight relapse, owing to indiscretions of diet or exposure to fatigue or chill.

The action of the bowels became normal; he regained his weight and strength, and resumed his ordinary life in this country. Long after his recovery from chronic diarrhœa, he was attacked by pneumonia, the result of exposure to cold at night, to which he succumbed.

This was a typical and very severe case of this disease. It had long resisted drugs and carefully regulated though varied diet, but yielded to milk diet and perfect rest. The mischief apparently had been repaired and normal health restored when the attack of pneumonia, the result of most incautious exposure out of doors during a cold night, brought his life to a close.

This case has been given in some detail, as it is a typical one.

No. 8.—Case of ordinary Tropical Diarrhœa. Recovery. Death some Years later from another Disease.

A gentleman, aged 49, who had been in India twenty-one years, had had diarrhœa off and on for nine years. Last attack came on in September 1884. By 27th January 1885, when he came under observation, he had lost

2 st. weight. Tongue red, shrunken, and smooth, tender to salt, pepper, or wine. No pain; motions frequent, frothy, whitish. He was put on a milk diet, 4 oz. every hour; to take moderate exercise and keep warm. Up to 17th March had taken nothing but milk, 4½ quarts daily. One motion in the day; hard, white, or faintly yellow. Had not gained in weight. Took long walks. Tongue less cracked, becoming quite normal. Better in every way. Head clear; took no stimulants; great craving for food occasionally. He ultimately perfectly recovered, but died seven or eight years later from an affection of the throat, quite independent of the former disease.

No. 9.—Case of ordinary Tropical Diarrhœa. Recovery.

A lady, aged 35, who had been in India for two years, and had had three children, had diarrhœa one month after arrival. Had hæmorrhage from the bowels also and fever. Had been home for three months. On 6th February 1885, when she came under observation, there were four white, frothy motions daily; flatulence; tongue smooth and red; lost 2½ st. weight; weak and nervous (catamenia disappeared for three months but ultimately returned). Put on milk diet, beginning with 3 quarts a day, 4 oz. every hour. She improved; the diarrhœa decreased; tongue became less red and tender, and papillæ began to reappear. At the end of February took a chill after a warm bath, which threw her back for a little while. Shortly afterwards biscuit added to the diet, and at the beginning of March bread and butter. The bowels were still acting twice a day, and were relaxed and white. Minced mutton was taken for two days, but it did not agree, and diarrhœa returned, and tongue again became red and tender. Returned to milk and biscuits. On this diet, gradually varied, she ultimately did well.

No. 10.—Case of obstinate Tropical Diarrhœa supervening some time after Return to England in a Man of advanced Age. Ultimate Recovery.

A retired officer between 50 and 60, who had served many years in India and returned a few years previously, had been suffering for a long time from diarrhœa. He was much wasted and reduced in health. Had been put on milk diet, but not in sufficiently small quantities, nor at short enough intervals, and not exclusively. On 3rd March 1885 was restricted to milk alone every half-hour. He took 3 to 3½ quarts a day, and by 31st April had improved much; tongue no longer red, papillæ well developed; gaining flesh, and looking much better. Allowed to add a little to diet. On the 27th April he was better, but still had some diarrhœa. Returned to the milk, as all else disagreed with him. Afterwards returned to varied diet, regained his health, and years afterwards was quite well.

No. 11.—Case of ordinary Tropical Diarrhœa, complicated with Hæmorrhoids. Recovery.

A gentleman, aged 47, had been in Singapore, China, and Batavia since 1864. In 1883, up to which time he had been quite well, diarrhœa came on. Came home in August 1883, returned to Singapore in January 1885. Diarrhœa returned immediately, and he was sent home again. Tongue was red; had bleeding piles, aphthæ in the mouth. Was ordered milk every half-hour, and salines to relieve portal congestion for two or three days, at intervals, on 24th October 1885. On the 28th he was better; on the 5th

November the tongue was much better, and there was one white or yellowish motion a day. Continued with nothing but milk for five weeks. By the end of December the diarrhœa had ceased; was taking corn-flour, and potass. tartrate of iron, 5 grains twice a day. He ultimately recovered.

No. 12.—Case of Tropical Diarrhœa in a Person over 60 some Years after Return to England. Recovery.

An English gentleman, aged 63, who had spent many years in India, but had been a considerable number at home, had suffered from malarious fever from time to time, and since his return to England, some eight or ten years ago, from gout, and from functional derangement of the liver, for which he had been to Carlsbad on more than one occasion. He was attacked slowly and insidiously by diarrhœa, which in a few weeks assumed all the aspects of exaggerated tropical diarrhœa — profuse and frequent white motions, without pain, rapid wasting, tongue became red, smooth, and tender. He was placed upon absolute milk diet of 4 increasing to 6 oz. every hour, with rest, care as to warmth, and avoiding fatigue and chill. Within six weeks the diarrhœa had entirely ceased; the motions were becoming natural, and he was gradually resuming a varied and simple diet. Before the end of the third month he was able to return to a normal diet, had gained 20 lbs. in weight, with perfect feeling of health, and was able to proceed on a long sea voyage on business in July 1892.

No. 13.—Case of Tropical Diarrhœa. Death from Exhaustion. (Dr. McConnell.)

A European, aged 62, was admitted to the Medical College Hospital, Calcutta, on 4th April 1892, having been suffering from dyspepsia and diarrhœa for five weeks. Very emaciated, anæmic, and sallow; skin shrivelled-looking; feet œdematous; tongue raw, red, and fissured, with a few small aphthous ulcers on the dorsum and sides and the mucous membranes of the cheeks; pulse small and feeble. Six to eight or more stools in the twenty-four hours; copious, frothy, very offensive, whitish and pultaceous; inclined to be moved soon after any food is taken into the stomach.

Treated with pil. hydrarg. and a carminative mixture. Diet, milk and lime water, a little corn-flour, and a pint of chicken broth.

After three or four days the motions diminished in frequency, but the colour remained the same.

The pil. hydrarg. and carminative mixture was then replaced by a mixture of lig. hydrarg. perchlor., tr. camph. co., spir. chloroform, and under this treatment the stools were reduced to one or two only, invariably passed in the early morning. They became darker in colour and more consistent. An extra half-pint of milk and a small quantity of liquor carnis added to the diet.

Improvement continued for a fortnight, when there was a relapse, motions becoming very thin though slightly coloured. Relief obtained by tincture of coto in 3 grain doses.

By 24th May patient was able to walk about the ward, digest plainly cooked chicken, two raw eggs, and 2 pints of milk. Stools one in twenty-four hours, semi-consistent, and muddy coloured.

Had a relapse at the beginning of June, the old characters of stools returning, mouth becoming sore and tongue dry. Diet again reduced to

milk and lime water and chicken broth, and recourse had to perchloride of mercury mixture.

There was a temporary improvement, but on 18th June he became very low, small serous like evacuations were passed constantly, pulse weak, extremities cold, tongue glazed, leathery, and dry; continued in this state for four days and then died, thoroughly worn out and exhausted.

No autopsy obtained.

The foregoing cases are merely abstracts; details have been omitted, salient and important points alone noticed. They were all typical cases of the disease, and the results show how amenable it may be to dietetic treatment when persistently adhered to. It is not to be assumed that all cases end so favourably; sometimes, after repeated ameliorations and recurrences, the patient has finally sunk from exhaustion; but in such, neglect of precaution, errors or indiscretions of diet, old age, or complications with other disease, have been concerned in producing the fatal termination.

CHAPTER XVI.

DYSENTERY.

BY SURGEON-CAPTAIN D. M. DAVIDSON, M.B., C.M., INDIAN MEDICAL SERVICE, AND ANDREW DAVIDSON, M.D., F.R.C.P.Ed.

Synomyms.—Gr. *Δυσεντερία*; L. Dysenteria, tormina; Fr. Dysenterie; Ger. Die rothe Ruhr; E. The bloody flux, dysentery.

Definition.—Symptomatically, dysentery may be defined as a disease characterised by frequent bloody, mucous, serous, or ichorous stools, generally with retention of the fæces, accompanied with tormina and tenesmus, and, in some cases, with more or less febrile disturbance.

Pathologically, it may be considered as a specific inflammation of the internal coats of the large intestine, occasionally extending to the lower part of the ileum, having a tendency to terminate in ulceration, suppuration, or gangrene of the affected tissues.

The term "dysentery" probably includes more than one disease. It may be sthenic or asthenic in type; sporadic, endemic, or epidemic in its manifestations; acute or chronic in its course, and may, under certain circumstances, spread amongst those associated with the sick.

History.—Although no connected account of the symptoms, seat, and lesions of dysentery is to be met with in the genuine Hippocratic writings, the disease is frequently mentioned, and in such a way as to show that its principal characters had already been clearly recognised. The prognostic significance of the different kinds of stool, of the presence or absence of fever, and of liver complications in dysentery is pointed out, and strangury, not connected with kidney disease, is mentioned as a symptom of the malady.

Hippocrates ascribes dysenteries, diarrhœas, and protracted quartans to the use of marshy, stagnant, and lake waters,[1] and also to meteorological conditions.

Aretæus gives a graphic account of the symptoms of dysentery;

[1] Airs, Waters, and Places, par. vii.

and his description of the lesions met with in fatal cases of the disease is at once so vivid and minute that, as Heubner remarks, "one would think he had made post-mortem examinations." According to Aretæus, dysentery arises from ulceration of the intestines. The ulcers may be only superficial erosions, and then they are comparatively innocuous; or they may be deeper and more dangerous; or, finally, they may be of a phagedænic, painful, spreading, and gangrenous character, leading to a fatal termination. When chronic, the edges of the ulcers are described as thick, rough, unequal, callous, and difficult to heal over, and, when cicatrised, as being apt to open afresh. The causes of dysentery, he says, are manifold—such as indigestion, continued cold, acrid articles of food, injuries, exposure to cold, and cold drinks.

Galen recognises two kinds of dysentery—that resulting from liver disease, or hepatic dysentery, and that arising from ulceration of the mucous membrane of the intestines. He is the first author who mentions famine as a cause of the disease.

The later Greek, Roman, and Arabic writers add little to our knowledge of its nature, causation, or treatment.

Sydenham, from his observations of the epidemics of 1669–72, describes three forms of dysentery. The first form, which he speaks of as "gripes without stools," or "dry gripes," is sometimes attended with fever, sometimes not. In the second variety there are frequent slimy motions, attended with griping and usually with fever, which, he says, is "a true bloody flux or dysentery, although there may be no passage of blood from first to last." In the third and more distinctive form of the disease, along with fever there are frequent bloody and mucous stools, with griping and tenesmus. After maturely weighing the symptoms of the disease, and the circumstances in which it arose, he came to the conclusion that dysentery is a fever—a *febris introversa*. By means of this fever, the hot and acrid humours contained in the blood are deposited in the bowels through the meseraic arteries.[1]

The observation of dysentery in an epidemic form as it occurred in camps and vessels, especially in slave ships, during the eighteenth century, led physicians to regard it as a contagious disease. This doctrine of the contagiousness of dysentery is nowhere to be found in the writings of Sydenham and his contemporaries. Pringle records an instance, to which we shall afterwards refer, in which dysentery appears to have been propagated by contagion or infection; and the opinion that it is a contagious disease found its fullest expression in Cullen's definition of dysentery: "Pyrexia

[1] Sydenham, *Works*, "Medical Observations," chap. iii.

contagiosa: dejectiones frequentes, mucosæ, vel sanguinolentæ, retentis plerumque fæcibus alvinis; tormina, tenesmus." This view of the nature of the malady obtained such a firm hold of the profession at the close of the eighteenth century that the existence of a non-contagious form was scarcely admitted, and the importance of the intestinal lesions was so minimised that the occurrence of ulcers in the large intestines was stated to be very unusual.

These extreme doctrinal views, opposed as they were to the teachings of clinical medicine and morbid anatomy, could not possibly survive a wider study of the disease as met with in tropical countries. Hunter, in 1796, gave a remarkably good account of dysentery as observed by him in Jamaica. He did not find it to be infectious in the hospitals of that colony. He looked upon it mainly as a local dssease, caused by the use of impure water, or as a sequel of remittent fever.[1] Annesley, whose faculties of observation and independence of judgment enabled him to profit by a long and extensive Indian experience, recognised the supreme importance of the intestinal lesions, both in the simple form of the disease and in that accompanied by marked hepatic disorder. He not only described the symptoms and course of dysentery as it is met with in Europeans and natives in the East, but he gave an accurate and minute description of the various lesions met with in the bowel itself and in the other viscera. While admitting that the disease may become contagious under certain circumstances, he states that he had never himself met with an instance in which it had exhibited a contagious character. Dysentery, according to him, "seldom supervenes as the effect of the operation of a single cause. Very frequently, in addition to the predisposition arising from plethora, fatigue, or a loaded state of the large bowel, and a deranged condition of the alvine secretions, several of the common exciting causes of the disease, such as intoxication, exposure to the night air, wearing wet or damp clothes, insufficient clothing, sleeping on the ground, and unwholesome food, act in conjunction."[2] When dysentery follows hepatic disease, he believed that it arose from a morbid secretion of bile. Dysentery is thus, according to him, a non-specific inflammation of the large intestine, seldom contagious; and, when fever exists, it is not primary, but symptomatic of the inflammatory process. These were the views held by the most distinguished predecessors and successors of Annesley in India from the second decade of this century down to comparatively recent times.

[1] Hunter, *Observations on the Diseases of the Army in Jamaica.* London, 1796.
[2] *Diseases of India.* London, 1841.

Later investigations have been chiefly directed to the histological changes consequent on the dysenteric process, and to the nature of the disease itself. Virchow distinguished two forms of dysentery—the catarrhal or sero-purulent, and the diphtheritic or fibrinous. The latter, which, according to him, is by far the more serious form, is characterised by an exudation of amorphous fibrin into the interstices of the tissues of the mucous and sub-mucous coats, leading to compression of the vessels, and necrosis of the infiltrated tissue. Parkes and Aitken considered the primary lesion of dysentery to consist in an inflammation of the solitary lenticular follicles of the large intestine, giving rise, first, to intumescence, and, finally, to ulceration and destruction of the gland tissue, and to softening and sloughing of the tubular follicles; extending, in grave cases, to suppuration or sloughing of the sub-mucous connective tissue, or even of the muscular and serous coats of the bowel.

Among the later writers whose researches have materially contributed to advance our knowledge of this disease, we may mention Kelsch and Kiener,[1] who refer the intestinal lesions to two primary types—the ulcerous and gangrenous; the former characterised by a dry eschar, and usually running a mild course; the latter, by a humid eschar, and much more dangerous.

We have, finally, to notice a very important monograph by Councilman and Lafleur,[2] describing a special form of dysentery which they have found to be associated with the presence in the intestinal ulcers and stools of the *Amœba coli*, and which they believe is caused by this parasite. It is interesting to remark that the *Amœba coli* has been found in dysenteric ulcers in Russia, Austria, Egypt, India, Central America, and the United States.

Etiology.

Distribution.—Instead of giving in detail the geographical distribution of dysentery, which will be found in special works devoted to the subject,[3] we think it will be more useful to present, as briefly as possible, some of the more important conclusions which may be deduced from a study of its distribution.

1. *Dysentery is an ubiquitous disease.*—There is no country, and no extensive district in any country, within or without the tropics, in which dysentery is not to be met with as a sporadic, endemic, or

[1] *Traité des Maladies des Pays Chauds.* Paris, 1889.
[2] *Johns Hopkins Hospital Reports.* Baltimore, 1891.
[3] Hirsch's *Handbook of Geographical and Historical Pathology*, Sydenham Society, Lond. 1885; Davidson's *Geographical Pathology*, Edin. 1892.

epidemic disease. In a sporadic form, dysentery occurs in the coldest inhabited regions, such as Iceland and Greenland, and in all temperate climates. In many temperate regions in Europe, America, and Africa it occurs both sporadically and also from time to time in more or less extensive epidemies. As an endemic disease it can scarcely be said to prevail beyond 40° N., either in the Eastern or Western Hemisphere. Its southern endemic limit in Africa may be placed at the 30th, and in America at the 35th parallel.

2. *Dysentery increases in frequency, although not constantly nor uniformly, as we approach the equator.*—This statement, so qualified, finds abundant support in the facts relating to the distribution of the disease. Dysentery is more fatal in France than in England. The dysenteric death-rate of the troops stationed in England (1886–88) was 0·04 per 1000; that of the French army (1872–80) was 0·22 per 1000. Dysentery, again, is distinctly more fatal in the south than in the north of France. According to Lombard, the ratio of deaths from dysentery to the total mortality in the north is 9·2, while in the south it rises to 27·3 per 1000.[1] The same relation is observed in Italy, as will be seen by comparing the death-rate from dysentery per 10,000 living in four northern with that in four southern departments:—

Four Northern Departments.		Four Southern Departments.	
Piedmont, . .	0·87	Basilicata, . .	8·03
Liguria, . .	0·50	Calabria, . .	5·03
Lombardy, . .	0·57	Sicily, . .	6·03
Venice, . .	0·97	Sardinia, . .	1·23

We have seen that the dysenteric death-rate of the French army (1872–80) was 0·22; that of the army of Algeria, with its warmer climate, was, for the same period, 0·62 per 1000. In Egypt, situated still further south, the ratio of deaths from this disease in the English army, for the five years ending 1888, was 2·24 per 1000. In 1889, a year of peace, the ratio fell to 1·46. In Senegal, about 13° N., the deaths from dysentery form, according to Dutroulau, more than one-third of the total mortality.

Hitherto we have noticed a gradual though not uniform increase in the fatality of the disease according to latitude; but, when we leave Senegal, dysentery instead of becoming more frequent as we advance south, appears to diminish alike in frequency and severity. Dr. Prout has remarked on the comparative mildness of the disease as he observed it on the Gold Coast, and the testimony of the French surgeons on the Gaboon is entirely to the same effect. Yet it should not be forgotten that dysentery has been one of the most fatal

[1] *Traité de Climatologie Médicale*, tome ii. p. 510.

diseases among our soldiers when on active service in this region, as it has been among the French in Dahomey, and it will certainly have to be counted on in any future expedition to the West Coast.[1]

The same gradual though irregular increase in the prevalence and severity of dysentery from north to south is observed in the United States, as will be seen from the following figures :—

TABLE SHOWING THE RATIO OF ADMISSIONS PER 1000 OF THE TROOPS FOR ACUTE DYSENTERY, AND THE PROPORTION OF DEATHS TO CASES ALONG THE ATLANTIC SEABOARD AND IN THE INTERIOR FROM NORTH TO SOUTH.

Atlantic Coast from North to South.

Region.	Ratio of Cases per 1000 of Mean Strength.	Proportion of Deaths to Cases.
North Atlantic,	67·0	1 in 306
New York Harbour,	77·0	1 in 62
Middle Atlantic,	94·0	1 in 158
South Atlantic,	101·0	0 in 346
Atlantic Coast of Florida,	171·0	1 in 104

Interior from North to South.

Region.	Ratio of Cases per 1000 of Mean Strength.	Proportion of Deaths to Cases.
Region West of Lakes,	67·0	1 in 196
Middle Interior Region East (Newport Barracks),	51·7	1 in 32
Middle Interior Region West (Jefferson Barracks, St. Louis Arsenal),	196·0	1 in 38
South Interior Region East (Forts Jessup, Pike, Wood, etc.),	166·0	1 in 72
Texas,	138·0	1 in 84

It may be remarked that the climate of the Middle Interior Region West, represented by Jefferson Barracks and St. Louis Arsenal, where dysentery is so severe, is marked by a high annual range compared with that of the Atlantic Coast and also with that of the South Interior Region East; but it is none the less evident that

[1] *Army Medical Report*, 1864, p. 82.

while a high temperature favours the development of the disease, other conditions go to determine its prevalence.

Dysentery takes a leading place in the pathology of India. As the returns for the native population include dysentery along with diarrhœa and other bowel complaints, we have no data very satisfactory for determining the mortality which it causes among the natives of India, nor its relative prevalence in the various provinces. The scanty knowledge we possess on these points is derived from the statistics of the Native and European Armies. Bryden gives the death-rate of the Native Army of Bengal for the period 1867–76 from diarrhœa and dysentery combined at 2·01 per 1000; that of the Indo-European Army (1870–79) at 1·60 per 1000. In 1878, when the combined death-rate of the Native Army was 2·58 per 1000, that from dysentery was 1·63, and from diarrhœa 0·95. In the same year the death-rates from dysentery and diarrhœa in the Indo-European Army were 1·73 and 0·7 per 1000 respectively. From this it would appear that dysentery is somewhat more fatal to the Europeans than to the natives. If we consider that dysentery gives rise to a death-rate of about 1·50 per 1000 in the Native Indian Army, living under comparatively favourable sanitary conditions, we may be quite sure that it is exceedingly fatal among the general population.

The incidence of the disease on the three Presidencies as judged by the admission and death rates of the European troops is as follows:—

	Bengal.		Madras.		Bombay.	
	Admission-rate.	Death-rate.	Admission-rate.	Death-rate.	Admission-rate.	Death-rate.
1870-79,	33·1	1·37	76·9	2·32	27·6	0·98

Madras always shows a high admission and death-rate from dysentery compared with the other Presidencies; but this is not entirely, perhaps it is not chiefly, due to a more tropical climate, for there are some parts of the south of India, such as the plains of Trichinopoly and Malliaporam, where dysentery is less severe than in regions fifteen degrees further north.

Other things being equal, temperature has, however, a marked influence on the prevalence of the disease in India as elsewhere. This will be seen from the decrease in the percentage of deaths from dysentery to the total mortality as we advance to the north-west from the hot plains of Bengal to the comparatively cooler region of the Punjab frontier. The figures, which are from Bryden, refer to the Native Army (1867–76):—

Bengal.	Gangetic Provinces.	Meerut and Rohilkund.	Punjab.	Punjab Frontier.
25·3	16·9	12·0	14·6	9·4

It has to be remarked in this connection that Singapore, upon the equator, with its hot, moist, and equable climate, is much less subject to dysentery than India. Another point of interest brought out by a study of the distribution of dysentery is this, that districts situated in the same geographical region, and so close to each other as to be practically under the same climatic conditions, suffer very unequally. A single instance of this relation will suffice. On the island of Goree, close to Senegal, dysentery is much less frequent than on the immediately adjoining mainland.

3. *The geographical distribution of dysentery does not correspond with that of malaria.*—Although it is quite common to find malaria and dysentery endemic in the same region and in the same locality, this is by no means constantly the case. Indeed, in some instances it would almost appear as if there were an antagonism, as regards locality, between these two diseases. Grande Terre, in the island of Guadeloupe, for example, is very malarious, but is almost free from dysentery; while Basse Terre, in the same island, is only slightly malarious, but is severely affected with dysentery. We have noticed that Madras is the Presidency in India that suffers most from dysentery,—it is also the one that suffers least from malaria. In the same way, in certain localities in India, such as Deesa, where malarial fever is very prevalent, dysentery is of rare occurrence; while in other districts, such as Belgaum, dysentery is severe and malaria mild. But the most convincing evidence of the independence of dysentery on the malarial miasm, is found in the fact that dysentery was a very common and fatal malady in Mauritius before the appearance of fever in that island. We may point, as an example equally convincing, to the island of Rodrigues, which is free from malaria, but where dysentery accounts for 29·6 per cent. of the total mortality. On the other hand, nothing is more common than an outbreak of dysentery in bodies of men who have been reduced by repeated attacks of malaria. The malarial cachexia undoubtedly predisposes to dysentery, or gives rise to a particular form of it, which is not always amenable to ipecacuanha. This relation between malaria and dysentery is illustrated by the following table, giving the number of deaths from dysentery and malarial fevers in the Sealkote district of the Punjab (1889–91):—

Diseases.	1888.	1889.	1890.	1891.
Fevers, ...	17,915	21,069	84,963	28,418
Dysentery, . . .	989	1,155	2,151	874

4. *Dysentery is not always more prevalent on the warm coast-lands of tropical countries than at higher elevations.*—In many tropical countries dysentery appears to be even more prevalent at moderate elevations than along the coast-line. This is stated to be the case in India, in Java, in Jamaica, in Cayenne, in some parts of South America, and in Mexico, although in regard to the last Jourdanet remarks that when we reach the higher levels of the central plateau, at an elevation of 7000 to 8000 feet, the disease again diminishes in prevalence. Béranger-Féraud relates that in 1840 the troops in Martinique were removed to a camp at an elevation of 1200 mètres in order to escape yellow fever, then raging. The post had to be abandoned in 1844, as it was found that the dysentery of the hills was equally fatal as the yellow fever of the plains.[1] It often happens that patients who have escaped dysentery while living in the equable, warm climates of a tropical coast or inland plain, are attacked with the disease for the first time on removing to a hill station. The risk of being attacked by dysentery in these circumstances is greatest in cachectic subjects.

5. *No soil is exempt from dysentery.*—It cannot perhaps be said that the physical condition of the soil is quite immaterial in respect to the endemic disease; but it is perfectly certain that it is met with, and in a severe form, both in very marshy and in very dry localities. If dysentery is common in many of the jungly districts of India, it is by no means rare on the bare burning rocks and sands of Aden. This point, however, demands a more careful study than it has hitherto received.

Limited epidemies of dysentery seem to have a preference for humid and waterlogged soils. We shall presently refer to outbreaks arising from the drying up of marshes or ponds. But in its larger epidemic extensions the physical characters of the soil appear to have absolutely no influence on its spread. To this it may be added, that the geological constitution of the soil has no appreciable influence on the disease, either in its endemic or epidemic forms.

Having considered some of the more important inferences to be derived from a study of the geographical distribution of dysentery, we shall now deal with other conditions which influence its prevalence.

The Influence of Season.—Dysentery in all its forms is notably a seasonal disease. Out of 705 accurately recorded epidemies collected by Hirsch, 529 raged in summer, or in summer

[1] Béranger-Féraud, *Traité Théorique et Clinique de la Dysenterie.* Paris, 1883.

and autumn; 137 in autumn, or in autumn and winter; 14 in winter; and 25 in spring, or in spring and summer.[1]

As an endemic malady, dysentery in Europe attains its maximum in summer and early autumn. Thus, in Italy, the largest number of dysenteric deaths are registered in the three months July, August, and September, the actual maximum falling on August, that is, on the warmest month of the year, and before the heavy rains have fallen. Summer is also the season when dysentery is most prevalent in the United States, although in the extreme south of the Union it tends to become an autumnal malady.

Within the tropics, dysentery is usually most fatal in the third and fourth quarters. The following table gives, according to Dutroulau, the quarterly distribution of 100 admissions and deaths in the three inter-tropical colonies—Senegal, Martinique, and Cayenne:—

Quarters.	Senegal.		Martinique.		Cayenne.	
	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.
Jan. to March,	18·34	22·30	28·40	18·76	21·92	24·32
April to June,	15·30	8·80	25·15	20·55	22·25	13·52
July to Sept.,	28·39	29·05	24·20	27·73	28·49	27·03
Oct. to Dec.,	37·97	39·85	22·16	32·96	27·32	35·13

In elucidation of the above table, we may note that in Cayenne the temperature is almost uniform throughout the year. In Senegal and Martinique the third quarter is the warmest. In Cayenne the period of heaviest rainfall is from February to May; the driest months, August to January. In Martinique there are heavy rains from December to February. In Senegal the rainy season extends from July to September; the three months October to December are dry.

It will be observed that although the period of the greatest dysentery prevalence, as judged by the admissions, varies, the highest death-rate uniformly falls on the fourth quarter, which is generally the dry season, when the temperature has already fallen considerably.

Dysentery in India, as a whole, is a disease of the cold season. Morehead[2] has shown that the maximum of admissions into the European Hospital of Bombay falls on the three cold months, November, December, and January; and the smallest number on

[1] *Op. cit.* vol. iii. p. 329.

[2] *Researches on Disease in India*, Lond. 1860, pp. 273, 274.

the hot months—April and May. In Bengal and Bombay the maximum of deaths from dysentery among the European troops occurs in the fourth quarter. In Madras the third quarter is usually that most charged with admissions and deaths. The same rule, so far as we know, holds in respect to the seasonal prevalence of dysentery among the natives. Amongst them, as amongst the Europeans, dysentery is most fatal in late autumn or winter. In Bengal it is distinctly a cold weather disease, as will be seen by the following figures giving the monthly distribution of 100 deaths in the Native Army of Bengal:—

Jan.	Feb.	March.	April.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
11·9	7·9	8·3	4·1	4·4	5·2	6·7	6·7	6·3	10·0	12·3	15·2

Dutroulau says that everywhere it is the warm season, and the moment of that season in which evaporation is taking place from the soil, that dysentery is most destructive. No such simple formula covers the facts. That a high temperature is favourable to the development of dysentery is clear enough, but in tropical countries it is only after the temperature has begun to fall, or even when it has reached its minimum, that dysentery usually attains its maximum fatality.

Vicissitudes of Temperature and Exposure to Cold and Wet.—The greatest unanimity prevails among all observers respecting the influence of vicissitudes of temperature in determining attacks of dysentery. The less we are inclined to admit that exposure to night air, the wearing of wet clothes, sleeping on the damp ground, and other chill-producing agencies are sufficient in themselves, and apart from a specific infection, to give rise to dysentery, the more necessary is it to recognise their vast importance as predisposing and exciting causes of the disease. So important, indeed, are these agencies in the causation of dysentery, that the specific cause, whatever it may be, will, in many instances, fail to give rise to the disease in those who are not exposed to these depressing influences.

The justice of this remark cannot be better illustrated than by quoting the account given by Pringle of the circumstances in which dysentery appeared in the main body of the army at Hanau, while a detachment in the immediate neighbourhood escaped the distemper. This is what he says respecting the causes of the outbreak in the main army:—

> On the 26th (June 1743), in the evening, the tents were struck, the army marched all night, and next morning fought at Dettingen. On the night following the soldiers lay on the field of battle without tents, and exposed

to a heavy rain. Next day we moved to Hanau and encamped on good ground in an open field. But it was wet, and the first night or two the men wanted straw. By these accidents a sudden change was made in the health of the army. For the summer had begun early, and the weather had been constantly warm; but the free and uninterrupted perspiration seemed to prevent any general sickness. Now the pores were suddenly stopped, the body was chilled, and the humours, tending to a resolution from the preceding heat, were turned upon the bowel and produced dysentery, which continued for a considerable part of the campaign. In eight days after the battle about 500 men were seized with that distemper, and in a few weeks nearly half the men were ill or had recovered.

So much for the circumstances in which dysentery appeared in the main body. His account of the condition of the detachment which escaped the distemper is as follows:—

> Three companies of Howard's, which had not joined us, marched with the king's baggage from Ostend to Hanau, where arriving a night or two before the battle, they encamped for the first time about half a mile from the ground that was afterwards occupied by the army. These men had never been exposed to rain nor had lain wet. By this separation from the line they were also removed from the contagion of the poison, and having pitched close upon the river, they had the benefit of a constant stream of fresh air. By means of these favourable circumstances it was remarkable, that while the main body thus suffered, this little camp almost entirely escaped, though the men breathed the same air (the contagious part excepted), used the same victuals, and drank the same water.[1]

The infectious cause was not absent from this small camp, for it is said they *almost entirely escaped;* there can be no doubt that the epidemy in the main army was determined by the exposure of the men to wet, cold, and fatigue.

Impure Water as a cause of Dysentery.—The substitution of a pure for an impure water supply has frequently been followed by a decrease in the prevalence of dysentery in an affected community. The instances in which outbreaks of dysentery have been traced to the use of water contaminated with fæcal impurities are very numerous. We shall simply notice one or two of the most conclusive instances of this nature. Dysentery broke out in 1870 in one of the regiments of the garrison of Metz, while the rest of the troops were almost free from the disease. It was found that the wells supplying this regiment were contaminated with fæcal matters filtering from latrines placed opposite and close to them. The spread of the disease was speedily arrested when the wells were closed.[2] In 1881 the troops who occupied the same barracks once more made use of the water from the same wells and were attacked with dysentery,

[1] Pringle's *Diseases of the Army.* London, 1768.
[2] Read, *Journ. de méd. mil.* par Dehorne, 1872.

which again disappeared as soon as the wells were closed.[1] Surgeon Oakes traced an outbreak of dysentery at Cape Coast Castle to the pollution of a tank by percolation from a cesspool. The disease disappeared as soon as the cause of the pollution was removed. Fayrer relates that the inmates of the native lunatic asylum at Calcutta were in the habit of drinking water contained in reservoirs placed outside the latrines for the purpose of ablution. The patients suffered much from dysentery, diarrhœa, and lumbrici. On putting a stop to this practice of drinking water, necessarily more or less polluted with fæces, these diseases disappeared. In all of these instances the water which gave rise to dysentery was polluted with fæcal, and possibly with dysenteric discharges. But the disease has also been ascribed to the use of marsh and brackish water,[2] of water contaminated with decaying animal matters, and of waters containing an excess of saline or irritant mineral substances. We may easily understand that water may in one case serve as a vehicle by means of which the specific cause of dysentery is introduced into the system, in another case it may act only as a predisposing cause of the infection by exercising an irritative action on the bowel.

Food.—Food of coarse quality; the use of tainted meat; the too long and exclusive consumption of salted meat, and indulgence in unripe or over-ripe fruit, may be regarded as occasional predisposing or exciting causes of the malady by giving rise to irritation of the intestinal canal; and some of these agencies also act by predisposing the system to the infection by inducing cachexia. The use of alcohol in excess is recognised as a powerful predisposing cause of relapses of the disease. Still greater importance is to be attached to deficient nourishment in the causation of dysentery. Dysentery constantly dogs the footsteps of famine.

Soil contaminated with Fæcal Matters as a cause of Dysentery.—Among the causes which gave rise to the great mortality from dysentery at Secunderabad in former years, pollution of the soil was probably one of the most important. Maclean informs us that the surface and subsoil around the station were thoroughly saturated with organic matter; that the soil in the neighbourhood was furrowed by ravines in which ordure was deposited by the natives; that the privy accommodation was of the worst possible description, and that the barracks were overcrowded and surrounded by a high wall. The outbreak of dysentery in the Cumberland Asylum, described by Clouston, was traced to the emanations of sewage applied to the land at the distance of about 300 yards

[1] Hirsch, *op. cit.* vol. iii. [2] Hunter, *op. cit.* p. 176; and Annesley, *op. cit.* p. 403.

from the ward where the dysentery occurred.[1] Kelsch and Kiener relate that dysentery raged for two successive years at Rochelle, and was almost exclusively restricted to the 21st and 34th Regiments of artillery occupying a locality impregnated daily and for several years by human and animal dejecta spread over the surface. It is also to the progressive infection of the soil by excreta that we are to ascribe the remarkable increase of dysentery in the camp at Châlons. The first year of its establishment (1857), only a few cases of diarrhœa and dysentery occurred, the succeeding year the cases numbered 96, and the number gradually increased until, in 1864, there were 457 cases and 5 deaths.

Numerous instances of the spread of dysentery by means of badly kept privies have been reported; and as these may contain dysenteric stools, the danger of infection can readily be understood. The contents of the latrine in such cases form the soil in which the dysentery "germ" may be supposed to flourish.

Emanations from drying up of Ponds and Lakes.—A number of instances are recorded by Kelsch and Kiener, all of them occurring in France, in which outbreaks of dysentery appear to have been caused by the drying up of lakes and ponds, and the exposure of the slime and mud to the action of the sun. One of these occurred in 1872 in the commune of Creusançay (Haute-Saône), and was attributed to the drying up of mud and detritus left by repeated inundations in the valley in which Creusançay is placed. Another, affecting the garrison of Versailles in 1843, arose from the drying up of stagnant and polluted waters on the estate of Ménagerie (situated to the west of the town) by the great heat of summer. Other instances of a similar nature are reported from Graverand (Cher) in 1873; from Saint-Aignan (Loiret) in 1836; and from Leymen (Haute-Rhin) in 1850, from the "curage et desséchement" of canals and reservoirs. In most of these instances the causal relation is not to be doubted. The disease, as a rule, affected only those who lived near the source of the infection; and it was specially observed, in some instances, to have first appeared in the houses nearest to the infectious focus, and afterwards to have propagated itself to those at a greater distance. It is not surprising to read that when dysentery arose in these circumstances, it was frequently accompanied with cases of malarial fever.[2]

Dysentery is, in some instances, transportable, and spreads from the Sick to the Healthy.—We have already quoted Pringle's account of the outbreak of dysentery in the English army after the battle of Dettingen. He goes on to inform us that

[1] *Med. Times and Gaz.* June 1865. [2] *Op. cit.* p. 101.

about 1500 sick, and of these the greater part ill of dysentery, were sent to the village of Feckenheim, which was employed as an hospital. "By these men the air became so vitiated that not only the rest of the patients, but the apothecaries, nurses, and those employed in the hospital, and the rest of the inhabitants of the place, were infected." Lombard informs us that in 1856–57 dysentery appeared in all those places in Central Russia through which the troops from the Crimea, who were infected with dysentery, passed, without regard to latitude or the configuration of the country.[1] In the winter of 1831–32, dysentery occurred among the Belgian troops in the camp at Diest; the patients, when the camp broke up, propagated the disease in the hospitals, towns, and villages to which they repaired. Space forbids us from citing any of the numerous well-authenticated instances in which the arrival of patients suffering from dysentery into a locality previously healthy has been followed by an outbreak, more or less extensive, of the disease.[2] Maclean insists strongly upon the risk of retaining dysenteric stools in the wards of an hospital; and both he and Fayrer assure us that they have seen dysentery propagated to those treated in the same ward with dysenteric patients by the effluvia of their discharges.

Personal Conditions.—*Age.*—All ages are liable to dysentery, but it is more fatal in children than in adults. In 1878 the dysenteric death-rate of the army of India was 1·73 per 1000, that of the European children was 3·84 per 1000.

Length of Residence in the Tropics.—Troops, during the first two or three years of their sojourn in India, show a somewhat greater liability to the disease than those who have been longer in the country; but after the seventh year the deaths from dysentery begin to increase, and they go on increasing according to the length of service. This will be seen by the following figures from a table prepared by Bryden, showing the ratio of deaths from dysentery to 100 deaths from all causes among soldiers, according to length of service:—

First year.	Second year.	First five years.	Fifth to seventh year.	Above seven years.	Above ten years.
9·6	10·4	9·0	10·1	13·3	13·7

This table proves that there is no acclimatisation for dysentery.

Sex and Race.—The two sexes suffer in nearly the same proportion, and all races are about equally liable, if allowance be made for habits and conditions of life. Its great prevalence among the

[1] *Op. cit.* vol. ii. p. 210.

[2] See Strack, *Tentamen medicum de dysenteria*, Moguntiæ, 1760; and Degner, *De Dysenteria*, pp. 4 and after.

coloured races is dependent on their coarse food, the impure water they often drink, and their exposure to wet, cold, and other debilitating influences.

Rural and Urban Life.—Dysentery, like malaria, and unlike diarrhœa and cholera infantum, is specially a disease of rural districts and small villages. Those who follow agricultural pursuits are more liable to contract dysentery than those following in-door occupations. Damp habitations and underground dwellings are found to favour its occurrence.

Epidemic Dysentery.—We have already referred to the occurrence of local outbreaks of dysentery as the result of certain definite causes, and restricted more or less to the localities within which the agencies which gave rise to them were operative.

Widely different from these localised outbreaks is true epidemic dysentery, a form of the disease which has from time to time devastated extensive regions. Sometimes, as in the European outbreak of 1719, these epidemies invade the greater part of a continent in a single year, appearing simultaneously at points far apart. At other times, as in the epidemy that overran Sweden in the years 1852–56, the disease at first appears in a few isolated districts, within a limited area, and gradually advances over the country during successive years, declining during winter and breaking out afresh in the following summer, until it covers the whole, or the greater part, of a region as large as Sweden.

These epidemics have been frequently associated at their commencement with some notable peculiarity in the weather, such as excessive heat and prolonged drought; but once set agoing, they may continue to spread independently of these conditions. The epidemies resulting from famine are not to be confounded with those to which we now refer.

Nature of the Disease.—We have now to ask ourselves, What light is thrown on the nature of dysentery by the facts which we have passed in review? Is dysentery, in all its forms and manifestations, the result of a single morbific cause, or are there more diseases than one included under the name? Should the existence of an amœbic form of dysentery be established,—and the evidence in favour of it is strong, if not conclusive,—we shall have to recognise two etiologically and clinically distinct forms of dysentery, and other varieties may hereafter be discriminated, although we have no reason for supposing that many etiologically distinct forms exist.

Different micro-organism parasites have from time to time been hastily identified as the cause of dysentery, and the claims made on

their behalf speedily abandoned. Recently Ogata[1] has found in the stools of patients suffering from an epidemic form of dysentery in Japan a short bacillus,—a quarter of the length of the tubercle bacillus,—which in pure cultures appears to have given rise to dysenteric symptoms in animals when introduced under the skin or absorbed by the buccal or intestinal surface. Whether these results will be confirmed remains to be seen, but we may rest assured that in epidemic and endemic dysentery we have to do with a parasitic disease.

Much of what we call dysentery is infectious in its nature. This is clearly the case in respect to the epidemic forms of the malady. In some instances it appears to be contagious, but a closer examination of its mode of propagation in hospitals may show that the dysenteric discharges only become infective after they have undergone some changes outside the body, as is conjectured to be the case in miasmatic-contagious diseases. Nor can we admit that the tropical disease is frequently propagated by anything resembling contagion. Instances of this kind are, upon the whole, exceptional, although the fact that they do occur is incontestable, and should never be lost sight of. We see no proof that dysentery in any of its forms can arise solely from the operation of meteorological agencies, or from the co-operation of these with a disordered state of the bodily functions.

The intestinal canal in a healthy state appears to be comparatively immune to most pathogenic organisms, and to those of dysentery among others. It is highly probable that the specific cause (or causes) of dysentery is often introduced into the system without giving rise to the disease. The healthy mucous membrane is not a soil favourable for its growth. It is when the nutrition of the intestine is impaired that it becomes vulnerable to the attacks of low organisms. A high temperature, vicissitudes of temperature, exposure to cold, alcoholic excesses, food of bad quality, and impure water, all tend to give rise to congestion and irritation of the bowel, and thus prepare it for the attack of the specific agent. In the same way functional disorders of the liver may be supposed to act; for in these the intestine is generally congested and its secretions vitiated. Cachectic conditions, whether arising from deficiency of food, as in famine; from the absence from the diet of some constituent essential to health, as in scurvy, or from the action of other debilitating diseases, such as malaria, are accompanied with serious impairment of the nutrition of the bowel, and thus expose it to the inroads of this disease. The etiological link of connection

[1] *Centralbl. für Baktr.* March 9, 1892.

between all these diverse factors is to be sought for in their influence in disturbing the nutrition of the large intestine.

Morbid Anatomy.

Acute Dysentery.—*Macroscopic Appearances.*—On opening the abdomen the peritoneal coat of the large intestine may be found healthy; it may be injected, or dull and cloudy from œdema; it may be coated with lymph, or it may present dark or black discoloured patches, corresponding with ulcerations within the bowel. Occasionally perforation will be found to have taken place with more or less fæcal extravasation and general peritonitis. This accident, however, is rare, and when it occurs, the sigmoid flexure and the rectum will generally be found to be the seats of the perforating ulcer.

When the disease has been of the gangrenous form, the colon is frequently found to be distended, in other forms its calibre may be natural or contracted. Whether distended or contracted, its sacculated structure is usually less marked than in health. Upon grasping the colon, as Annesley remarks, and running the fingers along it, "a different feeling is communicated to the touch in distinct parts of it; at one place it is thickened and doughy, in another, thin and membranous." In exceptional cases it is hard and rigid to the touch.

On opening the bowel, the extent of the disease will become apparent. In many of the fatal cases occurring in tropical countries, the whole of the bowel from the cæcum to the anus will be found to be involved, the disease stopping abruptly at the ileo-cæcal valve. Frequently enough the dysenteric process involves the lower part of the ileum. Bleeker states that he once observed the disease to extend to the duodenum;[1] but cases in which the upper part of the intestinal tract is involved are quite exceptional, for the morbid action is usually limited to the large intestine, and in many instances it is restricted to certain portions of it. When dysentery is associated with malarial fever the stomach and small intestines are often found congested in patches of a pink-red colour. The points of departure for the process appears in most cases to be the cæcum or the rectum. Béranger-Féraud observes that in the Antilles the disease generally begins in the cæcum and ascending colon, whereas in Cochin-China it is more common for it to commence in the rectum or sigmoid flexure.[2] It is rare that dysentery is localised in the ascending or in the transverse colon without the cæcum or the lower extremity of the bowel (according as it begins in the upper

[1] *Indian Annals*, Oct. 1853. [2] *Op. cit.* p. 105

or lower segment) being simultaneously affected. When the cæcum is extensively disorganised it is sometimes distended with a sanguineous, putrid fluid. This condition was very frequently met with in the autopsies of the soldiers who died of dysentery, complicated with malarial fever, during the Ashanti Expedition of 1874. A fluid of the same kind will often be found in the colon in gangrenous cases.

The diseased surface of the mucous membrane presents an irregular appearance. It is thrown into transverse folds, and is often covered with small, firm elevations and depressions. Larger round or oval elevations, soft to the touch, and giving issue to sanious pus on incision, are occasionally met with.

The colour of the affected portions of the mucous membrane is exceedingly varied. If the submucosa is œdematous at certain points, the mucous membrane covering these places is elevated and of a pale colour. Otherwise, it is of a vivid or dark red, of a grey, green, dark, or black colour. These and all intermediate shades, and in varying combinations, occurring in small points, streaks, or diffuse patches, will often be found in different parts of the same bowel. Portions of the mucous membrane, especially those covering the transverse folds, frequently present a bran-like coating. Hæmorrhagic extravasations and, when the disease has been of some weeks' standing, small pigmented patches are also met with.

The diseased membrane is altered in consistence; it is often thickened and doughy. It is easily abraded or lacerated. The closed follicles, at an early stage, are observed to be enlarged, prominent, and surrounded by hyperæmic zones. The agminated glands of the ileum, near the ileo-cæcal valve, are occasionally enlarged.

The membrane so altered in colour and consistence is uniformly the seat of ulceration, gangrene, or exudation.

The ulcers met with in dysentery may be few, and limited to one or more portions of the bowel; or they may be numerous, in which case they are found either disseminated irregularly over the mucous membrane or arranged in groups at certain places. Sometimes they are so small as to be detected only by a careful examination; or large portions of the intestine may be denuded of its internal coats, leaving, here and there, islets of mucous membrane, which have escaped in the general destruction, projecting from the ulcerated surface. In form the ulcers are circular, transverse, irregular, or sinuous.

Circular punched-out ulcers, with swollen edges and hyperæmic margins, varying in size from about a pin's head to a pea, are

frequently met with. Others, again, attain a size varying from a sixpence to a crown, and have either well defined, or ragged, undermined edges. Transverse ulcers chiefly implicate, as Morehead pointed out, the transverse folds or rugæ, and "are met with in bands, or in several bands, coalescing and occupying a greater or less extent of the mucous membrane." Irregular or sinuous ulcers seem to be formed by the fusion of a number of the circular or transverse ones. Their edges are sometimes ragged and undermined, at other times swollen and congested. They may cover a considerable area, like a fretwork, or they may be serpiginous in form, presenting the appearance of a coast line broken with gulfs and projecting promontories.

As regards depth, some ulcers involve the mucous coat only, others extend through the sub-mucous coat, while it is by no means rare to find the floor of the ulcer formed by the thickened peritoneum. Their bases may be firm or sloughy.

Gangrene of the mucous, or of the mucous and sub-mucous coats, is one of the commonest and most characteristic lesions in tropical dysentery. Perhaps it is not equally common in all warm climates. Hunter[1] states that he had never seen in Jamaica the mortifications, gangrene, or abrasions of the villous coat described by Pringle. In many tropical and subtropical countries it is common to meet with grey, livid, or black patches on the mucous membrane, which exhale a gangrenous odour. The size of these patches varies from a lentil to that of a crown or larger. When the gangrenous spots are small they are often discrete, the intervening mucous membrane being hyperæmic, and thickened by serous or sanguineous infiltration, or, in some rare cases, normal. On making a section of the intestinal wall through one of the more circumscribed of these gangrenous patches, the submucosa will be found to be thickened, softened, and often infiltrated with ichorous pus, which oozes out on pressure.

The submucosa is more widely involved than the mucosa, so that when the slough is disintegrated or detached, the resulting ulcer is more or less undermined, with its base soft and ragged. In this way extensive portions of the mucous membrane may be destroyed. In the worst cases the gangrene involves the muscular coat, and may even attack the serous investment, and end in perforation. The illustration (Fig. 47) exhibits the characters of this ulcer.

Another form of ulcer has been described by Kelsch and Kiener. This is the result of what they term the dry eschar. It appears as a furuncular tumour, more or less acuminated, of the size of a hemp seed to that of a pea, and usually pretty well defined at the periphery.

[1] Hunter, *Observations on the Diseases of the Army in Jamaica*, Lond. 1796, p. 184.

The more recent and smaller of these eschars have a uniformly red or livid red colour, and are of a semi-soft consistence; while the older and larger ones, although red at the periphery, have their central part coloured yellow by bile, and are firmer in consistence, and at the same time friable. These dry eschars may be disseminated over considerable portions of the diseased membrane, both on the folds and also in the depressions between the folds. The ulcers arising from the detachment or disintegration of these eschars are round,

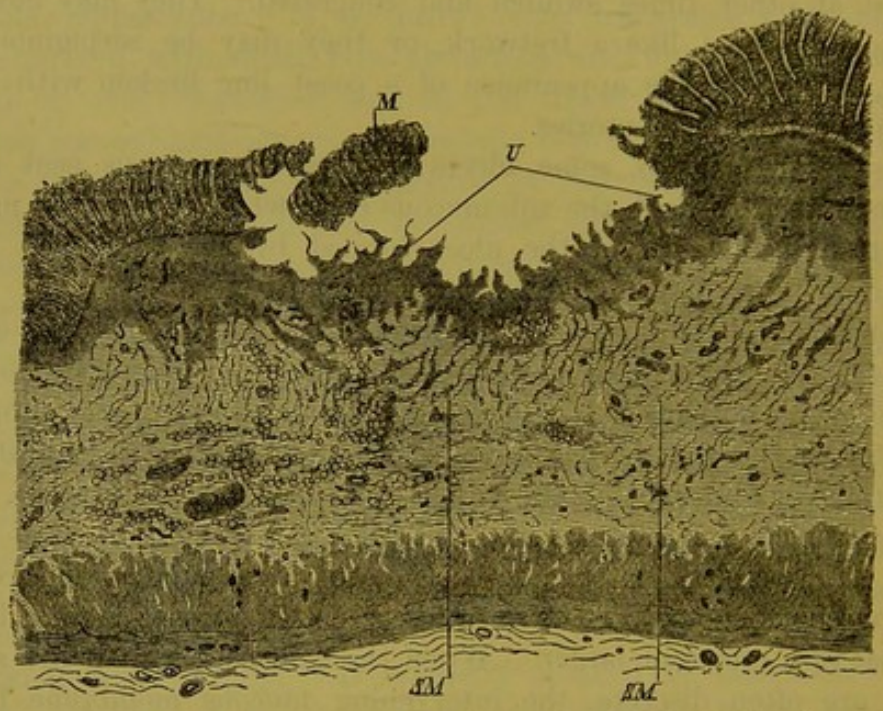

FIG. 47.—Section of gangrenous ulcer. (After Kelsch and Kiener.)
U, Shows the soft, ragged base of ulcer.
M, The sphacelated, detached portion of mucosa.
SM, Thickened, œdematous, infiltrated submucosa.

involve the mucous coat, and extend in the submucosa more widely than in the mucosa—the *ulcère en bouton de chemise* of the authors whom we have mentioned (Fig. 48). By their coalescence they give rise to larger sinuous ulcers. According to these authors, the primary lesions in dysentery are the dry and the gangrenous eschars.

The descriptions of the naked eye appearances of the large intestine in dysentery given by various authors are so widely different, not to say conflicting, that it is with some hesitation that we venture to identify the tubercles of Hunter with the dry eschars of Kelsch and Kiener. Here, however, is the description of what Hunter saw in the cases he examined: "There is first a small round tubercle of a reddish colour, and not more than one-tenth of an inch in diameter; it increases gradually till it be near a

quarter of an inch in diameter, and becomes paler as it grows larger. In this stage there appears a small crack in the top, with a slight depression, which gradually increase; and on examining the contents of the little tumour, I have generally found them to be a cheese-like substance. The pustule, for though it contains no *pus* I do not know any name more expressive of its appearance, is seated under the villous coat, between that and the muscular coat. As the opening enlarges the edges become more prominent, and the base grows rough and scabrous, from which matter oozes out that is sometimes tinged with blood. Such is the progress of one; but they are often in clusters, and become confluent, so as to form a rough, unequal ulcerated surface.[1]

Colin Chisholm also found in his autopsies in the West Indies, along with gangrenous blotches, tuberculous excrescences on the

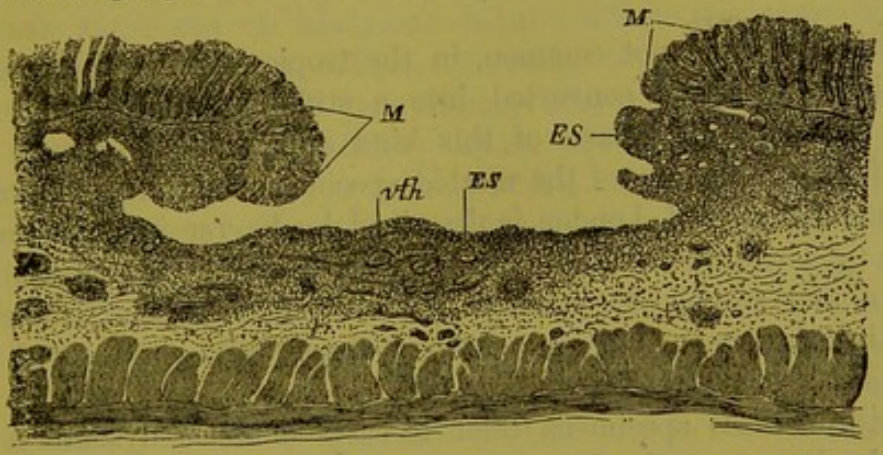

FIG. 48.—Section of ulcer caused by the dry eschar. (After Kelsch and Kiener.)

ES, Dry eschar at base and edges.
M, Undermined mucosa still retaining the form of the elements of the tissue.
vth, Thrombosed vessel.

internal surface of the large intestine, resembling "very much the pustules of smallpox."[2]

It is doubtful if a true croupous membrane is ever met with in tropical dysentery. Extensive exudations of a diphtheritic nature are also, in our experience, rare. Specks and films of yellow or grey exudation are, however, not uncommon, occupying by preference the summits of the transverse rugæ.

To complete our account of the naked-eye appearances in dysentery, it will be necessary to notice the appearance of the bowel in the diphtheritic or fibrinous form, as described by Virchow and Heubner,—a form which, we believe, is not of common occurrence in the tropics. In the fibrinous form of the disease—

[1] *Loc. cit.* p. 183.
[2] *Manual of the Climatic Diseases of Tropical Countries.* London, 1822.

The whole of the large and the lowest convolutions of the small intestine appear externally of a dark bluish-red colour. The fine vessels of the serous covering are injected, and the whole tube looks stiff, and feels hard and solid. The lumen is rather diminished than enlarged. Internally, the bowel presents a reddish-white, uneven surface, continuous from the lowest part of the ileum to the rectum, but patched with a great variety of crooked figures, of blackish, greenish, or dark red colour, which lie close on each other. . . . On cross-section the walls of the intestine are found exceedingly thick, but there can be distinguished only two layers: the thicker one being the muscular coat, which is puckered inwards in many folds, and over it a stiff, homogeneous tissue, partly yellowish and partly reddish, which offers considerable resistance to the knife. At places where furrows and cracks between the raised portions are situated, there is seen on cross-section only a small quantity of distorted tissue lying on the muscular coat, which is the remains of the mucous and sub-mucous coats that have atrophied from pressure. . . . In this form gangrene is sure to follow, unless death occur before this stage is reached.[1]

Although it is not common, in the tropics, to find the whole of the large intestine converted into a stiff, hard, solid tube, such as is here described, cases of this kind apparently do occur. We find, for example, one of the morbid specimens exhibited before the Medical Society of London is described by Fayrer in the following terms: "The gut is diseased from the cæcum to the anus; it is *swollen* and *rigid*." Of another specimen it is said: "The whole intestine is contracted in calibre, abnormally *firm*, *rigid*, and *shortened*."[2]

Both of these specimens refer to cases of *acute* dysentery. In chronic dysentery the intestine, as we shall see, is often stiff, hard, and solid in parts; but when this condition is met with in the acute form of the disease (not supervening on the chronic form, which it sometimes does) it may be taken to indicate fibrinous exudation into the tissues of the intestinal wall.

The naked-eye appearances of the intestine in the amœbic form of dysentery described by Councilman and Lafleur may be briefly stated, using as far as possible the words of these observers, as follows: In every case there is great œdematous thickening of the intestine, sometimes involving all the coats, but especially marked in the submucosa, to which in some cases it is confined. The surface of the mucosa presents sharply circumscribed, projecting nodular thickenings of various size, in which are observed cavities filled with a gelatinous looking pus, communicating with the surface of the mucous membrane by small openings, frequently no larger than the head of a pin, but sometimes much larger. There are also

[1] Heubner, *Ziemssen's Cyclopædia*, vol. i. Lond. 1875.
[2] Fayrer, *Tropical Dysentery and Chronic Diarrhœa*. Lond. 1881.

sinuous tracts, sometimes representing an extension of the cavity, at other times communicating with neighbouring cavities, all filled with the same gelatinous material. Various kinds of ulcer are observed: first, ulcers characterised by purulent infiltration, softening, and cavity formation in the mucosa, which often communicate with neighbouring ulcers by passages in the submucosa. Others with clean bases and smooth sides; others with undermined edges, representing simple excavations in the thickened sub-mucous tissue; others, again, with extensive sloughs adhering to the sides. When the disease has terminated in gangrene, the appearances proper to this condition will be present.

Nature of the Dysenteric Lesions.—It has been too readily assumed that dysentery always begins with a definite pathological lesion; that it uniformly develops in one particular way; that its lesions at each stage are of the same nature, affect the same tissues, and in the same order. Thus, according to some observers, the disease is essentially, in its primary stage, an inflammation of the solitary lenticular follicles; others, again, refer it to specific gangrenous processes, dry or humid; while, according to many pathologists, the severer forms of the disease are characterised by fibrinous exudations, which ultimately induce gangrene by compression of the tissues. Granting that dysentery is always the result of a specific cause; granting even, what has by no means been proved, that it is in every instance the result of *the same* cause, it does not necessarily follow that the lesions produced should always be of the same nature, or be evolved in a definite order of succession. If the same chemical substance is capable of producing only a slight irritation with hyperæmia, or, by impairing and destroying the vitality of the tissues of giving rise to suppuration or gangrene, according to the intensity of its action and the structure and vital condition of the tissue to which it is applied, it is not unreasonable to suppose that the dysenteric *virus*, if we may be allowed to call it so, may produce very different lesions according to its intensity and the tissues (mucosa or submucosa) primarily affected. These considerations serve to warn us not to allow ourselves to be influenced by theoretical considerations in our study of the pathology of a disease as yet imperfectly understood.

We shall confine ourselves, therefore, to a brief statement of the nature of the lesions more commonly met with in fatal cases of dysentery, without attempting to give an account of their evolution, which will apply to every case in all stages and forms of the disease.

Although we seldom have an opportunity of observing the lesions proper to the mildest forms of dysentery, which never prove directly

fatal, yet we frequently find the dysenteric process in its various stages in different parts of the bowel in fatal cases of the severer forms. The first departure from the normal manifests itself in a hyperæmic and swollen state of the mucous surface. The congestion may be punctiform, streaky, patchy, or diffused; and in every case the transverse folds are most affected. The hyperæmic membrane may sometimes be found covered with clear, thick mucus, which in some situations is tinged with blood, evidently derived from the rupture of the engorged vessels. As one of the first clinical symptoms of the disease is a discharge of such a mucus, we are justified in assuming that we have here before us the lesions proper to the earliest stage of the disease, in one if not in all of its forms. If we examine the hyperæmic membrane more closely, the solitary follicles, which in health are scarcely visible, are generally seen to be enlarged and prominent. They vary from the size of a millet seed, or less, to that of a pea, and are frequently surrounded with a vascular ring. When punctured, they are found to contain a white secretion, mainly composed of small round cells. The tissues surrounding the follicles suppurate, and thus, deprived of their vitality, they either disintegrate or are discharged as necrosed plugs, leaving a round ulcer having for its base the sub-mucous tissue. This tumefied state of the follicles is undoubtedly one very commonly met with in the earlier stages of dysentery, but it appears very doubtful if this condition is to be looked upon as a primary lesion. It seems more likely that these small glandular bodies become swollen as a result of the diseased condition of the surrounding tissues, and that they would become similarly affected if the bowel were inflamed from the action of any other irritant. If this view be correct, the *rôle* of these follicles in the dysenteric process is entirely a passive one. We have therefore to examine more closely the state of the hyperæmic mucous membrane in this stage. The only alterations that will be found in many cases is the disappearance of the epithelium from the mucous surface, an engorged state of the capillaries, some œdema and infiltration with leucocytes of the surrounding tissues, and occasionally interstitial extravasations of blood. Some observers have described the follicles of Lieberkühn as undergoing certain alterations in form. Generally they are constricted in one part of their length and dilated in another part, so as to present a beaded appearance, but these changes are not constant. The blood vessels by which they are surrounded are congested, the connective tissue infiltrated with exudation. The lining epithelium of the tubules is, however, always intact at an early stage of the disease, and nothing indicates that

they take any active part in the process. When the disease has advanced another stage, pus will be found underneath and around these tubules, which thus lose their vitality and connection with the subjacent tissues, and, being thrown off, give rise to superficial abrasions.

Before passing on to a consideration of the graver lesions observed in tropical dysentery, we have here to notice a condition described by Dr. McConnell of the Medical College Hospital, Calcutta, which he has met with at the autopsies of those who died at an early period of the disease. In these cases, he says, "The only visible alteration is in the solitary glands and follicles of Lieberkühn: the former especially, which I found enlarged to the size of small hazel nuts, and filled with that glazy, semi-transparent, glue-like mucus, which we almost always find voided with the earliest evacuations."[1] These observations are interesting, and are all the more remarkable that the appearances described were met with in those dying at an early stage. We have some difficulty, however, in believing that the solitary glands ever attain the size of hazel nuts. It would almost appear as if the lesions described were those of amœbic dysentery. Whatever may be the interpretation to be put upon these cases, they deserve to be more closely investigated.

In the more advanced stages and in the graver forms of the disease three processes play an active part in producing the characteristic lesions—these are sub-mucous suppuration, gangrene, and fibrinous exudation. Sub-mucous suppuration is not, perhaps, to be reckoned amongst the commoner lesions discovered after death; yet it deserves notice, inasmuch as its results may be traced in the extensive sloughing met with in some of the fatal cases. No better description of sub-mucous phlegmon can be given than that of Haspel: "The cellular tissue, which separates the different coats of the bowel, becomes thickened and infiltrated with serosity, blood, and pus. The mucous membrane is thus raised so as to form elevations, *mamelons*, and irregular swellings, flat at the summit, and sometimes the seat of ulceration. If these tumours are incised, a certain quantity of pus escapes. They seem to form so many small abscesses. When this suppuration of the sub-mucous connective tissue has invaded a large extent of the intestine, the mucosa is detached in large pieces, sometimes from the whole circumference of the gut, in the form of tubes, formerly mistaken for false membranes."[2]

[1] Fayrer, *Tropical Diseases*, Lond. 1881, p. 52.
[2] Haspel, *Maladies de l'Algérie*. Paris, 1850–52.

That sub-mucous phlegmon is often only an accident, as it were, in the course of the gangrenous process, may be admitted; but when we meet with collections of pus underneath a mucous membrane which shows no signs of primary gangrene, the truly phlegmonous nature of the process cannot well be doubted. The pressure of the pus in these cases, both by its irritant properties and the pressure it exerts, gives rise to secondary gangrene. We do not mean to imply that this sub-mucous suppuration is of the nature of an initial lesion; on the contrary, it is probably always the consequence of some morbid process which, by impairing the vitality of the submucosa, renders it vulnerable to the attacks of pyogenic micro-organisms. We have none the less to recognise it as one of the lesions of tropical dysentery, and as itself a cause of secondary gangrene. When we remember that the diseased mucous membrane teems with micrococci and bacilli, there will be no difficulty in accounting for these collections of pus.

In tropical countries, gangrene, limited or diffuse, is the lesion which, in our experience, is most frequently met with. How is it caused? It may arise in three ways: (1) as a result of the sub-mucous suppuration just described; (2) of thrombosis of the blood vessels and lymphatics, with more or less œdema and diapedesis into the surrounding tissues, depriving them of nourishment; (3) from the pressure of fibrinous exudation into the intestinal wall, occluding at once the vessels, and destroying the vitality of the tissues.

The way in which sub-mucous suppuration leads to gangrene has been sufficiently explained. The macroscopic characters of the dry and humid eschars have already been described. When the vascular area of the submucosa is primarily involved, and to such an extent as to cut off the blood supply suddenly and completely from a portion more or less extensive of the mucous membrane, the humid or gangrenous eschar is the result. When, on the other hand, the disease is primarily limited to the mucosa, and extends gradually to the sub-mucous coat, we have the dry form of eschar produced. In both forms the capillaries are dilated, engorged, and thrombosed; the lymphatics are obstructed by large polyhedral cells containing a round nucleus, and this obstruction doubtless favours the exudation of lymph into the tissues. In the dry form the elements of the tissues in the central part of the eschar, deprived of blood and compressed by effusion, are completely destroyed; around this is a zone in which the form of the elements, although necrosed, are still to be recognised; while outside this, again, is an area of œdema and fibrinous infiltration. In the humid form the primitive lesion differs from that observed in the other variety in being more exten-

sive, and in giving rise to more œdema, diapedesis, and interstitial hæmorrhage. Besides these points of difference, the necrosis in the humid form instead of progressing slowly from the surface inwards, involves the mucous and sub-mucous coats *en bloc*, and, instead of giving rise to a dry and friable eschar, ends in softening, having the characters of ordinary gangrene. The dry eschar is that proper to the milder, and the gangrenous to the severer forms of dysentery; but the two are met with in the same case (Kelsch).

In the diphtheritic or fibrinous form of dysentery the mucous and sub-mucous coats are said by Heubner to be transformed into an enormous extravasation, consisting chiefly of blood, less of pus, and of a stiff amorphous fibrinous exudation filling up the interstices of the tissues, compressing the glands of the mucosa so that they appear as elongated and narrowed double rows of epithelial cells. The fibrinous exudation may involve the whole or a more limited portion of the bowel; it may be primitive, or it may supervene on the catarrhal form; but the final result in any case will be either gangrene of the tissue, "or portions of the diseased bowel become changed into a brittle granular mass, and as the detritus is washed off, the loss of substance goes deeper and deeper into the intestinal wall."[1] This brittle granular mass corresponds to the dry eschar of Kelsch.

The part which this fibrinous exudation plays in the causation of gangrene in tropical dysentery appears to us small compared with that which is to be ascribed to thrombosis of the vessels. Whether this thrombosis is primary or secondary it is impossible at present to say.

Cicatrisation of Ulcers.—Repair in dysentery does not differ essentially from that observed in other sores; but the process is rendered slower and more precarious by the peristaltic action of the bowel, and the irritation caused by the passage of the fæces over the sore. When the loss of substance has been small, the contraction of the cicatrix suffices to bring the surrounding mucous membrane, with some puckering, over the sore. The puckering tends to disappear after a time, and ultimately a whitish, bluish, or pigmented spot is all that remains to mark the site of the ulcer. If the loss of substance has been extensive, recovery is slow, and the lesions are those proper to the chronic form. The place of the mucous membrane is supplied by new connective tissue, which on contracting may reduce the calibre of the gut more or less uniformly in some portion; or dense bands of white fibrous tissue are formed, which project into the lumen of the bowel and constrict it at a particular spot, giving rise to obstruction. In no case is there regeneration of lost glandular tissue.

[1] Heubner, *op. cit.* vol. i. p. 545.

Chronic Dysentery.

On opening the abdominal cavity, adhesions will often be found between the large intestines and the other viscera. The convolutions of the small intestines may adhere to each other and to the colon by lymph. The peritoneum covering the colon is often thickened. The bowel viewed externally may be either normal in size or of smaller diameter than natural. Frequently it is unequal in different segments. The walls are thickened, hard, and resistent to the touch in parts or throughout the greater part of its extent. The lumen of the bowel may be normal, or diminished by the formation of new tissue in the sub-mucous coat, and is often constricted in certain portions by old cicatrices. The bowel above the constricted portion will be found to be dilated. The colour of the internal surface of the bowel may be pale, pale red, or slaty;

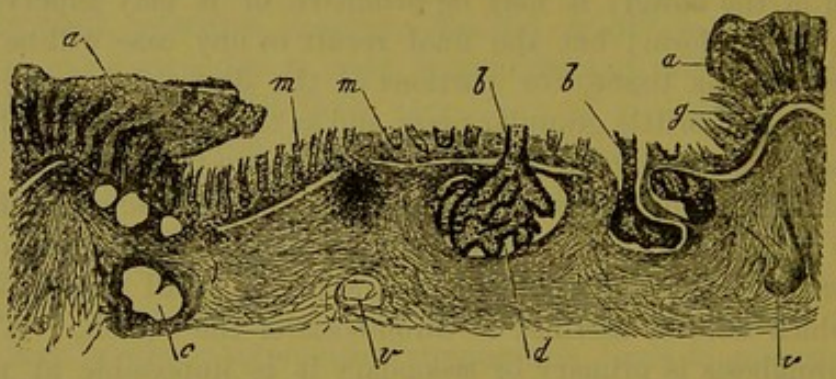

Fig. 49.—Chronic dysenteric ulcer. Magnified 20 diameters. (Cornil and Ranvier.)

a, *a*, Projecting portions of the mucous membrane with tubular glands.

b, *b*, Losses of substance filled up with mucus, and cylindrical cells opening by a narrow orifice on the surface of the mucous membrane.

d, Wall and contents of hiatus.

m, *m*, Tubular glands reduced to their *culs-de-sac* situated in the ulcerated part of the mucous membrane.

v, Vessels.

it is often deeply pigmented in places. When death has happened from the supervention of an acute attack, hyperæmia and recent inflamed ulcers will be present to a greater or less extent.

When death has resulted from chronic dysentery the mucous membrane is seldom free from ulceration. The ulcers of chronic dysentery are round, elliptical, transverse, or serpiginous, with thickened bases and callous edges. They seldom attain the size of those met with in many cases of the acute disease. The mucous membrane in some parts of the bowel will be found healthy, in other portions indurated, and the glandular structures are either atrophied or completely destroyed, the sub-mucous coat thickened, fibrous, or

almost cartilaginous. Whether the solitary chronic ulcer or the small groups of ulcers occasionally met in some part of the bowel are to be looked upon as dysenteric, may admit of doubt.

The illustration (Fig. 49), after Cornil and Ranvier, shows the appearance of the chronic dysenteric ulcer. The edges of the ulcers are thickened and congested; their surface slate-coloured. The glands of Lieberkühn, elongated and enlarged, are separated by connective tissue containing dilated and engorged blood vessels. The superficial muscular layer underneath the glands is normal; the connective tissue is hyperæmic. At the depressed and ulcerated parts the mucous membrane shows only the vestiges of the tubular glands. There are also to be observed follicular depressions visible to the naked eye, and filled with mucus resembling frog's spawn. Sometimes there is one single cavity, or the cavity is formed of many compartments completely or incompletely separated from one another by fibrous septa (Fig. 49, *c*). Both the single cavity and the multiple compartments are filled with mucus, and bordered by a layer of cylindrical, cup-shaped cells.

Associated Lesions of other Organs.

The *mesenteric glands* are always enlarged, softened, and reddened in the acute disease. In the chronic form they are enlarged, hard, and pale.

The *liver* may be normal in size, or enlarged and hyperæmic. In about 19 per cent. of fatal cases of dysentery the liver is the seat of solitary or multiple abscesses, the latter usually of pyæmic origin. The quantity, colour, and consistence of the bile in the gall bladder vary in different cases. In chronic dysentery the liver is usually fatty, a fact pointed out long ago by Dr. Bright; but this change is frequently met with in those who have died of the acute disease when it has been of some duration. According to Kelsch, fatty degeneration is constantly present in primitive gangrenous dysentery. In chronic dysentery the liver is often found to be atrophied.

The *spleen* is normal, unless the patient had previously suffered from malaria, in which case it is softened and pigmented, or enlarged and indurated, according as the malarial complication is recent or of old standing.

The *kidneys*, *lungs*, and *heart* present no constant or characteristic alterations in acute dysentery; but when the disease has lasted for a considerable time, and the patient has become anæmic and emaciated, these organs will usually be found to be more or less atrophied.

The *blood* in the larger veins is black, thick, and viscid. In the

subacute and chronic forms of the disease the mass of the blood is diminished, and the number of the red corpuscles very much reduced.

Symptomatology.

Acute Dysentery.—*Simple Form.*—Dysentery is seldom announced by prodromes, and when such is the case they are by no means so distinctive as to enable us to foresee with any certainty the approach of the disease. In some cases it will be found on inquiry that for a day or two before the attack the patient had been suffering from some irregularity of the bowels, such as constipation or diarrhœa, accompanied, it may be, by loss of appetite, slight nausea, and colicky pains.

Dysentery frequently makes its attack at night or early morning with frequent calls to stool. The motions are loose but feculent, differing little from those of ordinary diarrhœa; but even at this early stage a sense of fulness or uneasiness in the abdomen may be complained of, and griping pains precede the evacuation, which frequently affords temporary relief. Occasionally this preliminary diarrhœal stage is absent, and the disease begins with scanty mucus stools accompanied with gripes and straining.

In either case, when once established, there are frequent calls to stool, preceded by colicky pains—*tormina ventris*, accompanied by straining or *tenesmus*. The motions are now scanty, and consist of a clear gelatinous mucus, which soon becomes streaked or tinged with blood. As the disease develops, and in proportion as it is severe, the calls to stool increase in frequency. The tormina at the same time become more distressing, and while they come on spontaneously, they are also provoked by food or drink of any kind, and even by motion in bed. The tenesmus, too, becomes aggravated. The patient sits straining at stool without relief—passing only small quantities of mucus and blood, and in some cases a few hard masses of fæcal matter or *scybala*. The less the quantity discharged, the more the straining.

The motions from an early stage have a peculiar sickening odour, different entirely from that of normal fæces, and also to be distinguished from that of the stools when gangrene has set in.

When the disease has lasted for some days, the anus becomes inflamed, and is the seat of a burning pain. In severe cases the sphincter, at first contracted, becomes relaxed, and prolapsus may occur. Frequently the bladder is sympathetically affected, in which case there will be dysuria or even strangury. These symptoms are most pronounced when the rectum is much affected. The number

of motions in mild cases may vary from five to ten, and in severe cases from thirty to fifty in the twenty-four hours. The amount evacuated at each motion seldom exceeds a tablespoonful; frequently after long straining the whole discharge is only about a teaspoonful. In the simple form of dysentery, with which we are now dealing, mucus and blood form the principal constituents of the motions,—in some cases the mucus element predominates, in others blood is more abundant,—justifying the old English name of "bloody flux," by which dysentery was formerly known. Occasionally small quantities of pure blood—liquid or clotted—are passed, or the motions after a time may become serous or bilious, in which case they are more abundant. Pressure along the tract of the colon, especially deep pressure in the right or left iliac regions, causes pain.

The disease seldom begins with a distinct chill or rigor, although a sense of chilliness is now and then complained of, either at the onset or during the earlier stage of the disease. Fever is generally slight or absent, except when the patient has suffered from malarial fever. The skin is dry, or only becomes transiently moist during the attacks of tormina. The pulse in mild cases is normal; but when the disease is more severe, it is small and increased in frequency. The tongue is covered with a white coating. The appetite may be unimpaired, but more commonly there is anorexia. Vomiting, when it occurs in this form of the disease, is never urgent; the vomited matters consist of food or of bile-stained mucosities. The urine is scanty, of a high specific gravity, with an excess of urea and uric acid, and a diminution of the chlorides. Albumen is only occasionally present, and then in small amount. Sometimes the urine is found to give the reactions of bile, in which case the skin and conjunctivæ may present a subicteric tint.

When the patient is seen early, and subjected to proper regimen and treatment, the disease seldom lasts above a week. Frequently an improvement, which is signalised by the appearance of feculence in the stools, is observed as early as the second, third, or fourth day. In some instances the disease terminates as it began, in a slight diarrhœa, which readily yields to treatment. When the disease has been neglected, and in some instances, notwithstanding the most careful treatment, it may augment in violence and assume the characters of the gangrenous form; or it may, without becoming aggravated, persist in a subacute form and gradually subside into chronic dysentery.

Gangrenous dysentery begins in one of two ways: (1) With the symptoms of the simple form of the disease,—tormina, tenesmus, and scanty muco-sanguineous evacuations,—the symptoms in many

cases exhibiting an unusual intensity from the outset. (2) The disease may begin insidiously as a diarrhœa, often with comparatively little tormina or tenesmus. The stools, which are more copious than in the simple form, soon become mixed with mucus and blood, and at an earlier or later period acquire the cadaverous odour distinctive of gangrenous dysentery.

1. When the disease begins in the first of these ways, the symptoms, as we have said, are often severe from the beginning. There is frequently troublesome vomiting throughout the whole course of the disease. The calls to stool are in the worst cases incessant. The number of motions sometimes reaches 150 to 200 in the space of twenty-four hours. Scarcely has the patient left the stool when the recurrence of severe griping pains renders a return to it imperative.

When the rectum and sigmoid flexure are deeply involved, the straining is often agonising. The patient can scarcely be prevailed upon to leave the stool, although he passes next to nothing. His limbs and whole body tremble convulsively with pain. The sphincters become relaxed, and the prolonged straining in many cases brings on prolapsus of the lower part of the bowel. The irritation is propagated from the rectum to the bladder, and gives rise to dysuria or even to strangury. When the cæcum and ascending colon are the chief seats of the disease, the tenesmus will be less urgent, and, as Maclean remarks, "the depraved biliary secretions will be more abundant, and will be intimately blended with epithelium and blood."

The supervention of gangrene is announced by changes in the stools and in the condition of the patient. The motions become watery, of a reddish or brownish colour, like the washings of flesh, and on examination are usually found to contain flocculi, films, or thin shreds of mucus, or of the mucous membrane itself. Sometimes the muco-sanguineous stools of the earlier stage are first replaced by watery and intensely bilious motions. When the gangrene has made some progress, the stools are often thin, of a dark brown colour, like an infusion of coffee, containing in addition to membranous shreds, thin black sloughs; or, if the disease is still more severe, thicker sloughs of varying size, often infiltrated with pus or blood are passed. Now, too, we meet with fragments resembling flesh as large as peas or beans, infiltrated with blood and pus, which appear to consist of sloughs of the sub-mucous tissue. With these stools clots of blood and pus are frequently mixed; and in some cases large quantities of pure blood are evacuated. At the bottom of the vessel we frequently find a grumous sediment con-

sisting of the detritus derived from necrotic destruction of tissue. No less significant of the gangrenous condition of the intestine is the insupportably offensive odour of the stools—an odour which in some cases may justly be termed cadaverous, and which in all cases is so distinctive that by it alone a diagnosis of gangrenous dysentery may be made without the risk of error.

It must be remarked that the motions change from day to day in frequency, consistence, and character.

We have spoken of the discharge of fragments of mucous membrane by stool. These may be so small as to be detected only by careful search, but they are often of considerable size. The tubular sloughs, occasionally thrown off, have attracted attention from the earliest times. Aretæus states that "sometimes a substance of considerable length, in many respects not to be distinguished from a sound piece of intestine, has been discharged"; and he goes on to explain that this is the inner coat of the bowel, which becomes separated and protrudes externally, while the outer coat "incarnates and cicatrises, and the patient recovers and lives unharmed." Fayrer records a case in which a tubular slough, about a foot long, was discharged, and the patient made a good recovery. Dutroulau observed a similar case in which 35 centimetres of the mucous and sub-mucous coats were discharged. The patient recovered perfectly, and was known to be in good health six years after this took place. These tubular structures have been looked upon by some as false membranes; while Heubner, following Zimmerman, believes that the majority of these structures "consist without doubt of mucous only." Both of these views may, we think, be dismissed. The formation of tubes of false membrane in dysentery has never been proved; while no one who has seen one of these firm, tough organised membranes, is likely to mistake it for a simple mucous cast.

In the severe forms of dysentery a feeling of heat is frequently complained of along the tract of the colon, which may be felt thickened, doughy, and painful on pressure in certain parts, or more or less throughout its entire extent.

Pressure in the right or left iliac fossa will elicit signs of pain, according as the cæcum or rectum is the part most involved. When the peritoneum is inflamed, tenderness is generally present, but perforation of the bowel and fæcal extravasation into the peritoneal cavity have sometimes been found after death in cases in which no complaint of pain had been made by the patient; but in these instances the perforation has taken place during the last days or hours of life. When extensive gangrene has occurred, the severe pain, which usually characterises the earlier stages of the disease,

often subsides or at least moderates, and pressure may then elicit few or no signs of suffering.

2. When gangrenous dysentery begins insidiously in the second way which we have mentioned, the symptoms and progress of the disease are as follows: the patient is seized with a feculent diarrhœa, which at first excites little concern. It persists, and within a few days the motions become mixed with blood and mucus. The stools are now of a gelatinous consistence and copious, although not very frequent—say five to ten times a day. They vary greatly in colour in different patients, and in the same patient at different times, or even during a single day. They are frequently yellow, green, or variegated, and from time to time they may contain a considerable quantity of fluid or clotted blood. As the disease advances, they become more watery, and of a reddish or brownish colour, and are extremely fœtid. The diarrhœa is at first painless or nearly so; but when the motions become mixed with blood and mucus, colicky pains and straining are complained of, and at a more advanced stage the tormina and tenesmus are often urgent. Vomiting recurs from time to time, and interferes with the administration of food and medicine.

The following case shows the leading features of this form of the disease:—

Ramboojhawon, aged 20 years, a native of Calcutta, admitted into the Civil Hospital at Port Louis on the 11th October 1877.

History.—Just arrived from India, twenty-nine days at sea. States that during the latter half of the voyage he suffered from diarrhœa. Evacuations watery, greenish, and frothy. Attributes his illness to the salt fish he got on board.

Present condition.—Bowels opened fifteen or sixteen times a day; motions loose, frothy, greenish, offensive, tinged with blood and mucus; no straining, but complains of pain on pressure in the lower part of abdomen; great thirst; skin moist; pulse 120, weak.

Pulveris Ipecac. 15 grs.
Tinct. Opii, drachm 1.
Make a bolus to be given every eight hours.
Diet, milk and chicken.

October 12th.—Patient very weak. Has had eight loose, dark grey motions, tinged with blood; pulse 110; skin moist; great thirst; no appetite; morning temperature 37° C., evening 38°.

13th.—Condition as above; the motions contain shreds (probably of the mucous membrane), are copious and offensive; patient states that he feels worse. Pulse 110; morning temperature 37°, evening 38°. Urine high coloured. Was ordered bismuth 30 grains, quinine 5 grains, and Dover's powder 10 grains, every six hours, with daily injections of linseed infusion with a few drops of creosote.

14th.—Has had six motions since yesterday, of a dark colour, containing a little blood but no mucus; pulse 110; temperature at night rose to 38°·5.

17th.—Since last report the patient seemed to be improving. Motions

less frequent, and not mixed with blood or mucus. The temperature on the morning of 15th was 37°·5, rising as usual one degree at night; on the morning of the 16th it stood at 36°·8, and this morning at 36°·5. To-day, again, he feels worse. Has had eight dark brown motions; complains of pain in abdomen; frequent vomiting; skin moist and cold; great thirst; urine high coloured; pulse 110; tongue pale, but red at the edges.

Continue treatment.

18*th*.—Condition the same as yesterday.

19*th*.—States that he feels better. Bowels have been moved four times; motions contain a large quantity of blood and some shreddy matter; pulse, 112; tongue pale, but red at edges.

21*st*.—Feels worse. Has had ten yellow offensive stools, mixed with blood and mucus; great straining and griping.

24*th*.—Has continued in much the same state as when last report was made. Motions changing in colour, frequently dark green; yesterday he passed a considerable quantity of blood and mucus. Has had fourteen motions during the last twenty-four hours.

25*th*.—Has had frequent yellowish frothy stools mixed with blood and mucus, and large shreds of mucous membrane, accompanied by griping and straining; great thirst; pulse, 100; skin moist; tongue pale; ulcerous patches all over mouth and tongue. Omit the bismuth and repeat the bolus of ipecacuanha and opium.

27*th*.—Yesterday his bowels were opened involuntarily; to-day he is evidently sinking. Died at 7.35.

Autopsy eight hours after death:—

Brain and *lungs* healthy. *Heart* pale and flabby. *Kidneys* congested. *Liver* rather small. *Spleen* small, capsule not adherent. *Small intestines*—several spots of congestion on peritoneal coat. A few patches of ulceration on mucous surface; a number of diffused patches of congestion, the vessels forming a very distinct network, most numerous in ileum. Peyer's patches elevated and presenting the shaved-beard appearance. *Large intestines*—mucous coat greatly thickened and softened, presenting a number of dark brown fluffy villous projections. In many places the mucous coat has been destroyed, leaving the muscular layer exposed. Numerous sloughing ulcers about the size of a sixpence over its whole extent; also a number of congested patches exhibiting the same distinct network of vessels noticed in the small intestine. These patches are many of them larger than a dollar in size, and are somewhat circular in form. They are more numerous than in small intestine. *Mesenteric glands* enlarged, and some of them pulpy. Mucous membrane of mouth and pharynx covered with small ulcerous patches.

The general symptoms of gangrenous dysentery are those of asthenia. The pulse is weak and fast; prostration is well marked from the beginning and increases daily; consciousness remains perfect; the urine is scanty and often slightly albuminous, and towards the end the motions are passed involuntarily. These cases may run their entire course with little or no fever; or there may be a rise of a degree or two in the evening in the earlier stages of the disease, but the temperature falls to or below the normal when extensive gangrene develops. When the temperature sinks, the skin becomes cold and moist, and the pulse fast and weak. Occa-

sionally the patient falls into an algid state, the extremities become icy cold, the face pale, the lips livid, the features shrunken, the nose pinched, the eyes sunken, and the tongue cold. Sometimes in addition to these symptoms there are muscular cramps, copious liquid stools of a dark colour and of a gangrenous odour, and suppression of urine, presenting an *ensemble* of phenomena which closely simulates cholera. It is not easy to find a satisfactory explanation of the occurrence of algid and choleraic symptoms in dysentery. We may exclude entirely the theory of their malarious origin, for they have frequently been observed in non-malarious countries, and in patients who have never suffered from malarial fever.

Algid and choleraic symptoms, so far as we know, never occur except in the gangrenous form of the disease, either in its early or advanced stages; yet it is comparatively seldom that algidity, with or without muscular cramps and anuria, accompanies gangrenous dysentery. The fall in the temperature of the whole body, the moderate coldness of the extremities, and the moist condition of the skin so generally met with in the final stage of gangrenous dysentery, may reasonably be referred to the extreme feebleness of the heart's action. Here we have plainly to do with the coldness of collapse. But we are the less disposed to regard the true algidity, with which we are at present dealing, as a simple exaggeration of collapse, that in some few instances recovery from algid and choleraic dysentery has been observed. We must assume that, in these instances, the gangrene had been circumscribed, for it appears improbable that recovery should ever take place when the greater part of the large intestine is in a gangrenous condition. It follows, then, that algidity does not necessarily imply the existence of that extensive gangrene which would explain extreme collapse. If Cohnheim is right in explaining the *stadium algidum* of cholera as a result of the extreme weakening of the circulation which attends the inspissation of the blood in that disease, and which is naturally most pronounced in the peripherally situated vascular areas, and if he is right in believing that this inspissation of the blood also explains the suppression of urine, we may be permitted to conjecture that some analogous changes in the blood occasionally take place in dysentery, which accentuate the collapse caused by gangrene, and give rise to algidity and anuria.

Symptoms of Amœbic Dysentery.

Numerous varieties of bacteria, both aërobes and anaërobes, are constantly present in the contents of the intestinal canal in health,

and are always to be met with in the dejections of dysentery; but we have no evidence that any of these bear a causal relation to the disease. The sloughy tissue discharged in gangrenous dysentery, all the coats of the bowels, the walls and lumen of the veins of the submucosa, and the muscles of the latter, teem with micrococci, and bacilli are also very numerous; but these organisms, although they may well be allowed to play an active part in exciting suppuration after the disease has been established, do not seem to take any part in setting up the dysenteric process. The observations of Lösch,[1] Kartulis,[2] Koch,[3] Osler,[4] and others, prove beyond doubt that the *Amœba coli* is frequently met with, not only in dysenteric stools, but also in dysenteric ulcers, and in the contents of liver abscess complicating dysentery. This micro-organism was also met with in Egypt by Sonsino,[5] not only in those who had died of dysentery, but also in patients who had succumbed to other diseases. Councilman and Lafleur, to whose monograph we have already referred, consider that this amœba is the direct cause of a distinct form of dysentery met with in the tropics, which they distinguish as amœbic dysentery. Confirmative observations are required before an amœbic form of dysentery can be accepted as absolutely demonstrated. The evidence of the injection experiments hitherto made are not quite conclusive. Without prejudging the results of further investigations, we shall give in briefest outline the principal features of that form of dysentery which is ascribed to the presence of this micro-organism, following as closely as possible the description of Councilman and Lafleur.

Amœbic dysentery is either abrupt or gradual in its onset; it follows an irregular course marked by intermissions and exacerbations; it exhibits an immense latitude between the extreme periods of duration, and has a tendency to chronicity.

When the onset is sudden the patient is seized with colicky pain and diarrhœa, with or without nausea and vomiting, and, as a rule, without fever, although during the course of the disease the temperature may rise to 100° F. The stools are frequent and watery, and ultimately (if not from the beginning) they contain blood and mucus. When blood and mucus are present from the onset, the stools are scanty and not so watery.

When the onset is gradual a moderate and painless diarrhœa sets in, alternating with short periods of constipation. Blood is in this

[1] "Massenhafte Entwickelung von Amœben in Dickdarm," *Virchow's Archiv*, 65, 1875.
[2] "Zur Ætiologie der Dysenterie in Ægypten," *Virchow's Archiv*, 1885.
[3] "Arbeiten aus dem Gesundheitsamtes," No. 3, *Cholera Bericht.*
[4] *Johns Hopkins Hospital Bulletin*, vol. i. 1890.
[5] See article by Sonsino in this work, section on Parasitic Diseases.

case absent, or is only occasionally present, the stools being watery and containing more or less mucus.

Whether sudden or gradual in its onset, there are irregular periods of intermission (from one day to three weeks) and of exacerbation (one to ten days) in the dysenteric symptoms. A case of moderate severity may subside into chronic dysentery, or prove fatal from an acute exacerbation, and at the autopsy show gangrenous lesions; or, again, an acute exacerbation may supervene on the chronic form. Amœbic dysentery seldom lasts less than three weeks,—six to thirteen weeks being the more usual range,—but the disease may be protracted for a longer period without presenting the features of the chronic disease. Chronic cases last for months or years. "A chronic state may be inferred when a moderate diarrhœa has continued for many weeks with or without intermissions, the stools being liquid, homogeneous, without blood, and unaccompanied by pain." A fatal issue may result from intestinal ulceration or exhaustion, or from some complication, such as liver abscess, peritonitis, hæmorrhage, or from the supervention of diphtheritic inflammation of the intestine.

In all protracted cases there is anæmia with diminution of the corpuscular elements and of the hæmoglobin. Abscess of the liver, or of the liver and lung, appear in about 50 per cent. of the cases; but the supervention of liver abscess does not occasion an exacerbation of the intestinal symptoms.

In the gangrenous form of amœbic dysentery the motions at the outset may number thirty to forty in twenty-four hours, but they subsequently become less frequent. The amount voided is at first small, and consists of clear mucus mixed with blood, and occasionally small fæcal masses. As ulceration advances the stools become more copious, watery, and less homogeneous; blood is less frequently observed, and small shreddy masses of a greyish or light yellow colour appear, mixed with blood-stained mucus. When extensive sloughing is taking place, the character of the stools is even more varied. They are then of a greyish, greenish, reddish-brown, or variegated colour, sometimes quite liquid, at other times pultaceous, mixed with stringy masses of necrotic tissue, and having a very penetrating offensive odour.

The stools of chronic amœbic dysentery are more uniform, and are watery, or of the consistence of thin gruel, of an earthy or dull yellow colour, and contain particles of clear mucus. During an exacerbation blood and greenish pultaceous material may be seen.

Amœbæ are found in all varieties of the dysenteric stool, at all

periods of the disease, and are most abundant in those cases in which the lesions are most acute and extensive.

Epidemic Gangrenous Rectitis.

We meet, scattered up and down in medical and general literature, with accounts of an epidemic gangrenous rectitis or colonitis which deserves brief notice, the more so that the disease, although probably etiologically distinct, is closely allied to some of the forms of dysentery which we have described.

This disease was formerly known in Trinidad.[1] It is occasionally epidemic among the Indian tribes of Guiana. Brett's account of it is as follows:[2] "In the year 1856 our Arawâks were further thinned by a sore disease, an epidemic known by the name of 'Caribi' or Indian sickness. . . . It is highly infectious (*contagious understood*), and when it seizes a person, eats its way upwards through the rectum and other intestines until the person dies."

Leblond, quoted by Sigaud,[3] states that the Spaniards as well as the Indians in South America, who live in marshy localities and who are badly nourished, are affected with this disease.

Two forms are distinguished, the "high" and the "low," according as the colon or rectum is chiefly involved. When the rectum is the part affected, it commences, we are told, with a dilatation of the anus, rapidly followed by gangrene, accompanied by fever, prostration, and an inclination to sleep. The symptoms of the form which begins in the colon are not given. It is stated, however, that when the colon is primarily attacked, the malady may afterwards extend downwards to the rectum. A similar complaint is observed in Fiji. Corney gives an account of an epidemic gangrenous stomatitis with an analogous condition of the rectum which he observed in Fiji, and which, he says, is the most fatal disease of the South Seas.[4]

Symptoms of Chronic Dysentery.

Chronic dysentery sometimes begins insidiously as a diarrhœa, which intermits and recurs. After a longer or shorter time the stools are found during the exacerbations to contain mucus and blood, and instead of being passed painlessly as in the first stage,

[1] We have found a reference to its existence in Trinidad, but have been unable to consult the original. O'Connor, *Medical Recorder*, Philadelphia, No. 32, 1825.

[2] Brett, *Indian Tribes of Guiana*. London, 1868.

[3] Sigaud, *Du climat et des malad. du Brézil*. Paris, 1844.

[4] Corney, *Trans. Epidem. Soc.* 1887–88.

they are accompanied with some degree of tormina and straining. Under care and treatment the dysenteric symptoms may cease and a looseness remain, which in many cases alternates with constipation. After a varying interval it too frequently happens that the dysenteric symptoms recur, and chronic dysentery of an obstinate kind is established.

In other cases an acute attack, instead of ending in death or recovery, subsides imperceptibly into the chronic form. In still another class of cases, after a real or apparent cure of the acute disease, there are repeated relapses which at length end in chronic dysentery.

The motions in chronic dysentery vary greatly in different cases, and in the same case in different stages of the disease, or even in the same stage from day to day.

Diarrhœa alternates with constipation, but looseness is the ruling condition throughout the whole course of the disease. During the short intervals of quiescence, which often give rise to disappointing hopes, the motions may be healthy. More frequently between the exacerbations they consist of thin frothy offensive feculence,—yellow, green, or light-coloured,—mixed occasionally with specks or clots of blood and with small masses of mucus. When exacerbations occur—and in most cases they are frequent, lasting from two to ten days—the stools are watery, of a reddish-brown colour and mixed with blood, mucus, or pus, or all combined, have a distinctly dysenteric odour, and are accompanied by colic and straining.

The disease, if not overcome by treatment, lasts for an indefinite period, which may be measured by months or years, and tends to become aggravated in proportion as it is protracted. The patient's health fails. The appetite is either bad, capricious, ravenous, or variable. The digestion is impaired, and, as a result, portions of undigested food pass along the intestinal canal, irritating it on its way, and are found in the stools. Vomiting is of occasional occurrence. The tongue is red and glazed, the skin dry, although in the later stages night-sweats are often troublesome. The patient loses in flesh and strength, and becomes visibly anæmic. The pulse is always weak, and often accelerated. Œdema of the feet frequently occurs towards the end, and the patient dies of exhaustion.

Modified Dysentery.

Dysentery may be modified in its manifestations by the concurrence of other diseases or morbid conditions, such as malaria, common continued fever, scurvy, rheumatism, septicæmia, typhoid or typhus fever.

Malaria.—When patients who have previously suffered from malarial fever are attacked with dysentery, the disease is often observed to begin with chills—sometimes repeated and severe. A moderate degree of febrile disturbance of a remittent or intermittent character marks the course of the disease in these patients. The morning temperature may be normal or a degree above normal; in the evening it rises to 99°·4, 100°, or 101° F., even although the dysentery be of the mildest type.

Unless malaria has induced a cachectic state of the constitution, the fact of having previously suffered from fever does not sensibly modify the course of the disease. On the other hand, those whose constitutions have been broken down by repeated attacks of malarial fever are specially liable to dysentery, and the disease in these subjects often assumes a grave character.

When the two diseases—malarial fever and dysentery—occur simultaneously, the result varies in different cases. The two diseases may run their course without the one affecting the progress of the other, or both may become aggravated by association. When an attack of malarial fever supervenes on dysentery, the dysenteric symptoms are sometimes temporarily or permanently ameliorated. A very formidable type of malarial dysentery is met with in troops on active service in tropical countries during the fever season, especially when subjected to much fatigue, and exposed to the sun by day and chills at night, and have to wear wet clothing. The patient generally has suffered from an attack of remittent or bilious remittent, which has reduced his strength before the dysentery declares itself. A relapse of fever ensues, accompanied with a simple looseness, which sooner or later develops into dysentery, with frequent motions of a chocolate or brownish-red colour, passed with little straining, alternating with discharges of blood. There is usually marked tenderness over the cæcum, and more or less along the tract of the colon. The fever at first is high, with distinct remissions or intermissions. After four or five days the fever subsides, prostration ensues, the motions are passed involuntarily, and the patient dies in a state of collapse. The autopsy reveals extensive gangrene of the cæcum; more or less gangrene and ulceration of the colon; patchy congestion of the small intestine, and sometimes ulceration of the lower part of the ileum. The disease may prove rapidly fatal, or it may be protracted for a period of three weeks to a month.[1] Dysentery may attack bodies of men who have been exposed to malaria in its more intense

[1] Some cases of this form occurring during the Ashanti war of 1874 will be found recorded in the *Med. Times and Gaz.* of that year.

manifestations, and whose health has been impaired by fever. This form of dysentery, which is not necessarily febrile, has been noticed in the article on malaria.

In the dysenteric form of pernicious attack described in the article on malarial fever, the dysenteric symptoms are to be looked upon as symptomatic of the malarial infection. The same may be said of the intermittent dysentery, occurring every other day, which is mentioned by various authors,[1] and of the so-called hæmorrhagic dysentery, in which large quantities of blood are evacuated at quotidian or tertian intervals.[2]

Common Continued Fever.—Common continued fever, with or without bilious symptoms, may complicate dysentery. Johnson [3] relates how, after a day's shooting under the burning sun of Bengal, in the marshes near Kedgeree, he was exposed to wet and cold. The same night he had chills alternating with flushes of heat. Next evening high fever, with delirium, and very severe dysentery came on; he ultimately recovered under scruple doses of calomel. It is probably only in such circumstances that active delirium is observed in acute dysentery. The delirium is symptomatic of the fever, not of the bowel disease.

Scurvy.—Patients suffering from latent or fully developed scurvy are specially liable to suffer from dysentery. Indeed, all cachectic states of the constitution powerfully predispose to this disease. The scorbutic condition modifies in a marked way the symptoms and course of dysentery. The disease in scorbutic subjects seldom commences with scanty, blood-tinged mucus discharges, but with diarrhœal motions, which soon become watery and mixed with blood and mucus. The stools frequently consist of blood, or rather, of a sanguineous fluid, of a highly offensive odour, with mucus and shreds of sloughy tissue. They are usually only moderately frequent, but are copious, and accompanied by tormina and tenesmus, but less severe than in ordinary cases of acute dysentery. Scorbutic dysentery runs a somewhat protracted course, is characterised by great prostration, and tends to end in gangrene. The pale, sallow, leaden colour of the skin, the spongy state of the gums, and the presence of hæmorrhagic extravasations into the skin or connective tissue, will indicate the nature of the modifying cause. Attacks of malarial fever predispose to scurvy.

Rheumatism.—Many authors have recognised a rheumatic form of dysentery, characterised by prodromic chills recurring

[1] Wood, *Practice of Medicine*, 4th ed., Philad. p. 595; Torti, *De febribus*, lib. iii.
[2] Béranger-Féraud, *op. cit.*
[3] *Influence of Tropical Climates.* London, 1827.

during the course of the malady; by intense pains in the head, loins, and limb; by high fever, hard pulse, abundant sweats, and unusually severe tormina. This form of the disease we have never met with in the tropics. But we occasionally meet with swelling of the joints during the course of dysentery or towards its termination.

In certain cases the joint symptoms appear without any marked febrile disturbance or sweating. The pain is slight, the swelling moderate, and the affected joint is neither very red, nor hot to the touch. The disease may remain fixed in the joint first attacked, which is usually the knee joint, but more frequently several joints are affected in succession. When the disease seizes a fresh joint, the inflammation does not rapidly subside in that first attacked. The heart is seldom affected in this form of the disease.

In another class of cases the joint affection begins with fever, is accompanied by more or less profuse perspiration; there is much redness, pain, and swelling of the articulation. When the disease seizes another joint, the pain and swelling rapidly subside in that previously inflamed. In both forms the appearance of the joint disease has been observed to be accompanied or followed by an amelioration of the dysenteric symptoms, but this is not uniformly the case.

In the first class of cases it has been thought, and with some reason, that we have to do with a specific dysenteric arthritis, not, strictly speaking, rheumatic in its character. In the other class of cases the rheumatic nature of the disease is not doubtful. The joint affection, although it may last for weeks or even months, usually gets well, and leaves no bad effects; but Trousseau has observed a case in which it was of so severe a character that the synovial effusion caused rupture of the capsule.[1]

Septicæmia.—We ascribe to septic poisoning those forms of dysentery which have been known as *typhoid* or *malignant*. The symptoms in this form are those of the typhoid state: chills, fever, nervous prostration, stupor or sub-delirium, sunken features, dry, black tongue; occasionally parotitis, diffuse abscesses in various parts of the body, and suppurative peritonitis. It is when the disease has reached the stage of gangrene that the typhoid symptoms declare themselves.

Typhoid or septicæmic dysentery may appear sporadically, but it is more frequently met with in armies, camps, besieged cities, or in circumstances in which large numbers of broken-down dysenteric subjects have to be treated together under unfavourable hygienic conditions.

[1] *Clinical Med.* vol. iv. Sydenham Soc. edition, p. 171.

Typhoid Fever.—Dysentery is sometimes observed to appear during convalescence from typhoid fever, and typhoid fever has also been known to occur in persons convalescing from dysentery. The two diseases may declare themselves simultaneously in the same patient, and this double infection has been observed occasionally to occur in an epidemic form.[1] In the last mentioned case the symptoms of the two diseases are combined, and at the beginning are alike pronounced; but as the disease advances the dysenteric symptoms yield—sometimes suddenly—to those of typhoid fever. Whether the two maladies run concurrently, or the one arises during convalescence from the other, the prognosis is alike unfavourable.

Typhus Fever.—Many of the fatal epidemics of the Middle Ages, especially those occurring in camp, are believed to have been of the nature of typhus associated with dysentery. The same combination has frequently been observed in Ireland in the last and the beginning of the present century, when both diseases were often epidemic at the same time. The symptoms are those of typhus, with frequent dark sanious, bloody, and mucous stools, with tormina and tenesmus. The mortality in these cases is very high.

Complications, Relapses, and Sequelæ.

Functional and Organic Liver Disease.—It is comparatively rare to find the liver perfectly healthy in those who have died of tropical dysentery.

Out of 411 fatal cases of dysentery occurring in Senegal, Béranger-Féraud found the liver in 313 instances (76·1 per cent.) to present evident *macroscopic* morbid alterations. In 39 per cent. of those cases in which the disease was so marked as to be readily detected by the naked eye, the lesions present were hypertrophy, softening, or congestion; and in 46 per cent. there was abscess. In summing up his observations on the condition of the liver in dysentery, he states that he has never met with a case of confirmed dysentery in which this organ, if healthy to the naked eye, was not found to present some morbid change when examined microscopically. It must not, however, be overlooked that the liver is frequently found diseased in Europeans who have resided long in the tropics, whatever may be the nature of the fatal disease. But if we are not justified in ascribing every morbid change met with in the liver in fatal cases of dysentery to the intestinal disease, we are still less justified in doubting that in many cases of dysentery the liver is more or less seriously involved.

[1] Kelsch and Kiener, *op. cit.* p. 80.

In a certain class of cases the more prominent symptoms of hepatic disorder in connection with dysentery are nausea, bilious vomiting, yellow and green stools, uneasiness or tension in the right hypochondrium, which in some instances is increased on pressure.

In other cases, instead of an excessive discharge of bile by the mouth or bowels, there is an entire absence of it from the stools for a longer or shorter time. The urine in some of these cases is found to contain biliary colouring matters, and the conjunctivæ and skin may exhibit a yellow tinge. The condition of the liver upon which these symptoms depend is to a large extent a matter of inference; for there are few opportunities of examining the liver in the earlier stages in which these symptoms are marked.

The liver symptoms are in many instances so early and so closely associated with those of dysentery, that it would appear as if the cause of the latter gave rise directly and at the same time to the former, the dysentery and the liver disorder being the effects of the same morbific cause. Be this as it may, a high degree of functional disturbance of the liver usually indicates a severe form of dysentery.

Hepatitis, whether ending in resolution or in suppuration, may precede, follow, or accompany dysentery. The influence of the one disease upon the course of the other cannot be better stated than in the words of Annesley: "During the progress, not only of dysentery, but also of hepatitis, it will occasionally be observed that the one affection supervenes upon the other, disappears for a while, and then returns. Thus, during hepatitis, dysentery will sometimes take place, disappear after two or three days, the hepatic disease becoming more acute, and again return in an aggravated form. . . . Thus, also, during the progress of dysentery, hepatic disease sometimes evinces itself, the dysenteric disorder either becoming somewhat alleviated or being for a time altogether removed, but afterwards returning, and accompanying the hepatic disease to its termination."[1]

Invagination of the Intestines.—This condition, which sometimes complicates dysentery, may be recognised by the supervention of bilious vomiting, which afterwards becomes feculent; by a tympanitic condition of the abdomen, which will be found to be painful at certain spots on deep pressure; by the complete arrest of feculent discharge, if any had before been present in the evacuation; by coldness of the body, and by the shrunken and pinched appearance of the features.

Among occasional complications may be mentioned, iliac or peri-

[1] *Op. cit.* p. 390.

rectal abscess, tænia and other worms, intestinal obstruction, occlusion, or perforation.

Relapses.—Relapses are apt to follow indiscretions in diet and exposure to cold during convalescence, especially in those whose constitutions have been enfeebled by previous disease, by the abuse of alcohol, or by a long residence in tropical countries. The danger of a recurrence of the disease increases in proportion to the number of previous attacks.

Sequelæ.—*Diarrhœa.*—Acute dysentery frequently terminates in a diarrhœa, which in most cases yields readily to treatment. Occasionally, however, it happens that the diarrhœa persists, and, if neglected, may form the stepping-stone to chronic dysentery. *Rectitis*, to which we shall presently have occasion to refer, is also a sequel of acute dysentery.

Paralyses.—Paralysis of the lower extremities, of the sphincter ani, of the bladder, of the tongue, of the face, or even of the upper extremities, are amongst the rarer sequelæ of dysentery. Paralytic symptoms are seldom observed except towards the end of severe or protracted cases of the acute disease, but they also occur in the course of chronic dysentery. Motion alone, or motion and sensation, may be affected. As a rule, the paralysis disappears after a few weeks or months, although in a few instances it has been known to become general and to end in death.

Dysenteric paralysis may be looked upon as analogous to that following diphtheria, the latter having its point of departure in the throat, the former in the rectum.

Anæmia and *dropsy* of the lower extremities are frequently met with consecutive to prolonged attacks of the acute form in cachectic subjects, particularly in those who have suffered from malarial fever or scurvy. They are still more common during the course of the chronic disease.

Constrictions of the intestinal canal may follow upon acute or chronic dysentery.

Diagnosis.

Acute Dysentery. — The diagnosis of acute or chronic dysentery will seldom give rise to much difficulty, if due consideration be given to the symptoms and the character of the stools. Neglect of the obvious duty of examining the motions has led to deplorable errors. An extraordinary and fatal mistake arising from this cause came under the observation of one of us. A lady, who had previously manifested hysterical symptoms, was taken ill. Her motions were scanty and serous, by no means very frequent, but

passed with some tenesmus. Her chief complaint was, not the intestinal trouble, but dysuria. She was treated for hysteria by cold baths; and, incredible as it may seem, the true nature of the disease from which she was suffering was not recognised until the protrusion of a tubular piece of the mucous membrane rendered it impossible any longer to mistake its dysenteric character. Had the motions been inspected, the nature of the disease would have been recognised at the beginning. Maclean records an instance in which the intestinal disorder consequent upon the recession of the eruption of measles was treated for dysentery. The absence, in this case, of the dysenteric odour in the evacuations, suggested a doubt as to the accuracy of the diagnosis. The patient having been placed in a warm bath, the rash of measles soon appeared, and the patient recovered.

Rectitis, which may be a sequel of acute dysentery or a primary disease, may be mistaken for dysentery unless attention be paid to the general symptoms and to the character of the stools. The patient passes healthy motions once or twice a day, according to his habit; but along with and also independently of these he passes blood, mucus, and pus frequently, and with more or less straining. The patient's health may otherwise be good; and in this respect a sufferer from rectitis presents a marked contrast to one labouring under acute or chronic dysentery. This is the "tenesmus" of Celsus, which he thus describes; and his description is as good as any that can be given: "In hoc æque atque in torminibus (or dysentery) frequens desidendi cupiditas est; æque dolor, ubi aliquid excernitur. Descendunt autem pituitæ mucisque similia, interdum etiam leviter subcruenta: sed his interponuntur nonnunquam ex cibo quoque recte coacta." It is this alternation of healthy with dysenteric stools which should lead us to suspect rectitis. This disease is chronic, and unless recognised and suitably treated will go on interminably. On examining the lower part of the rectum by means of the speculum, swelling, redness, and ulceration of the bowel will be detected. Rectitis readily yields to local applications—emollient enemata, followed by astringent injections, or touching the ulcers with nitrate of silver, constipation being obviated by the mildest laxatives.

The chronic form of dysentery may be, and often is, mistaken for diarrhœa. The presence of tenesmus, of pain on pressure, along the tract of the colon, and of mucus in the stools, are characteristic of dysentery. For the differential diagnosis of tropical diarrhœa, the reader is referred to Chapter XV.

The symptoms of Bilharzia disease when localised in the intestinal canal may resemble those of dysentery. For the

diagnosis of this condition, the reader is referred to the article on this subject by Sonsino.

The diagnosis of the particular form of the disease, and of the extent of the intestinal lesions, is determined by the symptoms and by the character of the stools. The distinction between the simple and gangrenous forms may be made by the odour of the motions, apart from the presence or absence of sloughs, although these should always be sought for and carefully examined. The amœbic form, as we have already stated, is to be recognised by means of the microscope; the malarial complication mainly by the history of the case, aided by the thermometer. The symptoms of typhoid, scorbutic, and rheumatic complications cannot be overlooked or mistaken.

Mortality and Prognosis.

Mortality.—The dysenteric death-rate in the army of India for the five years 1871–75 was 3·33 per 100 treated. In the two military hospitals of Martinique the mortality from dysentery was 2·8 per cent. for the period 1852–72. This low case-mortality is deserving of remark, inasmuch as the ipecacuanha treatment, as we understand it, is not generally adopted by French physicians. It must, however, be remembered that the severity of the disease differs in different countries, and in the same country in different seasons. Then there are considerable differences in the strictness of diagnosis. Some practitioners include severe forms of diarrhœa in their list of dysenteric affections. All this should make us cautious in the application of the statistical method in deciding the comparative merits of different modes of treatment.

In general hospitals the mortality is much higher than among the troops. Of 68 Europeans treated for dysentery in 1879 in the Medical College Hospital, Calcutta, 14—a ratio of 22·2 per cent. —died. The proportion of deaths to the cases treated among the natives, in the same institution, and in the same year, was 37·5 per 100. In the civil hospital of Mauritius, out of 202 dysenteric patients treated there in 1888,—mostly Creoles and Indians,—45 died, a ratio of 22·7 per 100. In the civil hospitals of Ceylon, 1217 patients were treated for dysentery in 1885; and of these 372, or about 30 per cent., died. It will readily be understood why the disease should prove so much more fatal in a general hospital, receiving all classes of patients, than in a military hospital, in which the patients are picked men in the vigour of life, and who are brought under treatment and proper regimen as soon as the earliest symptoms make their appearance. In general

hospitals many of the patients are worn out by misery or disease, and come under treatment at an advanced stage of the malady; some of them are at the extremes of life, when dysentery is more dangerous. The high mortality in general hospitals shows that dysentery is a dangerous disease, especially in cachectic and intemperate subjects, when not promptly treated.

Prognosis.—The prognosis in a particular case will be determined (*a*) by conditions peculiar to the individual, (*b*) by the form and complications of the disease, (*c*) and by the presence or absence of certain symptoms.

(*a*) The influences of age, constitution, and habits on the prognosis have been already stated; we may just add, that the puerperal state increases the danger of a dysenteric attack.

(*b*) The simple form of the disease, unless neglected in its early stage, is seldom fatal. Under proper regimen it would probably, in most instances, end favourably without the use of drugs; otherwise those submitted to homœopathic treatment would generally die, which they do not. In this form even bad treatment, such as repeated bleedings, does not necessarily prevent recovery. The danger lies in the fact that simple dysentery may pass into the gangrenous form.

When the stools present a distinctly gangrenous odour the prognosis should be guarded, even if no sloughs are detected in the stools. If sloughs are present, their prognostic significance depends upon whether they are superficial or deep, small or extensive, infiltrated with pus or otherwise. Thin grey or black sloughs—not infiltrated with pus, especially if they present traces of the intestinal tubules, indicating gangrene limited to the mucous coat—are of the least unfavourable import. They become more ominous in proportion to their size and number. Thick, black or fleshy sloughs, involving the sub-mucous coat, indicate a serious type of the disease, and the more so if they are infiltrated with pus or blood.

Chevers says that the black or coffee-coloured slough, resembling "an old sooty cobweb rolled together," supposed to consist of the areolar tissue of the sub-mucous coat, is a sign of inevitable death.

Algid, choleraic, and typhoid symptoms, although occasionally recovered from, justify a very unfavourable prognosis.

The prognosis in amœbic dysentery is grave in proportion to the severity of the symptoms, the number of amœbæ in the stools, and the duration of the disease. The appearance of gangrene is, perhaps, more unfavourable in this than in other forms of dysentery.

(*c*) The special symptoms which portend a fatal issue are a fall in

the temperature, with coldness of the extremities, and the appearance of the *facies Hippocratica*, or a subsidence of tormina and tenesmus along with collapse. Hiccup occurring at an advanced stage of the disease, constant and severe vomiting, paralysis of the sphincter ani, dry, black, or aphthous state of the mouth, fever and delirium, and convulsions in children, are justly looked upon as dangerous symptoms. Hiccup at the commencement of the disease is not a dangerous symptom.

If the strength of the patient is maintained, if the temperature is neither much below nor much above the normal, and if the pulse is regular and firm, recovery may be anticipated. A return of feculence in the stools is one of the most reliable signs of a favourable turn in the disease. The prognosis of chronic dysentery depends upon the severity of the symptoms, the duration of the disease, and the extent to which the health of the patient has been compromised.

Prophylaxis.

The prophylaxis of dysentery is to be drawn from its etiology. We have seen that it occasionally spreads in hospitals where large numbers suffering from the complaint are under treatment. This indicates the necessity of ample cubic space and of free ventilation, and of the prompt and thorough disinfection of the stools, which should never be thrown into a common privy. The bed-pans, commodes, and clyster pipes used by dysenteric patients should be carefully disinfected before being used by others. The infection may be spread by the soil of a camp becoming contaminated with fæcal matters. Prophylaxis demands that the contents of the latrines be disinfected and buried in deep trenches at a distance from the camp and human habitations, or, what is better, that they be subjected to combustion. Considering the dangers arising from the present system of disposing of excreta in camps, it is deserving of consideration whether all excreta should not be burned. The risk of diffusing typhoid fever as well as dysentery would thus be diminished. The only objection that could be raised is on the score of expense. This should not be viewed as an insuperable difficulty; at many stations fuel could be obtained at a reasonable cost. Dysentery has broken out amongst men using drinking water contaminated with fæcal discharges, and there is reason to suspect that water otherwise impure from the presence of organic animal or vegetable matters may predispose to dysentery or prove the vehicle of infection. These considerations point to the extreme importance of protecting drinking water from accidental contamination, and the

prudence of using boiled water when its source is open to suspicion. It is probable that many African travellers who have succumbed to dysentery might have escaped the disease if they had adopted the simple precaution of using boiled water only.

Finally, chills after being overheated, sleeping on damp ground, a deficient or imperfect diet, are important predisposing causes of dysentery. Such measures as prudence and foresight can devise should be taken to avoid exposing individuals or bodies of men to the operation of these agencies.

Treatment.

Acute Dysentery.—The history of the treatment of dysentery illustrates better than that of most diseases the truth of the Hippocratic aphorism, "*Experience is fallacious, and judgment difficult.*" Not only have certain remedies, such as arsenic and tartar emetic, formerly trusted in, dropped out of use, and the value of others, such as opium, been variously appreciated by individual observers, but the methods of treatment followed by the most experienced and sagacious physicians of the past, and from which they believed that they obtained the best results, are now regarded, not only as useless, but as positively harmful.

In comparing the results of the practice of to-day with that of a not very remote past, we are apt, however, to overlook the vastly improved hygienic conditions under which civilians, soldiers, and sailors in tropical countries are now placed, compared to those that obtained fifty or even thirty years ago. Dysentery in modern times is not only much less prevalent, but it is also much less severe than it was formerly. During the five years 1825–29, the admissions for dysentery formed 49 per cent. of the total for all diseases in the military hospitals at Martinique; while in the five years ending 1877, the ratio had fallen to 18 per cent. Along with this decrease in its prevalence, dysentery has assumed a milder type, or, what amounts to the same thing, the patients are now less frequently, than was formerly the case, the subjects of the scorbutic and malarial cachexias which render the disease so formidable and fatal. The soldiers are also less enfeebled in constitution by a prolonged residence in the tropics since the period of foreign service has been reduced. While we appeal, and justly, to the statistics of late years as a proof of the superiority of present methods of treatment over those followed by our predecessors, it is only fair to acknowledge that other circumstances besides treatment have very largely contributed to reduce the mortality from dysentery. That this is so is evident

from the very favourable results now obtained in French military hospitals, in which the ipecacuanha treatment is not generally practised.

Medical theory necessarily affects medical practice. The treatment of dysentery has been influenced by the pathological doctrines prevailing at different periods, although favourite modes of treatment have often outlived the theories upon which they were based.

Sydenham, who regarded dysentery as a fever "turned inwardly on the bowels," grounded his treatment, which in his hands was doubtless a successful one, upon this theory of the disease. "The indications," he says, "were plain, and I had nothing else to do but to cause a revulsion of the acrid humours by venesection, by which I tempered the remaining volume of the blood, and then to draw off the aforesaid humours by purging." The purgatives were followed by an anodyne of 16 to 18 drops of laudanum administered in a cordial. This method of treatment is essentially that which was followed during several succeeding generations.

When the doctrine changed, and dysentery came to be looked upon as a simple inflammation of the large intestines, the practice of bloodletting—general and local—received a new impulse; but now it was used with the intention of subduing inflammation, and not with that of freeing the blood of "peccant humours." This practice was certainly in many instances carried to an excess. The following case, quoted by Chevers from an old record, shows the results of a treatment which was, perhaps, only too frequently employed in the earlier part of this century:—

January 1*st*.—Ill three days. Fiat V. S. ad deliq. Evening.—Blister to epigastrium.

2*nd*.—Eighteen ounces of blood drawn, causing syncope; pulse 100, feeble; fifteen leeches to anus.

3*rd*.—Of opinion that the colon is, and was on admission, in a disorganised (almost gangrenous) state; a most unfavourable prognosis. One measure of wine, one pint of milk and sago; spoon diet. *Vesp*. Died. The colon, from cæcum to anus, gangrenous.

Calomel, as an auxiliary to bloodletting, given in scruple doses, was largely used in this disease on account of its purgative and supposed antiphlogistic properties towards the end of the first quarter of the century. It came to be looked upon as a remedy which in itself was capable of fulfilling most of the indications derived from the disease.

This is how Johnson speaks of its action in dysentery: "It does more to resolve irritative fever, to equalise the circulation, disgorge capillary vessels, restore the balance of the nervous power, and open

the sluices of the various healthy secretions and excretions, than any other remedy with which I am acquainted."[1] Under the influence of Annesley and others, bloodletting and mercury were used with greater caution.

The principles of treatment laid down by Morehead in 1860 remained very much the same as those enunciated by Annesley in 1840, but their application had become somewhat modified in certain particulars. General bloodletting, he says, may be used with advantage, within the first two or three days of the attack, in the case of Europeans of good constitution not long resident in India, and unaffected by climatic and malarious influences. The benefit of general bloodletting being, in such cases, maintained and increased by the subsequent application of leeches. He recommends local depletion when general bloodletting was unadvisable.

Mercurial treatment with a view of bringing the system under the influence of the drug was condemned, but calomel in doses of ten grains, combined with ipecacuanha and opium in doses of a grain and a half or two grains each, was given at bedtime, and followed by castor oil next morning. This combination was repeated once or oftener during the first days of the disease, with the view of increasing the action of the liver and of the mucous membrane of the small intestines. Morehead, while condemning the routine system of treatment by scruple doses of calomel, placed considerable faith in the efficacy of mercury. After the use of bloodletting and calomel in the manner described, ipecacuanha in doses of three to six grains was used in the early stages of the disease in combination with two to five grains of blue pill, and this was given every three, four, six, or eight hours. This treatment was continued steadily till amendment took place, but not sufficiently long to give rise to salivation. When the patient was cachectic the blue pill was omitted. If the secretions were scanty, the abdomen full, and the tongue coated, opium was looked upon as contra-indicated; but if the discharges under the use of the ipecacuanha and blue pill were free and frequent, the tenesmus distressing, and the abdomen soft, a grain or a grain and a half of opium was added to the medicine. Blisters to the abdomen were not approved of. He recommended fomentations, carefully used, as aiding materially the more important measures. The use of the warm bath he regarded as harmful rather than beneficial. This is the final form in which the antiphlogistic treatment of dysentery was practised and expounded in India.

As we are ignorant of the cause of dysentery, our treatment

[1] *Op. cit.* p. 235.

cannot be directed to its removal. This remark is not intended, however, to apply to amœbic dysentery, the treatment of which will be afterwards considered. No more, as we have seen, can we find safe indications for treatment in theoretical views respecting its febrile, inflammatory, or spasmodic nature. The more obvious symptomatic indications are to allay pain, to soothe the irritation of the diseased mucous membrane, to support the strength of the patient by nourishment furnishing little fæcal residue, and to combat complications by appropriate remedies.

These symptomatic indications should never be neglected; but the principal methods of treatment now adopted, viz. by large and repeated doses of ipecacuanha and by saline purgatives, the latter method so successfully employed by French physicians, are not founded on indications derived from the cause, from the disease, or from the symptoms, but are purely empirical. They are found to furnish better results than those more heroic measures approved of by our predecessors. The *rationale* of their action is quite a secondary matter.

Ipecacuanha was first described and figured by Piso in 1649, who gave an account of its anti-dysenteric properties. In 1686, Jean-Adrien Helvetius, who had studied at Leyden, but who had repaired to Paris in order to sell, it is said, certain nostrums of his father's composition, obtained possession of a quantity of this drug, which had been recently imported from Brazil by a physician named Legros. Helvetius, who kept his remedy secret, was so successful in his treatment of dysentery by the use of large doses of ipecacuanha that his reputation spread rapidly throughout the city. The Dauphin, son of Louis XIV., happened at this time to be taken ill of dysentery, and was successfully treated with this remedy. After its virtues had been further tested in the Hôtel Dieu, Helvetius obtained a reward of one thousand Louis d'or for his secret. The value of ipecacuanha in the treatment of dysentery soon became widely known, but it appears to have been used rather as an auxiliary to other remedies than as the sole or principal means of treatment. When used in large doses it was for the purpose of obtaining its emetic action; when this was not desired it was generally prescribed in small doses.

About the beginning of the present century, Balmain, and afterwards Playfair, adopted, or rather, we may say, rediscovered, the method of administering it in large doses, giving it in combination with laudanum. The former frequently gave ipecacuanha to the extent of two drachms, along with sixty drops of the tincture of opium, and found that in many cases a dose or two was sufficient to remove

every dangerous symptom.[1] This mode of treatment, although it attracted some attention at the time, did not secure the confidence of the profession. Annesley says that the use of ipecacuanha in large doses was frequently followed by benefit in the simpler cases, but in the more serious cases little advantage was to be derived from it beyond its action as an emetic.

It is to Docker that we are indebted for having reintroduced the use of ipecacuanha in large doses, not as an auxiliary, but as the main part of the treatment. His is the merit of having re-established the claim of ipecacuanha to be considered the *radix anti-dysenterica*. It is the method of administering it recommended by him that is now adopted in India and in most other tropical countries, and which we shall now describe.[2]

The great secret in the treatment of dysentery is to attack the disease early. If the patient has been constipated for some time before the onset of the dysenteric symptoms, or if he passes small, hard lumps of fæces, the scybala of which we read so much and see so little, it may be well to begin the treatment with a dose of castor oil, or some other mild purgative. If, on the contrary, the disease has been preceded by diarrhœa, this will not be necessary. The patient should be sent to bed. As Celsus says, "Oportet in primis conquiescere, siquidem omnis agitatio exulcerat." Next, a mustard plaster should be placed over the pit of the stomach, and after a short time, from twenty to sixty grains of ipecacuanha in the form of a bolus, or in pills, are to be administered. The object of the sinapism is to prevent the ipecacuanha from being rejected by vomiting. With the same object, twenty to thirty minims of the tincture of opium is often prescribed half an hour before giving the ipecacuanha. If there has been much bilious disorder before the onset of the disease, indicated by nausea, bilious diarrhœa, and a coated tongue, vomiting, in moderation, will do no harm, and may even do good. As a rule, therefore, it is better to give the first dose of the drug without the addition of opium—running the risk of, without soliciting, its emetic action. Strict recumbency should be insisted on, and no liquids or food are to be given for three or four hours after the medicine has been taken. With these precautions there is more or less nausea, but severe vomiting is not of frequent occurrence. It often happens that the slight retching brings up gastric mucosities without expelling the medicine.

Should the ipecacuanha be retained for an hour or more, its therapeutic effect will not be lost even should the greater part of it

[1] *Mem. of Medical Society of London*, vol. v. p. 216.
[2] *Lancet*, July 1858.

be then rejected. If the medicine be brought up a short time after it has been taken, the dose will have to be repeated as soon as the stomach is settled, all necessary precautions in the way of premising an opiate, and the application of mustard externally, being taken. This procedure is to be employed morning and evening in mild cases; but in the more severe forms of the disease it will be advisable to repeat the ipecacuanha every eight hours, the intervals between the doses being judiciously utilised for the administration of food.

This treatment should be persisted in till the tormina and tenesmus have ceased, and the stools have become feculent and bilious. In the mildest cases one or two doses will entirely change the aspect of affairs; in more severe cases the remedy may have to be persevered in for several days in succession. Poultices may be used in some cases with advantage; but they often annoy the patient after a time by their weight, and, unless carefully managed, they may do harm by wetting the bed-clothes. A flannel wrung out of hot water, placed over the abdomen, and covered with gutta-percha, will answer every purpose, and is not liable to the objections urged against the use of poultices.

When the stools, although normal, are too frequent, bismuth alone, or combined with small doses of Dover's powder, will usually complete the cure. Opium administered *per rectum* is useful in allaying spasm and irritation. A good combination is—bismuth, 120 grains; powdered opium, 1 to 2 grains; gum arabic, 60 grains; water, 2 oz. The severe tormina and tenesmus are often relieved by this means, and the patient's condition made much more tolerable. This rectal administration of opium finds its most useful application in dysentery occurring in pregnant women; the risk of abortion is thereby diminished. The warm bath is often a useful adjunct to the treatment. Given at the very beginning of the disease, it sometimes produces a marked improvement in the state of the patient. At a later stage, again, when tenesmus is urgent, or when there is dysuria or strangury, warm baths may be resorted to with great advantage. No doubt there is a danger of the patient "catching cold" if the greatest care be not taken in their use. The bath should be brought to the bedside, the patient should be protected from cold while in the bath and while being dried, and should be immediately placed between blankets, the abdomen being subsequently swathed in flannel. It may be necessary to say that the bed-pan, properly warmed, should be used, to prevent the patient getting out of bed to the night stool.

Such is the method of treatment to be followed in all cases of

simple dysentery,—a method which, begun at an early stage, when the disease is uncomplicated, and when the remedy is tolerated, leaves little to be desired. It is unnecessary to discuss here the various explanations that have been advanced of the therapeutic action of ipecacuanha. Whether it acts as a nauseant, as a diaphoretic, as a laxative, as a special stimulant to the liver, pancreas, and intestinal canal, or in all of these ways, are questions of greater speculative interest than practical importance.

The treatment of dysentery by means of saline purgatives demands attention, inasmuch as the results obtained by this method are highly favourable. The saline treatment is one that may be safely adopted in all mild cases of the disease, and is invaluable when ipecacuanha cannot be supported.

The sulphate of sodium is the salt which is to be selected, but the sulphate of magnesium, although somewhat more irritating and nauseous, answers very well. An ounce of the sulphate of sodium, dissolved in eight or ten ounces of water, may be given in a single dose or in divided doses. Perhaps it is better to give half an ounce at once, and then dissolve the other half in four ounces of water, and give it in doses of two tablespoonfuls, every half-hour or hour, until the bowels are freely opened. The purgative effects of this medicine commence within four hours and are over in twelve hours, so that, if taken in the morning, the patient's night is not disturbed by its untimely action. The medicine is repeated daily in the same or in larger or smaller doses, according to its action on the bowels and its effects on the disease. Trousseau says that the administration of the neutral salts in purgative doses once daily, or morning and evening, so as to induce diarrhœa, is the best treatment for dysentery. We confess that we have not given it a sufficient trial to entitle us to speak with confidence upon the subject, but we think that in all but the mildest cases it is safer to use ipecacuanha.

Dr. Leahy, of the Bengal Medical Service, has recently called attention to the success he has obtained in the treatment of dysentery by the use of sulphate of magnesium, with the addition of dilute sulphuric acid, so as to act gently on the bowels. Out of ninety-five patients treated in this way, and these belonging to the poorer classes, only three died, and two of them were moribund on admission.[1]

Ipecacuanha may be given to children either alone or in combination with bicarbonate of sodium, in doses suitable to their age; and in many cases it is well supported. If the young patient does not take the powder readily, the wine may be given in syrup.

[1] *Lancet*, October 4, 1890.

Birch advises the use of castor oil *emulsion* in the case of children, after having given a dose of the common oil to evacuate the contents of the bowel of all offending matters. "In a couple of days the motions will lose their slimy, bloody, and curdy appearance, and, only a little looseness remaining, the case is resolved into one of the simplest forms of diarrhœa." In some forms of bowel complaint in children, with frequent slimy stools containing blood, and passed with straining, one of us has found much good from the use of the bichloride of mercury given in the manner recommended by Ringer, viz. one grain of the bichloride dissolved in half a pint of water, and a teaspoonful given every hour. Whether these cases are etiologically and pathologically the same as true dysentery is perhaps doubtful, but they are very common in tropical countries, and yield readily to this treatment.

If the disease has been primarily of the gangrenous form, that is, has rapidly run on to sphacelation; or if the gangrene has developed at a later stage, and before coming under treatment; or if, as sometimes happens, the disease is running on to gangrene in spite of treatment, what is to be done? In the second of these contingencies, in which ipecacuanha has not been tried before the gangrene has set in, it should be prescribed at once and persevered in for a reasonable time, unless signs of collapse appear, which would contra-indicate its use. The same procedure is advisable when the motions have a gangrenous odour at an early stage of the disease, and before the ipecacuanha treatment has had time to manifest its effects. The gangrene is in many cases limited to circumscribed portions of the mucous membrane, and although the existence of gangrene is in itself serious, the case is never hopeless as long as the patient's strength is maintained. The treatment by ipecacuanha should not therefore be suspended until it has had a fair trial, for under its use many recoveries take place after the stools have become of a gangrenous odour, and after sloughs have been passed for some time. In these grave forms of the disease, as in scorbutic dysentery, the internal use of the oil of turpentine in drachm doses, combined with one and a half or two drachms of castor oil, daily or every other day, or in smaller doses (say of twenty to thirty drops) by itself, at intervals of two, three, or four hours, is sometimes followed by good results. Turpentine stupes applied to the abdomen so as to produce redness of the surface are also of service. If there is reason for believing that the gangrene is chiefly located in the rectum and descending colon, emollient and antiseptic enemata are indicated. The amount injected should never be sufficient to distend the bowel immoderately or to cause pain. Ten to fifteen

ounces will generally suffice. Infusion of linseed is as good an emollient enema for washing out the lower bowel, for removing putrilage, and for soothing the parts, as any that can be employed. Enemata of warm water, to which permanganate of potassium or carbolic acid has been added, are indicated when the presence in the stools of black sloughs and offensive débris warns us of the risk of septic absorption. If carbolic acid is selected, it should not be forgotten that it may be absorbed and give rise to poisonous symptoms; it should therefore be used in moderate quantities.

The most perplexing cases are those in which the ipecacuanha treatment has been fully carried out from the beginning and without advantage; although no special complication may be detected which would explain its failure. If in these circumstances the disease is making headway and threatening a fatal issue, there is evidently no reason for persisting in the use of ipecacuanha. Such cases are rare, yet they do occur. Speaking for himself alone, the editor would counsel a trial of small doses of calomel before extensive gangrene, which contra-indicates the treatment, has set in. One grain of calomel, combined with two grains of ipecacuanha, and a quarter of a grain of opium, repeated every hour until five or six doses have been taken, and then, every two hours during the day, should be employed. This treatment is not recommended on theoretical grounds, for theory is adverse to the use of mercury in any form of the disease, especially when gangrene is threatened; but solely on the good results that have followed in some unpromising cases the use of this remedy after other means had proved ineffectual. Used in this way, calomel will not give rise to salivation, even when continued for two or three days, and it is seldom necessary to continue its use longer. As soon as a change in the character of the motions is observed, the medicine should be given at longer intervals, or replaced by ipecacuanha. While this course of treatment is being carried out an enema of an infusion of ipecacuanha, two drachms in ten ounces of water, should be given once or twice a day, if the discharge from the bowels is scanty. Administered in this way, ipecacuanha generally produces nausea and procures free evacuation of the bowels. It will be understood that this treatment is neither to be adopted as a *dernier ressort*, nor too hastily, before the ordinary remedies have failed.

Algid and choleraic symptoms indicate the use of means calculated to restore the peripheral circulation, to sustain the heart's action, and maintain the strength of the patient.

In *amœbic dysentery*, while ipecacuanha should be resorted to at the beginning of the disease, its use should be conjoined with enemata

of quinine, $\frac{1}{5000}$, $\frac{1}{2500}$, or $\frac{1}{1000}$, as recommended by Councilman and Lafleur. Injections *per rectum* of solutions of the bichloride of mercury, of a strength of $\frac{1}{5000}$ or $\frac{1}{3000}$, half a litre of this solution being injected twice or thrice daily, were employed in two cases; but bichloride enemata should be employed very weak, and with great caution. Fifteen to thirty ounces of fluid should be injected at one time, so as to bring the remedy in contact with the diseased surface.

In the *gangrenous rectitis*, which we have described as occurring in an epidemic form in South America and elsewhere, repeated enemata of water acidulated with lemon juice have been recommended, and used with good results. As the disease is at first generally limited to the rectum, local applications seem specially indicated. At the same time, the action of the bowels should be maintained by gentle purgatives; and if the motions are dysenteric in character, ipecacuanha should be given.

Malarial fever is the disease which is most commonly associated with dysentery. When severe febrile attacks occur periodically along with dysentery, it will be necessary to give quinine in full doses alternately with ipecacuanha. It occasionally happens that the malarial infection manifests itself in determining periodic hæmorrhages from the bowels in those suffering from dysentery. This form of hæmorrhagic dysentery usually yields to the administration of quinine. If this should fail, recourse is to be had to ergotin. It was found that the dysentery which attacked the troops in Mauritius, who were broken down by fever during the epidemy of 1867, was not benefited by ipecacuanha. The remedy which was found to be most effectual in this instance was the tincture of the perchloride of iron in large doses. The perchloride and pernitrate of iron with quinine are useful adjuvants in dysentery occurring in subjects suffering from the malarial cachexia, even if no febrile symptoms are present. Removal from the malarious locality should, if possible, be effected.

Common continued fever as a complication of dysentery does not demand special treatment. If the fever, however, is high, twenty to thirty grains of antipyrin may be of service in reducing the temperature.

Scorbutic symptoms in connection with dysentery call for the free use of fresh lemon or lime juice. Grapes, oranges, pomegranates, or guavas may be used if limes and lemons are not to be had. The patient should be warned to reject the skins and stones of the fruit, as fatal results have followed the ingestion of grape stones. Maclean speaks highly of the employment of the fresh Bael fruit in

dysentery complicated with scurvy. He considers it to be most useful when given in the form of a sherbet. The patient should be put upon a milk diet, which in this form of the disease is to be looked upon as an essential part of the treatment. In scorbutic dysentery the motions frequently contain much dark, liquid blood. In this condition the solution of the pernitrate of iron may be given. If this fail, the oil of turpentine in the dose of fifteen to thirty minims, in almond emulsion, three or four times a day, will often succeed in diminishing the hæmorrhage and improving the general condition of the patient. If a laxative be required, the oil of turpentine may be administered in drachm doses conjoined with two or three drachms of castor oil.

So much for the general treatment called for by the scorbutic taint. For the rest, it may be said that in this form the motions are more abundant than in simple dysentery; and if ipecacuanha be given, it will often be necessary to conjoin it with small doses of opium.

Rheumatism, or rheumatoid symptoms, appearing during the course of dysentery, are seldom so severe as to necessitate the suspension or modification of the usual treatment. Local applications to relieve the pain may be employed until the dysentery has been subdued, when the joint disease should be treated in the usual way.

Septicæmia, arising from the absorption of infective substances from the diseased bowel, gives rise to what are called typhoid symptoms. Typhoid or malignant dysentery is thus always associated with gangrene, limited or diffuse, of the large intestine. The treatment will be that of gangrenous dysentery; but we have here an urgent indication to meet, viz. to remove and neutralise the septic matters contained in the bowel by means of large enemata of warm water, containing the permanganate of potassium, carbolic acid, or oil of turpentine. If the fever is high, quinine in five to ten grains, dissolved in dilute sulphuric acid, should be given two or three times a day, and the strength of the patient maintained by a suitable diet. Abscesses are to be opened and treated on antiseptic principles. When dysentery is associated with *typhoid* or *typhus* fever, the treatment will vary according as the symptoms of the one or other disease predominate, and according to the general condition of the patient.

Functional disorders of the liver, in other words, bilious symptoms, more or less severe, accompanying dysentery, constitute what was formerly termed "bilious" dysentery. This complication does not demand any modification of the ordinary treatment. The emetic and purgative action of the ipecacuanha relieves the liver at the same time as it cures the dysentery. Nor does the coexistence of hepatitis, so long as an abscess has not actually formed, contra-

indicate the use of ipecacuanha in large doses. Some authorities, indeed, contend that this drug is nearly as efficacious in hepatitis as in dysentery. It is in such cases that the application of fifteen to twenty leeches to the anus often proves of signal service both as regards the liver and bowel symptoms. In some cases of this nature, ipecacuanha gives rise to troublesome or unmanageable vomiting, and fails to act efficiently on the intestine. The ipecacuanha treatment must then be supplemented or replaced by the use of salines. When abscess of the liver and dysentery coexist, only harm can be expected from a treatment which may give rise to depressing vomiting. The treatment of the dysentery then becomes subordinated to that of the liver affection.

In severe hæmorrhage arising from the accidental erosion of a vessel in the intestinal wall, indicated by the copious discharge of clotted blood, ergotin may be employed hypodermically, or in enema.

When the patient is infested with round or thread worms, it will be advantageous to get rid of them at once by means of santonin. When the more troublesome complication of tænia is present, its early expulsion by the tannate of pelletierin or of some other appropriate remedy should be attempted.

The paralyses which may accompany, but which more generally follow dysentery, are associated with a state of anæmia and debility that indicates the necessity of a tonic treatment. Iron and quinine, along with a nourishing diet, will, therefore, form the principal part of treatment. Should recovery be delayed, recourse may be had to the constant or interrupted current.

Chronic Dysentery.—Experience has shown that the treatment of chronic dysentery in Europeans is very unsatisfactory so long as they remain in the tropics. Much is to be hoped for from a change to a temperate climate, provided it be effected early. It may be laid down as a general rule, that the longer the change is delayed, the less will be the prospect of a speedy and permanent recovery. It is therefore a matter of primary importance, in all cases where the patient's circumstances permit it, to insist upon a return to a temperate climate before the disease has induced extensive structural changes in the large intestine. The arrangements now made for the dieting and general comfort of invalids on board the best steamers, render it possible for patients suffering from dysentery to undertake the voyage, who could not have done so with any hope of advantage twenty or thirty years ago.

If it should unfortunately happen that the patient has to face the severity of an English winter or spring on his arrival, and this cannot always be avoided, great care should be taken that he is well

provided with warm clothing; and he should always wear flannel next the skin. In the case of Europeans who are unable to return home, and also in the case of natives, to whom such a change is out of the question, a short voyage in a well appointed vessel, or even a temporary visit to a seaside resort, may be of great advantage. A change to the hills is not to be recommended. The cold moist air of the hill sanitaria of India often proves very prejudicial in such cases; but there are probably to be found dry, equable, cool, and healthy hill stations in some tropical countries a change to which might be beneficial, at least during certain seasons. Considering how difficult it is for many residents in tropical countries to undertake a long and expensive voyage, it is desirable that climates suited to this class of patients, as well as for natives, should be sought for.

The treatment during the exacerbations which mark the disease, is to be conducted on the principles applicable to acute dysentery. The patient should be strictly confined to bed, and ipecacuanha should be administered in larger or smaller doses, at longer or shorter intervals, according to the urgency of the symptoms.

During the intervals the treatment must be directed to the conditions inferred to exist in the bowel, such as congestion, ulceration, general thickening with contraction or stricture, and with a due regard to the state of the patient's health. In a disease which lasts for months or years, prolonged confinement to bed is, of course, out of the question; but when the symptoms indicate that the large intestine is the seat of acute or subacute ulcerative processes, rest should be enjoined, although carriage exercise may, in many cases, be allowed.

If a scorbutic or malarial taint is present, the treatment appropriate to these conditions should never be neglected. If the patient is anæmic, while no acute inflammatory action is going on, the pernitrate of iron, quinine, and other tonics are indicated.

When ulceration is inferred to exist, constipation should be obviated by the mildest laxatives, and diarrhœa checked by Dover's powder and bismuth. When the ulcerative process is chronic, the oil of turpentine will often be serviceable. The use of the water belt or a wet compress over the abdomen, covered with gutta-percha, and retained in position with a flannel binder, is often grateful to the patient, and may be supposed to act favourably on the diseased mucous membrane, both by maintaining a moist and equable heat of the surface, and by the gentle support it affords and the pressure it maintains on the abdominal viscera. When the ulceration is limited to the rectum or sigmoid flexure, enemata, at first emollient and afterwards astringent.

should be cautiously used to promote cicatrisation; but even in these cases much harm may be done by the too frequent use of irritating injections. When the bowel is thickened and ulcerated, and the patient is liable to recrudescences of the disease, Fayrer recommends the employment of the Bael fruit, alone or in combination with Dover's powder or opium as the circumstances may require; and he believes that in such cases the Bael fruit will sometimes materially aid in restoring the diseased intestine to its normal condition.

If the calibre of the bowel is much diminished, either by general thickening and contraction or by cicatricial stricture, so as to impede the evacuation of the fæces, the abdomen above the stricture becomes tumid, and a sense of tension and of great discomfort is complained of. The patient is troubled with flatulent eructations, and the breath may have a feculent odour. Such evacuations as are passed are scanty, and voided with difficulty.

Little can be done when the disease has reached this stage to alleviate the sufferings of the patient beyond maintaining the fæces in a fluid state by means of gentle laxatives. Warm baths, gentle frictions to the abdomen, and emollient injections may help to promote the evacuation of the bowel.

Diet and Regimen.—The diet in all forms of dysentery is a matter of much importance. In the acute stage it is a safe rule never to allow solid animal food until the dysenteric process has completely subsided, and resort to the mildest farinaceous diet had better be deferred until a marked improvement in the symptoms has taken place. Milk, when it can be obtained fresh and pure, is the most suitable diet in acute cases. It is best given mixed with lime-water, as it is apt to cause colic if taken pure; and unsweetened condensed milk may be employed if fresh milk cannot be obtained. Good beef-tea, essence of beef, or strong chicken soup without pepper or spices, may be substituted if milk cannot be had or disagrees.

When the saline treatment is adopted, we have not to contend with nausea; and in this case the best diet, undoubtedly, is milk, which should be continued until recovery takes place. Milk is an essential part of the treatment of scorbutic dysentery. It is also recommended in the amœbic form of the disease. When it is not tolerated Councilman recommends that egg-albumen or clear soup be substituted for it. In the earlier stages of chronic dysentery, when the intestine is congested and more or less ulcerated, the patient should be restricted to a milk diet. In the more chronic stages, and during convalescence, if milk agrees with the patient, it should still form a principal part of the diet, but supplemented

with farinaceous food, eggs, fish, and fowl. Alcohol should be avoided, both in acute and chronic cases. In the latter, if a stimulant is really required, a sound Bordeaux, free from acid and of a certain astringency, should be prescribed.

During convalescence, and, indeed, throughout the whole course of the disease, the clothing of the patient should be warm. Great care should be taken to avoid chills, which are the frequent cause of relapses. A return to the tropics should be deferred until perfect recovery has taken place.

CHAPTER XVII.

TROPICAL DISEASES OF THE LIVER.

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It is a generally accepted opinion that residence in tropical and hot climates predisposes to affections of the liver, and that there are some forms of hepatic disease that are seldom met with except in such climates, or in people who have been exposed to such climatic influences. This opinion, though in a great measure true, is applicable only to the case of foreigners coming from temperate regions, and not to the natives of the countries themselves. These latter are not generally found to suffer more from liver diseases than do the dwellers in Europe or in any other temperate climate. Again, taking the case of India, where there are more temporary European residents, civil and military, than in any other country in or near the tropics, we find this liability to liver disease is seen only in certain classes of Europeans, as will be more fully noticed later on.

All forms of liver affections, both functional and organic, may be met with in India just as in cold climates, such as tumours; hydatids, and other parasites; fatty and lardaceous degenerations; cirrhosis from alcohol and other causes; passive congestion and enlargement due to obstruction of the circulation, as in some forms of heart and lung disease; abscess, the result of pyæmia or other form of septic poisoning; catarrhal affections of the gall-bladder and ducts; billiary calculi, and so on. It has been noticed, with reference to gall-stones, that they are less common in the tropics than in colder climates; but they do occur, and when present give rise to similar symptoms. Jaundice also may be set up by any of the usual causes. All such affections are, however, in no way specially the result of living in a hot climate or in malarious localities, but are met with equally in all countries, and they do not lie within the scope of this article. There are, at the same time, certain forms of liver disease that are very common in hot countries, and are undoubtedly due to the effects of climate

and to the mode of life and habits usually followed by Europeans residing in such countries.

The most frequent and most marked of these tropical affections of the liver are—

1. Acute congestion or inflammation of the substance of the liver, usually known as *hepatitis.*

2. Abscess of the liver; *tropical* abscess, as it may be termed. This may be the result of acute inflammation, or it may come on gradually in the course of chronic congestion without active symptoms.

3. Abscess of the liver, set up by pyæmia, or by septic absorption from unhealthy ulceration in the large intestine in cases of dysentery.

4. Chronic congestion and enlargement of the liver; which is frequently met with in Europeans in India, and is due to the effects of heat, of prolonged residence in a hot climate, combined with an over-stimulating diet.

5. Chronic enlargement and induration of the liver, the result of malaria and of attacks of malarial fever, and which often ends in waxy or lardaceous degeneration, or in fatty enlargement.

Hepatitis.

Etiology.—There are many causes at work in hot climates tending to the production of the several forms of tropical liver disease. The most potent of these is undoubtedly the climate, the effects of prolonged high temperature acting injuriously on the constitution of the natives of temperate regions, and having a special influence on the liver. At first the organ is stimulated, there is increased secretion and elimination of bile. We often find that Europeans are, during their first hot season or two in India, subject to feverish attacks accompanied by bilious diarrhœa, burning acrid stools, and general symptoms of liver derangement. After a time, however, the activity of the liver seems to become diminished, and its secreting power exhausted or perverted. We see the effects of this in the pale sallow complexions, constipation and irregularity of the bowels, with pasty motions and generally impaired digestion, a condition so often met with in old residents in India. Such symptoms as these are often followed by active congestion or acute hepatitis. In hot climates, again, the lessened activity of the lungs is another cause of increased action of the liver. As was shown by Dr. Parkes, the proportion of oxygen in the air varies inversely with the temperature; the higher the temperature the less the amount of oxygen taken in each inspiration. In hot climates, also, the rate of breathing is diminished, and consequently

the exhalation of carbonic acid by the lungs is less than in a cold climate, and so more work falls upon the liver. Such causes of liver disease are greatly intensified by defective ventilation, by overcrowding of men in barracks and guard-rooms, and by other insanitary conditions to which formerly little importance was attached. Residence in a hot climate also causes impaired nutrition and general blood deterioration, and increased destruction of the red corpuscles; and as the elimination of the products of their destruction takes place mainly in the liver, this leads to increased activity and increased formation of bile, followed by exhaustion and a tendency to disease. Such causes, however, seem to have little power in producing disease when people adopt habits suitable to the climate; but, as a rule, the Europeans in India and other hot climates commonly continue the habits they are accustomed to in cold climates, which are often unsuited to the new conditions. They are apt to take far more food and drink than the system requires; they take a diet too rich and stimulating, and especially containing too much animal food and too much alcohol, and this, combined with the heat of the climate and diminished bodily exercise, leads to chronic congestion and enlargement of the liver, or sets up acute inflammatory attacks. When people take a full meal of animal food two or three times a day, as is so commonly done, it overloads the blood with excess of nutritive material, and throws undue work on such secreting and excreting organs as the liver, which in health helps so largely in the oxygenation and destruction of the albuminous materials in the blood. A full meal, especially if combined with alcohol, will always produce temporary hyperæmia and swelling of the liver; and, if frequently repeated, the congestion and enlargement become permanent. We find that among European soldiers and their families, and indeed among all classes of Europeans in India, *men* suffer much more from liver disease than either *women* or *children*. As a rule, women are much more abstemious in respect to both food and drink than men are; and children, who generally live on much lighter diet and take no alcohol, hardly suffer at all from inflammation or abscess of the liver, although they are especially liable to dysentery and other forms of bowel complaint. In a hot climate the habitual use of alcohol in any form tends to induce liver disease, even when it is taken in what is usually considered strict moderation. It is not only those who drink freely who are liable to suffer, but the habitual daily consumption of beer, wine, or spirits, unless in very small quantity, will undoubtedly act as a predisposing cause. It is found that alcohol in combination with sugar, as in the form of beer, or the stronger wines, as port,

sherry, champagne, or brandy, are more injurious than the lighter wines, such as claret and hock, or perhaps than well diluted whisky. In some people hepatic disease is very easily set up, so that a slight or occasional indulgence in liquor in quantities that would generally be regarded as harmless, or perhaps as even conducive to health, will in them produce serious derangement of the liver. Some people suffer in the same way if, from any cause, they are deprived of their regular exercise, or change from an active to a sedentary mode of life.

Alcohol in any form should never be taken during the heat of the day, or until the principal meal in the evening. Beer at breakfast, or a peg at lunch or between meals, followed by exposure to heat and the sun, will almost always cause congestion of the liver; and then a chill to the surface, or other slight exciting cause, will set up inflammation. There is no doubt that a great deal of the illness among Europeans in hot climates is due to habits unsuitable to the climate, rather than to the climate itself. In a hot climate alcohol is in no way necessary to health, and as a rule people keep better who do not as a daily habit take it in any form. We seldom meet with cases of hepatitis or liver abscess among total abstainers, except the pyæmic form directly associated with dysentery; but moderate drinkers are liable to suffer. It has been shown very conclusively from the statistics of the Temperance Association among the European troops in India, that the sickness and mortality is far less among the temperance men, the total abstainers, than in the rest of the army. By their late published returns for 1889, in the Bengal Army there were 10,000 men enrolled as members of the Temperance Association out of a total of nearly 50,000. Among the abstainers, the number constantly sick was only 41 per mille, and the mortality from all causes 3 per mille, against 75 and 15 per mille respectively of the whole army. These figures show most strikingly the immense advantage to health of total abstinence in India—a reduction of mortality from 15 to 3 per 1000 men. One may hear it said that many of the men who are enrolled as abstainers occasionally break their rules and have a hard drink. If this be the case, it would tend to show that it is the habitual moderate use of alcohol that predisposes to disease rather than an occasional excess, and general experience points to the same conclusion. In the French settlements in India the prevalence of liver disease is much less than among the English, and we know that, as a rule, the French are much more abstemious than the English in respect both to meat and drink.

The returns of sickness and mortality of our European troops serving in India show the great prevalence of liver disease among

them. According to Bryden's tables, in the years 1871–76, the admissions for liver disease annually (hepatitis and abscess) were 55 per 1000 strength, and the deaths 2·6 per 1000, and the invaliding home 6·5 per 1000.

From returns of sickness among the European troops in Madras and Bombay in the early part of this century, collected by Ranald Martin, the admissions for liver disease were at the rate of 90 per mille annually.

In the year 1889, of European troops in India there were 24 admissions per 1000 strength, and 1·43 deaths per 1000, from liver disease.

These figures would not include a large number of the cases of chronic enlargement of the liver associated with malarial or other fevers, nor all the cases of abscess coming on during an attack of dysentery.

The improvement in respect to liver diseases that has taken place of late years among our troops serving in the tropics is very marked. They are much less prevalent and much less fatal than they were even a few years ago. The improvement also in this respect among officers and non-military residents is equally great. One main cause of this is, no doubt, that all classes drink less and are more abstemious than they were, even a generation ago. To some extent also early invaliding to Europe in cases of chronic disease among soldiers diminishes the sickness.

In the home army the sickness and mortality from liver diseases is far less than in India. The effect of free living in causing liver disease is less in cold than in hot climates, but even at home it is very marked.

From the Registrar-General's returns we find that the mortality from liver disease is six times greater among publicans than among other classes at corresponding ages.

The admissions into Netley Hospital of soldiers invalided from foreign stations, the greater part coming from India, show also the great prevalence of liver disease in that country.

During the five years 1867–71, there were 1231 admissions and 14 deaths from hepatitis and abscess, and in the five years 1887–91 there were 240 admissions and 4 deaths,—showing that among the men invalided also there has been of late years a greatly diminished amount of liver disease.

Deaths from liver disease, which used to be frequent at Netley, are now rare. This improvement is, no doubt, partly due to the invalids from abroad being sent home at an earlier period of disease than formerly, but chiefly to the diminished prevalence and severity of hepatic disease among the troops in India.

Exposure to the sun and to great heat, and also over-fatigue, may act both as predisposing and as exciting causes of liver inflammation; and no doubt one of the reasons why, in India, European women suffer less than men is that they are much less exposed to such influences. It is found also that men who lead active out-door lives, and at the same time live carefully, are far less liable to suffer from exposure to the sun or from great fatigue than are those who are more indolent and less abstemious. In the condition of chronic congestion and enlargement of the liver, so often met with in hot climates, an attack of hepatitis or abscess is easily set up by any slight *exciting* cause; and by far the commonest and most potent immediate cause is a chill to the surface, especially when heated and perspiring. A draught of cool air at night, when perspiring during sleep, is not an unfrequent cause. A cold bath will sometimes have the same effect. If the bath be taken when the body is heated by exercise and the circulation vigorous, even though perspiring freely, it seldom does harm; but if the surface be allowed to cool down, and the circulation has become languid, reaction does not readily follow, and the bath will then often set up hepatitis. If, however, the liver is in a sound condition, a cold bath seldom acts injuriously, and is in India as bracing and invigorating as at home.

Among the natives of India there is very little liver disease, although they suffer quite as much as Europeans do from such diseases as malarial fevers and dysentery. Mr. Waring used to consider the natives almost exempt; and, in his day, in the jails at Madras there were only 5 cases of hepatic disease among 10,000 convicts. In the native hospitals and dispensaries in Bengal there are very few admissions and deaths from hepatic disease. In the year 1889 there were only 0·1 deaths per 1000 in-patients treated.

Native troops also suffer very little. The following comparative table (1889) gives the relative prevalence of liver disease among different classes in India:—

PRESIDENCY OF BENGAL, PER 1000 STRENGTH. 1889.

	Admissions.	Deaths.
European Troops, . .	24·5	1·43
Native Troops, . . .	1·6	0·11
Jails, ...	1·5	0·21

Showing an enormous excess among Europeans.

The natives of India are very liable to dysentery, but they do not often get abscess of the liver, and the ordinary tropical abscess is very rare amongst them. There are certain classes of natives who indulge in various forms of fermented drinks, but at the same time they live chiefly on vegetable diet. No statistics are available for comparison between drinking and non-drinking natives. In the Mayo Native Hospital of Calcutta, which admits all classes of natives, there are many more admissions for hepatitis and abscess than in the country hospitals. The reason seems to be that, among the natives of Calcutta and other large towns, drinking habits are becoming very prevalent; and it is rare to find a case of abscess of the liver in a native in which there is no history of some kind of spirit-drinking. Among the wealthy and educated classes of natives of Calcutta and other Presidency towns, who with Western ideas of civilisation have to some extent adopted European habits, and have, perhaps, in many cases taken to the bad habits rather than the good, and who eat and drink much as Europeans do, hepatitis and liver abscess are not unfrequent; they are, indeed, nearly as prevalent as among Europeans; and there is no doubt that the indulgence in alcoholic drink, and in a richer and more nitrogenous diet, has been mainly the cause of this. With regard to the influence of age and sex, liver disease may occur at any age and in either sex; but, as already noticed, it is very unfrequent among European children, and is far less common in women than in men, for the reason previously referred to. Among men all ages are liable to suffer, but attacks of acute inflammation tending to abscess are most common among young men during their first year or two of residence in India. At a later period men are more liable to chronic congestion and enlargement, and to the more indolent forms of abscess.

It is found by experience that European troops suffer much more from liver diseases in India than in the West Indies and other hot countries; also, that there is a larger proportion of deaths from hepatitis and abscess in Madras than in either Bombay or Bengal.

In 1889 the deaths per 1000 strength from these diseases were —of troops in Madras, 2·28; in Bengal, 1·31; in Bombay, 1·08. The same excess in Madras has always been noticed. The reason of this is not quite obvious, and our present knowledge does not enable us to assign any very reliable cause for the difference. It may be because in the Madras command, which is chiefly in the tropics, the heat of climate is more continuous all the year round, and there is no compensating cold season such as we find further north. In such stations as Ceylon and the West Indies the heat is tempered all the year round by sea breezes. Jamaica has always

enjoyed a singular freedom from liver disease, except amongst the hard drinkers.

Where there is hepatitis, or a tendency to liver congestion, the hill stations and sanitaria in India, with their cool climates, are not found favourable. This, probably, is partly due to the liability to chills from the frequent and rapid changes from hot sunshine to cold winds. Also the higher altitudes, and consequently diminished supply of air and oxygen to the lungs, will throw more work on the liver, already weakened and predisposed to disease. Again, in the hill stations in India there is a great tendency to diarrhœa, which is so often accompanied by symptoms of liver derangement.

Pathology and Symptoms.—Acute hepatitis or inflammation of the parenchyma of the liver is one of the most common forms of liver disease of hot climates. At the commencement of an attack the liver enlarges; the swelling may be localised and limited to a portion only, or it may be general, and involve nearly its whole substance. At first the enlargement is mainly due to hyperæmia. In some cases it takes place very rapidly, so that in quite an early stage, if the inflammation be widely diffused, the liver swells enormously. More often the inflammation is more localised, and the swelling will be most marked in some particular direction, as upwards towards the right lung; or there may be fulness and bulging of the lower ribs of the right side, or in the intercostal spaces; or the swelling may be felt below the costal margin. The right lobe is the part most frequently affected. When a post-mortem examination is made in an early stage of an attack, the liver is found intensely congested and vascular; when cut into, the substance is of a deep red colour, and its consistence is soft, and it bleeds copiously. With this hyperæmic condition are mingled greyish spots or patches of softened tissue, from the cut surface of which serous fluid exudes; these patches being interspersed with, and surrounded by, the hyperæmic portions. The whole inflamed part may be surrounded by a firm layer of condensed liver tissue.

Subsequently, the inflamed patches become more softened, and of a yellowish colour, the outlines of the lobules of the gland are obliterated, and the secreting cells broken up and converted into a sort of granular débris containing oil globules and pigment molecules, leading to atrophy and destruction of the gland tissue. The capsule of the adjacent parts generally becomes rough and opaque, or its surface may be covered with lymph. As pointed out by Lauder Brunton, the liver is capable of rapid and enormous vascular distension; and its usual firm consistence, as seen on section, is a

post-mortem change. He shows that if the circulation through the portal vein in the fresh liver of an animal recently killed be kept up artificially, when the pressure of the fluid injected through the vein is increased, the liver will enlarge enormously, and will contract again quickly as the pressure is taken off. The liver can, in this way, be made to expand and contract like a sponge. The same thing occurs in a less degree when the hepatic artery is injected. This distensibility of the liver tissue explains the rapid enlargement of the organ that is often observed to take place in acute congestion, and sometimes as the result of a sudden attack of ague.

The symptoms of acute tropical hepatitis are generally well marked. The attack is often set up by a chill to the surface, or from an impression or sensation of cold, as from exposure to a cool draught when heated; and this is especially likely to occur during sleep, when the power of resistance is lessened. The chill that in a cool climate would be likely to cause bronchitis, pneumonia, or nephritis, would, in a hot climate, cause an attack of dysentery or hepatitis. An attack of ague, or other form of malarial fever, may give rise to liver inflammation. An error in diet, as a too abundant and stimulating meal, or too much alcohol, over-fatigue, or great exposure to sun and heat, will often do the same. Such exciting causes are most likely to set up an attack when the liver is predisposed by the condition of chronic congestion and enlargement previously referred to. The attack often commences suddenly with a sense of chill or a rigor, or it may be ushered in by a few days of previous malaise. The chill is quickly followed by rise of temperature. There is loss of appetite, often nausea and vomiting; the tongue is white and coated, and the bowels are confined, the constipation, perhaps, alternating with diarrhœa; and often there is slight jaundice. There is generally a feeling of pain and fulness in the right side, with some tenderness on percussion over the liver. There may be general enlargement of the liver, shown by increased dulness on percussion extending in all directions, or the right lobe only may be affected, and often the enlargement is circumscribed and limited to a portion only of the organ. On standing up the patient feels a weight like a "cannon ball," as they sometimes describe it, dragging in his right side. Usually he cannot lie on either side, particularly on the left side, without discomfort. Pain in the right shoulder, and running down the arm, is commonly present. This pain is of a peculiar dull, gnawing character, difficult to describe and localise. It depends on the connection of the phrenic nerve with branches of the brachial plexus. The breathing generally is hurried; a deep inspiration gives pain; and there is often

some dyspnœa, and a dry, teasing cough. In some cases, especially where the inflammation is near the surface, and when the capsule of the liver is involved, the pain in the side will be very severe; and when the diaphragmatic surface is implicated, there will be acute spasmodic pain, with cough, and difficulty of breathing. There is always a sense of great weakness and depression. As the attack runs on, the fever, which is frequently of an irregular remittent type, increases, the pain and tenderness get worse, and there is often spasmodic contraction and rigidity of one or both recti abdominis muscles. The jaundice may become more marked, though this symptom is often hardly more than faint yellowness of the surface. If such an attack runs on unchecked, it is very likely to lead to the formation of abscess. It is not unfrequent to meet with cases of hepatitis in which the symptoms are not well marked, and the diagnosis may be difficult and obscure; the fever may be very slight, and the pain, tenderness, and enlargement hardly noticeable; and this is likely to be the case when the inflammation is deeply seated in the substance of the liver. The symptoms may, in some of these cases, point chiefly to the lungs, such as dry, frequent cough, mucous expectoration, and some dyspnœa. Such cases may progress very insidiously, and may end in abscess.

The fibrous capsule may, in some cases, be the part chiefly affected. There is *perihepatitis*. This is usually associated with interstitial hepatitis, and is often due to syphilis; or it may be from extension of inflammation from some neighbouring structure, as the pleura; but it may arise as part of an attack of acute hepatitis. When the surface is affected, such symptoms as pain, tenderness, cough, and catch in the breathing are more pronounced, though there may be very little, if any, *enlargement* of the liver.

Cases 1 and 2 are cases of acute hepatitis.

Case 1.—Hepatitis, the result of Hot Climate and Intemperate Habits.

Private G. M., aged 28, service six and a half years, of which over five were passed abroad, chiefly in the Mediterranean and Egypt. Never had ague or dysentery; of rather intemperate habits. In the autumn of 1891, when at Cairo, he began to suffer from constipation, pain, and feeling of weight and fulness in the right hypochondrium, pain in the right shoulder, loss of appetite, bitter taste in the mouth, and sense of depression and inability for exertion. He did not at first go sick, but in November he was admitted into hospital suffering from hepatitis. The symptoms were not very acute, and he had no marked rigors, and the fever was never very high; but as he did not improve much under treatment he was invalided home, and arrived at Netley on the 11th April 1892. He was then slightly jaundiced, his

tongue covered with a creamy fur, bowels irregular but generally constipated, and the stools clay coloured. There was pain and tenderness over the liver, and pain in the right shoulder. The liver was enlarged, chiefly in the right axillary line, and below the costal margin on the right side.

For treatment he was put upon very careful diet, chiefly soup, milk, light puddings, and eggs, the food being varied from time to time. Chloride of ammonium in doses of 15 grains, with dilute hydrochloric acid, was given three times a day, occasional laxatives and saline purgatives. For the first four weeks there was not much improvement. After that he was given Vin. ipecac. with acid. nitro-muriatic, dil. and extract. cascaræ liquid., 20 minims of each, with glycerine and water, three times a day. This acted freely on his bowels, but without causing irritation.

He soon began to show marked improvement; the jaundice quite disappeared, the tongue became clean, and the bowels regular and motions healthy. The enlargement of the liver went down, and the pain and tenderness passed off, and he was able to take regular exercise.

On the 30th of May he was convalescent, and soon afterwards became fit to return to duty.

This was a simple case of inflammation, due to hot climate and free indulgence in alcohol, and not complicated by malaria or dysentery.

Case 2.—Congestion of the Liver leading to Acute Hepatitis, the effect of Hot Climate and Malarial Fever, and coming on after Dysentery.

Private J. O'H., aged 24, three years in India. In the hot weather of 1890 he began to suffer from pain and weight in the right side, and pain in the right shoulder, loss of appetite, nausea, and constipation, alternating with irritative diarrhœa. He then had an attack of dysentery, lasting nearly three weeks, from which he quite recovered. In November he began to suffer from ague, and continued to do so, on and off, for about six months, the hepatic symptoms continuing all the time. He then recovered sufficiently to resume duty; but in July 1891 he was again admitted into hospital for congestion of the liver, and after two or three weeks began to suffer from chills, occasional shivering, fever, and night sweats. He had pain and weight in the right side, and tenderness over the liver. His bowels were very irregular, and the stools at times dysenteric, and he was slightly jaundiced. He lost weight rapidly.

Under treatment the more active symptoms again passed off, and he was invalided home and admitted into Netley in March 1892. He was then pale and anæmic, but with slightly yellow skin and conjunctivæ. Tongue thickly furred. There was pain and tenderness over the liver, which was enlarged, chiefly in the mammary line, and pain behind the right shoulder. He had occasional nausea and vomiting, the bowels were irregular, and the stools at times loose and pale and offensive. He had occasional night sweats. Treatment: at first rest in bed with light, unstimulating diet. He was given occasional doses of euonymin and podophyllin, and salines and chloride of ammonium, with salicylate of soda, three times a day. Compresses of diluted nitro-muriatic acid were applied over the liver. For the first five or six weeks there was not much improvement, but the jaundice got less, and he gained a little in strength, and was able to be up and about. He was then ordered ammon. chlorid., grains xij; extract. cascaræ liquid., minims 40; tinct. nucis vomicæ, minims 6, with glycerine and water, thrice daily.

The symptoms now greatly improved, and the pain almost subsided, but some tenderness over the liver and slight fulness in the intercostal spaces remained.

On 24th May the liver was aspirated between the seventh and eighth ribs, but no pus was found. No marked change or relief followed the puncture, and the patient declined to have it repeated, though further exploration was called for. As the symptoms referable to the liver did not quite subside, and the man did not properly regain his strength and weight, he was invalided.

In this case the symptoms, though obscure, rather point to the presence of an abscess, which very likely might have been discovered by further exploratory punctures. If there is an abscess present, it may absorb or may become encysted, and may eventually show itself by more direct symptoms.

An attack of acute hepatitis in a fairly healthy subject, if taken in time, will generally yield to careful treatment, and end in resolution.

Treatment.—The patient should be kept in bed, and as cool as possible, but without being exposed to a draught. General bleeding is now seldom practised in the treatment of inflammatory affections in hot climates; and in a subject weakened by previous illness or by long residence in a hot climate, or where there is suspicion of suppuration, it should not be employed; but in a young and vigorous subject, and especially if there be distress of breathing from pressure on the lung, a moderate but rapid loss of blood will often be followed by great relief of all the symptoms. The experiences of the older writers on Indian diseases are strongly in its favour. Free local bleeding by leeches or cupping should almost always be employed. Or leeches may be applied round the anus to relieve the portal circulation. The leeching over the liver should be followed by warm fomentations. In many cases a full emetic dose—20 grains or so—of ipecacuanha will give much relief. A free purge should be given, unless serious diarrhœa or dysentery be present, when the ipecacuanha should be continued in small doses. A full dose of calomel, followed by a saline, is often the best purgative, and the bowels should be kept active by an occasional dose of calomel, or blue pill with podophyllin, followed by some aperient mineral water, as Friedrichshall, or Hunyadi Janos, or of Carlsbad salts; but care must be taken not to excite violent purging. Chloride of ammonium, in doses of 15 to 20 grains every three or four hours, is a most valuable remedy, and, given in combination with 20 to 30 minims of cascara, is especially useful. It allays pain and subdues inflammation in the liver, and is said to check the formation of abscess, and even to promote absorption of the pus after suppuration has

taken place. Tartar emetic in doses of grains $\frac{1}{6}$ to $\frac{1}{4}$, with nitrate of potash in frequent doses during the early stage, also tends to reduce the pain and other symptoms. Locally, relief will be afforded by the application of turpentine stupes, or still better, a compress of diluted nitro-hydrochloric acid, of the strength of 2 oz. of the strong acid to 1 gallon of warm water, kept constantly applied over the liver. A warm or tepid bath will often relieve some of the distressing symptoms. When the more acute symptoms have passed off, nitro-hydrochloric acid with chiretta or gentian is useful in restoring the action of the liver, and improving digestion. An occasional dose or two of euonymin, 2 to 4 grains, with rhubarb, is often very useful in relieving the pain and weight in the side which often remain, or which are liable to recur for some time after the more urgent symptoms have subsided; and care must be taken to keep the bowels free by an occasional aperient. Tonics of quinine or iron may afterwards be called for. The *dieting* of the patient is of the utmost importance. During the acute stage of an attack, it should be of the most simple, non-stimulating description, as milk, soups, farinaceous food, and given only in small quantity; and all alcoholic stimulant should be absolutely prohibited. For a long time the food should be very plain and simple; rich and spiced dishes should be avoided, and fatty and saccharine matters only very sparingly taken. All alcoholic drinks should be abstained from. Experience shows that people who have suffered severely from inflammation, or even from abscess, may quite recover, and afterwards be able to stand a hot climate perfectly, if they remain abstainers, or almost so, from alcoholic drink. But when the liver has once been partially damaged by disease it is rendered much more susceptible to the effects of alcohol. During convalescence, fresh air and gentle exercise are most beneficial; but over-fatigue and exposure to cold or great heat should be avoided. In many cases an attack of acute hepatitis will end in rapid and complete recovery.

Tropical Abscess.

Acute hepatitis may run on to suppuration, and this is the most *frequent* cause of *tropical abscess of the liver*. Suppurative inflammation of the liver is generally circumscribed and limited in its extent. In the inflamed and hyperæmic portions of the liver, yellowish discoloured patches appear which soften and break down and form an abscess.

Abscess may be developed in any part of the liver, either deep in

its substance or near the surface, but a common situation is the posterior and upper part of the right lobe. Such abscesses are generally single, though there may be more than one.

In some cases the abscess is enclosed in a more or less defined cyst wall, and this sometimes forms a thick, tough investing capsule; or the abscess may present ragged ill-defined walls formed of inflamed sloughy tissue which readily bleeds. When suppuration occurs the symptoms are often well marked. The pain will become more acute and throbbing, and in some cases, especially where the surface and capsule are involved, it may be excruciating. In some cases, as suppuration takes place, it is noticed that the pain subsides, but without any abatement of fever or other symptoms.

When the abscess is situated towards the upper surface of the liver, and presses upwards on the diaphragm and base of the lung, there will be troublesome cough and much distress of breathing and acute spasm of the diaphragm. There may be condensation of the lower part of the lung with diminished resonance and feeble respiratory murmur, or there may be well marked symptoms of pleuro-pneumonia which may mask the liver symptoms and render the diagnosis difficult and obscure. In such cases the real nature of the attack may be overlooked until perhaps a liver abscess may burst through the lung or show itself in some other direction.

When an abscess is situated on the lower aspect of the liver and presses on the stomach and duodenum, as pointed out by Maclean, obstinate vomiting is often a constant symptom, and may be difficult to control. The decubitus is generally dorsal and towards the right side, with the shoulders raised and often the thighs, especially the right, flexed, so as to relax the abdominal muscles; but in many cases the rectus muscle is rigid. As an abscess progresses the swelling generally takes some particular direction, and may present as a fulness or prominence which can be made out, either below the costal margin, or it may bulge between the ribs. Often when suppuration has occurred there may be detected a slight œdematous condition of the skin over the lower ribs with a smooth shiny surface; this is often a valuable diagnostic sign of abscess having formed. In cases where the abscess is small and deep seated there may be little or no pain or other symptom pointing to serious disease of the liver, and the difficulty of diagnosis may be very great.

The formation of an abscess is generally attended by more or less characteristic symptoms. The fever is often of an irregular or hectic type. The temperature is usually highest in the evening; there are often chills or rigors and profuse perspirations, often taking the form

of night sweats, the patient waking out of sleep bathed in perspiration and feeling cold and prostrated. The tongue is furred and the mouth dry, and often there is nausea and vomiting. Diarrhœa is a frequent symptom, and in some cases it is of a dysenteric character. The urine is generally at first scanty and high coloured; but when the substance of the liver has been extensively destroyed by suppuration the excretion of urea is found to be greatly diminished, and the urine of low specific gravity, pale and watery. All the symptoms will vary greatly in degree according to the extent and the situation of the abscess. The more acute cases are generally found in young or vigorous subjects who have not been many years in the country. The more chronic form, when the abscess forms slowly and insidiously, and when the symptoms may be very obscure, is more often met with in older residents, whose health has become impaired from living in a hot climate, and who have suffered previously from chronic congestion and enlargement of the liver, a condition very conducive to the formation of chronic abscess, which may then arise from very slight exciting cause.

In such chronic cases there may be no fever or rigors, little pain or tenderness, perhaps only some slight uneasiness or sense of burning in the side, and slight dull pain in the right shoulder. There is generally some loss of appetite with symptoms of indigestion. The bowels are irregular, and there is often diarrhœa with painless, watery stools, and perhaps a dry cough. The patient is ailing and out of health; he loses weight and strength, without any defined symptoms of what is wrong, until perhaps after a time the presence of an abscess is accidentally revealed. The case may be still more obscured by symptoms of malarial fever or cachexia. An unsuspected liver abscess has not unfrequently been found after death from some other cause. An abscess will sometimes remain for years enclosed in a firm investing cyst, giving rise to few or no distinct symptoms.

There is no doubt that in some cases a liver abscess becomes absorbed and cured spontaneously; the clinical history of many cases shows this, and the remains of such abscesses have been found after death. It is highly probable also that occasionally an abscess opens into some part of the bowel, its contents are discharged unnoticed, and the abscess cured.

A liver abscess may point externally, or it may burst into the lungs or stomach or any part of the intestinal canal, or even into the adjacent serous cavities; but the conditions and management of such cases need not here be considered.

Pyæmic Abscess.—Another form of liver abscess, often

known as *pyæmic* abscess, is frequently met with in tropical countries, and is dependent on dysentery, and is set up by absorption of septic material from unhealthy ulcers or sloughing in the intestines; or the abscess may be of distinct pyæmic origin, the result of suppurative inflammation or of infective thrombi forming in some of the veins which enter the portal system. It is considered by some, and particularly by the late Dr. Budd from his experience at the Dreadnought Hospital, that all, or nearly all, abscesses of the liver could be traced to such a cause; but experience in India tends to the conclusion that in the majority of cases tropical abscesses, whether acute or chronic, are not caused by dysentery or any septic absorption. At the same time, in many cases dysentery does give rise to abscess, and usually to the small multiple variety. The statistics of European hospitals in India show that liver abscess occurs in about 20 per cent. of cases of dysentery; but in many of these the abscess is not the direct result of the dysentery, but both diseases occur together, and are dependent on the same general causes, such as heat of climate, malaria, and so on, and only a small proportion of the cases are actually due to septic absorption. Often the symptoms of the two diseases appear together; and in not a few cases the illness begins with symptoms of hepatitis, and dysentery comes on later during the course of the attack, as in Case 3.

In the cases which result from dysentery or from septic absorption, the abscesses are often small and multiple. They are sometimes very numerous, and the whole liver may be studded with them.

Cases of liver abscess:—

CASE 3.—ACUTE HEPATITIS, ABSCESS OF THE LIVER BURSTING INTO THE LUNG. DEATH FROM EXTENSIVE SUPPURATION OF THE LUNG.

Private J. W., aged 27, two and a half years in Egypt, admitted into hospital at Cairo, on 8th February 1889, for hepatitis. The liver was enlarged, the increased dulness on percussion extending both upwards and downwards; there was pain and tenderness, and he had fever and diarrhœa. He was invalided home, and arrived at Netley on the 15th April. His symptoms then pointed to the presence of an abscess, the liver dulness extended upwards to the level of the fourth rib in front, and downwards to 2 or 3 inches below the costal margin, but there was no circumscribed swelling or bulging of any part. The lower part of the right lung was consolidated. There was some pain and tenderness over the liver, dysenteric purging, and constant fever and dry cough.

After a few days a large abscess burst through the lung, and from 6 to 18 oz. of thick discoloured pus were expectorated daily. As this expectoration continued, the liver was explored by the aspirator with the view of drainage, but no abscess could be found. Extreme weakness and emaciation supervened, with hectic fever, and the man gradually died from exhaustion on 29th September.

On autopsy, the whole of the right lung was found to be converted into an enormous abscess cavity, with ragged walls of softened lung tissue, but in parts lined with a smooth, pyogenic membrane. The liver was enlarged, and extended 3 to 4 inches below the costal margin on the right side. On the upper surface of the right lobe there was a shallow, saucer-shaped depression, about 4 inches in diameter and ½ inch deep, lined by a firm membrane, outside which the liver tissue was softened.

The liver substance was pale generally, but otherwise appeared healthy.

The mucous surface of the large intestines was studded with numerous small ulcers, and the coats were thickened.

In this case there was acute inflammation of the liver, with the formation of a shallow abscess close to the upper surface, which burst through the diaphragm into the lung, and there setting up destructive inflammation and the formation of an enormous lung abscess; the liver itself being very little involved in the suppuration.

CASE 4.—ACUTE HEPATITIS AFTER RECOVERY FROM DYSENTERY. ABSCESSES IN THE LIVER, ONE OPENED EXTERNALLY BY INCISION, A SECOND BURSTING THROUGH THE LUNGS. RECOVERY.

Private H. B., aged 30, six years' service, two and a quarter years in the Straits Settlements. Habits rather intemperate. In September 1891 had an attack of dysentery, from which he recovered in three weeks. Twelve days afterwards was readmitted into hospital with symptoms of acute inflammation of the liver, severe pain in the right hypochondrium and the right shoulder, rigors, fever, and sweating. The symptoms quickly subsided, and he only remained in hospital fourteen days; but after a few days they returned, and he was readmitted. He now also suffered from a short hacking cough, but no expectoration. An abscess formed in the liver, and, twenty days after admission, was opened by an incision between the seventh and eighth ribs in the axillary line, and about 2 pints of greenish pus escaped. A drainage tube was inserted and remained in for three months, during which time a varying quantity of pus continued to drain away, and then finally ceased, and the incision closed. The operation gave immediate relief to the pain and other symptoms; but after a few days the pain and cough and fever returned, and a month after the incision another abscess burst suddenly through the right lung, and a quantity of reddish purulent fluid was coughed up. This was followed by severe cough and constitutional disturbance lasting for a week, when the fever and all the symptoms again subsided; but he continued to cough up daily about 3 oz. of reddish pus. He was invalided home, and arrived at Netley on the 12th April 1892; he was then sallow and anæmic, and rather emaciated. Was free from fever, but had a troublesome cough, and expectorated about 3 oz. daily of reddish fluid, containing pus and blood cells but no tubercle bacilli. There was dulness on percussion over the base of the right lung up to the angle of the scapula behind and above the sixth rib in the axillary line. Moist, crepitant râles were audible over the lower part of the right lung. There was no liver enlargement below the costal margin, and no fever; tongue clean but pale; appetite good, but occasional vomiting after food. Bowels regular and stools normal, urine rather scanty, passing 30 to 40 oz. in twenty-four hours, rather high coloured and with abundant deposit of lithates. Urea deficient, passing only about 270 grains in the twenty-four hours.

Treatment.—Non-stimulating but nutritious diet, gentle exercise, and

tonics. The cough and expectoration gradually diminished, and in time quite ceased, and his general health and condition greatly improved, and he left the hospital after about two months in fair health.

There were evidently two abscesses in this case, one opened externally and one burst through the lung. The abscess followed closely on an attack of dysentery.

Case 5.—Abscess of the Liver coming on in the Course of Chronic Dysentery, and bursting through the Lung. Recovery.

Private W. G., six years' service, eighteen months in India. Soon after arrival there he had a slight attack of dysentery, which was quickly relieved, but irritability of the bowels and slight symptoms of chronic dysentery persisted for a long time. In September 1891 he first noticed pain in the hepatic region, extending into the right shoulder and arm. The pain was not constant, and was variable in intensity. At times he had cold sweats, but had no marked rigors.

In January 1892 the dysenteric symptoms became much worse, and the liver pain increased in severity, and was attended by slight fever, his temperature rising to 101° or 102° F. at night. Towards the end of the month a dry cough set in, and on 2nd February he expectorated a quantity of greenish pus mixed with blood. This continued profusely for about a week, and then gradually became less. All the other symptoms now diminished, and the temperature became normal. He was, however, weak and anæmic, and much below his proper weight. He was then invalided home, and arrived at Netley on 2nd May 1892. He kept fairly well during the voyage, with the exception of mild dysenteric symptoms. On admission into Netley the liver was found to be enlarged, but there were no other symptoms pointing to hepatic mischief; but on the 20th he was again attacked with pain in the right side shooting up into the shoulder and down the arm, and there was great tenderness over the liver, but no fever, and his appetite and digestion were fairly good. He was given 3 grains of euonymin and 2 grains of rhubarb every night, and 20 grains of chloride of ammonium three times a day. He was also put on a simple, non-stimulating diet. After three or four weeks' treatment his general health again improved, and he gained in weight steadily. He had no more fever, cough, or expectoration, and the dysenteric symptoms quite ceased, but the pain and tenderness over the liver continued for some time; the pain was felt especially on deep inspiration. He also frequently felt sick in the morning, and sometimes vomited. He was now given dilute nitro-muriatic acid, with infusion of chiretta thrice daily. All the symptoms now gradually passed off, and in the course of a few weeks he was quite well, and only very slight enlargement of the liver remained, and he returned to duty. The abscess of the liver in this case, though associated with chronic dysentery, did not present the characters of abscess of pyæmic origin.

Case 6.—Injury to Side. Abscess of Liver three Months afterwards, treated by Free Incision.

Gunner K., aged 28, one and a half years in Mauritius, had good health until November 1891, when he met with an injury to his right side from a man falling on him at gun drill; but after a few days in hospital all pain and symptoms subsided, and he returned to duty. In March 1892 he was again

admitted into hospital for hepatitis, with symptoms of shivering, night sweats, and dysentery, and followed by enlargement of the liver and fulness of the right side. He was invalided home, and arrived at Netley in August. There was then constant pain in hepatic region, great enlargement of the liver in both upward and downward directions, but no localised fulness or bulging in the side, but severe pain at one spot over the lower ribs in axillary line.

There was no fever or dysentery.

On 6th September an aspirating needle was inserted between the eighth and ninth ribs, where there was fulness and bulging, and an abscess seemed to be pointing, and about 10 oz. of thick dark coloured pus was drawn off, and on the 8th a free incision was made into the abscess. The abscess was nearly 3 inches deep in the liver substance. About 1½ inch of the rib had to be removed as the intercostal space was very contracted, and a large drainage tube was inserted. At the operation a large quantity of pus was drawn off, the abscess cavity being very large.

The day after the operation there were no bad symptoms, and the temperature was 96° F.; no fever followed, and the man is now progressing favourably; there is a moderate discharge of pus still going on, but in all likelihood the man will soon recover.

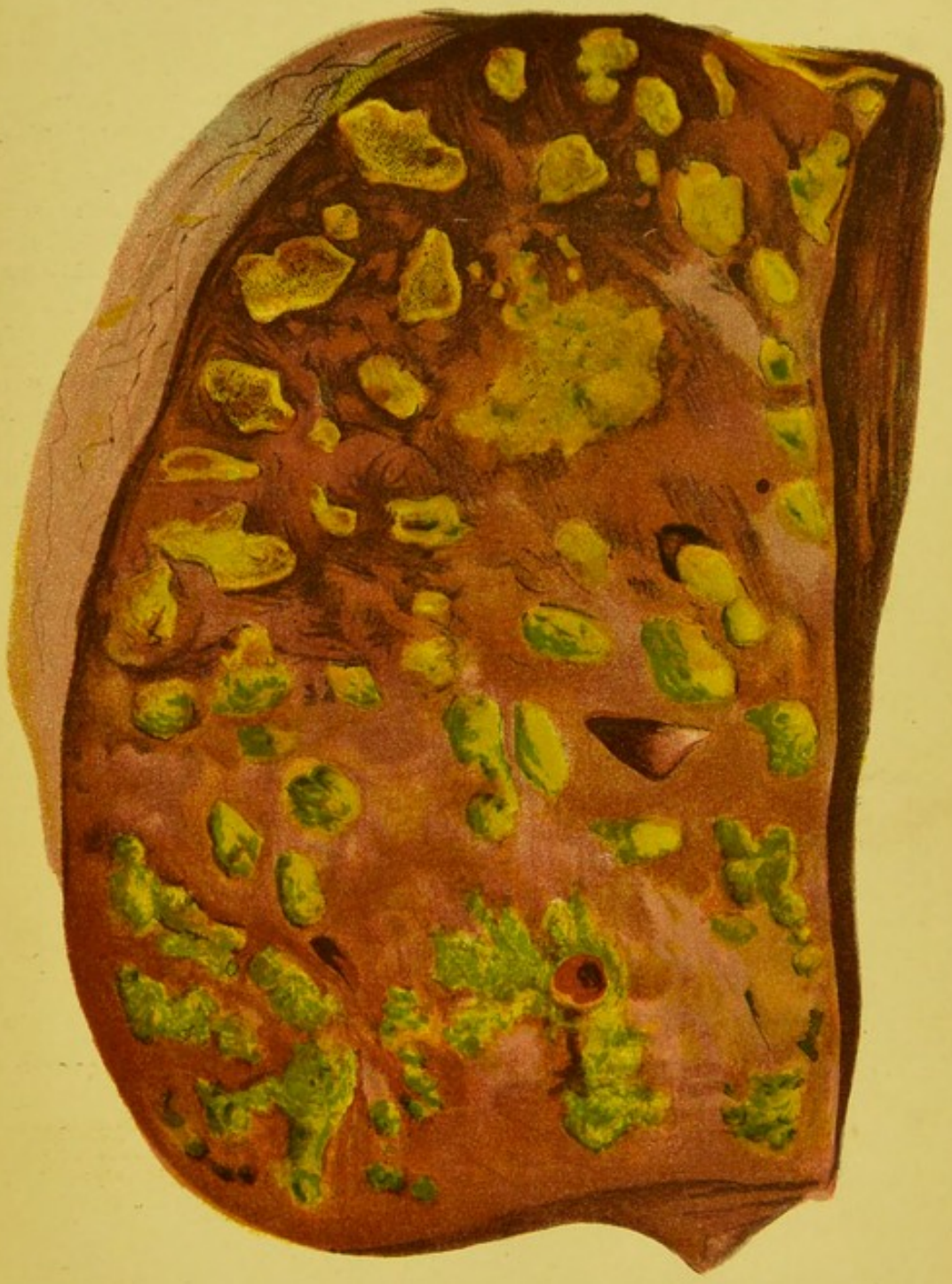
Another form of liver abscess, associated with dysentery, has for some time past been under investigation by Drs. Councilman and Lafleur in America;[1] they describe a special form of dysentery caused by an amœba, the *A. coli*, or "*A. dysenterica*" as they call it, which sets up characteristic lesions of the intestines, and in some cases it makes its way direct to the liver and lungs and sets up abscess, differing from the usual form of *septic* abscess. In the pus of such abscesses they found the actively moving amœbæ.

The subject requires further investigation, but similar forms of amœbic abscess have recently been described by observers in India.[2]

In the case No. 7 we have an example of a rare and unusual form of liver disease, and the symptoms present during the rapid and acute attack did not at all point to the condition found after death. The nature of the attack was obscure, and was only cleared up by the post-mortem examination. The patient was a young, healthy man; he had not been in a hot climate, had no history of dysentery or other disease likely to give rise to pyæmic abscesses, and had never before suffered from symptoms of gall-stones or from any form of liver disease. The symptoms were those of acute jaundice from inflammatory obstruction of the gall ducts, and the disease was rapidly fatal. The numerous points of softening of the liver substance, with patches of gangrene and suppuration, were, no doubt, caused by the complete obstruction of the bile ducts, and the

[1] *Boston Medical and Surgical Journal*, 24th December 1891.

[2] *Indian Medical Gazette*, November 1892.



CONDITION PRODUCED IN LIVER BY IMPACTION OF GALL-STONES IN COMMON DUCT.

accumulation and backward pressure of bile in the dilated and distended ducts in the liver substance. For the post-mortem examination, and the drawing of the appearance of the liver, I am indebted to Surgeon-Captain Bruce, Army Medical Staff. The case affords an explanation of other obscure cases of rapid jaundice and death which are occasionally met with, but in which there is no opportunity of clearing up the obscurity.

CASE 7.—MULTIPLE ABSCESSES AND SLOUGHING OF THE LIVER, THE RESULT OF GALL-STONES, AND CLOSURE OF THE COMMON BILE DUCT.

Private A., aged 28. Admitted into hospital at Netley on 19th February 1891 for jaundice, which had commenced about one day previously. Was before the attack in good health, had never been abroad, and never suffered from colic or pains in the liver. On admission there was deep yellow staining of the skin and conjunctivæ. He felt no pain or uneasiness in the hepatic region. The bowels were confined and the tongue furred. On the fifth day of the attack he felt great thirst. The jaundice had much increased; the liver had become enlarged. It was not painful, but was tender on pressure or percussion; temperature, 100·2° F.; on the seventh day the liver was further enlarged, but still there was no pain. Bowels moved nine times in twenty-four hours, tongue dry and brown. Eighth day temperature, 104°·2 F. in evening. Appearance typhoid, sordes on teeth and lips, pulse weak and rapid. Ninth day temperature, 103°·6 F. in evening; says he feels much better, has no pain, bowels moved eight times in the twenty-four hours, no bile in the stools. Tenth day temperature, 104°·8 F. last evening, and 101° F. at 3 A.M. this morning, still no pain; at 4 A.M. he had a fit of coughing, and expectorated some clear mucus and became faint, but was revived by brandy and then perspired profusely. At 5.30 A.M. he suddenly vomited some "coffee-ground" matter and fell back dead.

Post-mortem examination.—Body well nourished, lungs and heart normal. A few ounces of opaque yellowish purulent fluid in peritoneal cavity. Four oz. of yellow serum in pericardium. Liver much enlarged, extending 5½ inches below the ribs in front. Weight 8 lbs. 12 oz. The surface smooth, and covered with a thin layer of greenish lymph. The gall-bladder and the cystic and common ducts much thickened and filled with yellow semi-solid matter of clayey consistence, in which were found sixteen gall-stones, two of which were the size of small cob-nuts, composed chiefly of cholesterine and bile pigment. The ductus communis was completely closed, and bile could not be forced through it. The upper portion of right lobe of the liver was soft and pulpy, and on section almost all parts of the organ were to be seen studded with small patches of gangrenous liver tissue, and small cavities containing greenish pus of cheesy consistency, and particles of sloughy tissue. These cavities varied in size from a pea to a small walnut. The liver substance was everywhere soft and friable, and in parts dark-coloured and congested. The appearance of a section of the liver is accurately depicted in the accompanying illustration (Plate III.), sketched from nature.

In this case it would appear that the numerous suppurating cavities and the points of gangrene were caused by the backward pressure of the bile in the bile ducts all through the substance of the liver.

Treatment.—When, during an attack of hepatitis, suppuration seems to be threatening, the treatment should be somewhat on the same lines already recommended for acute hepatitis; but lowering remedies, such as bleeding, emetics, and strong purgation, must be at once discontinued. The chloride of ammonium should be given regularly. Dr. Stewart, from a large experience of cases in Madras, held that chloride of ammonium was capable of causing absorption of an abscess after it had formed, the patient being kept perfectly at rest and on low diet. This requires confirmation; but the drug has undoubtedly a powerful effect in arresting inflammatory changes, and so checking the formation of abscess. The diet should still be simple and non-irritating, and no stimulants allowed.

Where there is strong suspicion of matter having formed, great help is derived, and often great relief afforded, by puncturing the liver with an aspirating needle. If pus is reached, and the abscess emptied, as a rule it refills, and further operation by free incision and drainage is called for. In many cases where no pus is detected the greatest benefit and relief often follows the puncture, which may be repeated several times, at intervals of a few days. The needle may be thrust deeply into the liver either between or below the ribs, choosing the part where the local signs of disease are most pronounced. In some cases only a few drops of blood and serum are drawn off, in others the amount will be larger; but in either case relief often follows. Cases are often met with of acute congestion of the liver in which the symptoms are very severe, and point strongly to abscess, but which seem to stop short of the actual formation of pus. Such cases are often relieved to a remarkable extent by this small operation. It seems to be attended with little or no risk; but the strictest antiseptic precautions should always be taken. The cases treated in this way in India and at Netley Hospital are very numerous.

The same proceeding may often be usefully employed in cases of chronic liver enlargement with obscure signs of abscess; and when the symptoms are urgent or persistent, the greatest benefit often follows the puncture of the liver with the aspirating trocar.

The operative treatment of liver abscess does not come within the scope of this article.

Chronic Congestion.

Etiology.—Chronic congestion and enlargement of the liver is a very common affection in hot climates; and leaving out the cases

due to such causes as are equally rife in all climates, such as mechanical obstruction to the circulation from heart or lung disease, tumours, and so on, it is a disease from which many Europeans suffer after a few years' residence in India, and is often one of the special troubles of life in that country. The condition may follow one or more attacks of hepatitis, but it more often comes on slowly and gradually without any previous acute symptoms. It is, undoubtedly, an effect of long continued high temperature, but it is generally found in those who lead an inactive life, or live too freely, and especially in those who habitually take some form of alcohol.

Malaria also, and attacks of malarial fever, will often lead to congestion and enlargement of the liver.

At first the condition is that of hyperæmia, and the enlargement will vary greatly from time to time, and will be influenced by slight causes. It will be increased by the stimulation of a too full meal, or by exposure to cold, and will be diminished by free purgation, and may disappear under appropriate treatment; but when persistent for a long time or often repeated, it will lead to a condition of fibrosis or hypertrophy of the connective tissue, with atrophy of the secreting elements of the gland. In these cases the surface of the liver is smooth, its substance is dense and firm, and it generally bleeds freely on section; but at a later stage the appearances are those of cirrhosis; and in advanced cases the liver may become reduced in size, or it may undergo fatty or lardaceous degeneration. This condition is not unfrequently found in the livers of people who have spent some years in the tropics, and is the commonly known "Indian Liver" from which so many old Indians suffer; and the large livers, yellow skins, and irritable tempers of Indian *Nabobs*, as they used to be called, men who had survived their Indian career and returned to live at home, are proverbial. All this is now very much an idea of a past generation, of a period when people lived much less suitably to the climate than they do at the present day; but chronic liver congestion and enlargement is still a frequent source of ill-health in India, and often leads to attacks of a more serious or dangerous character. The enlargement of the liver in these cases is generally uniform, extending in all directions, and varies much in degree. Sometimes being very great, so that the organ can be felt for some distance below the ribs, even as far as or below the umbilicus; or it may be slight, and only traceable by careful examination. When slight, the edge of the liver may generally be detected some little distance below the costal margin by its giving a dull note on percussion; generally also, there is enlargement with increased dulness in an upward direction, and

the lower part of the lung will often be compressed and give a defective breath sound.

Symptoms of Chronic Congestion.—The patient feels more or less constant uneasiness and sense of weight in his side, hardly amounting to pain, and often a slight feeling of soreness or burning. A sudden pain or shock is felt from any jar on the feet, as from a false step. There is generally some dull pain in the right shoulder, over the scapula and down the arm. The bowels are irregular, constipation often alternating with diarrhœa; there is a tendency to piles, and great relief is often afforded by their bleeding. Heartburn and flatulency are troublesome. Appetite and digestion are impaired. Often there is no inclination for food in the mornings at breakfast time, though later in the day the patient may feel hunger and eat well.

There is generally some sense of languor and weakness, and some loss of weight. There is often no jaundice, but the patient has a sallow, anæmic look. The symptoms may all be slight and obscure, but when this condition of chronic congestion and enlargement is present there is always great liability of acute inflammation or abscess from very slight cause. An indolent abscess may form, and remain for a long time without giving rise to any marked symptoms. In a hot climate it is very important that such cases should be detected early and submitted to treatment. In an early stage, before the liver has undergone marked structural change, recovery may be complete; and in a later stage, though part of the organ may have undergone fibrous degeneration or become atrophied, the remainder will be able to carry on its functions if aided by a suitable regimen.

In the treatment of this condition diet is all important. It should be restricted in quantity, and very simple. All rich food, hot spice, and stimulating articles should be avoided; and fermented liquors should be eschewed. The patient should take regular active exercise, avoiding over-fatigue, or too great exposure to sun and heat; and he should carefully guard against sudden chills. The bowels should be kept regular and active. This may be done by taking daily, or as often as required, some mineral saline, as Hunyadi Janos water, or Pullna, or any other, or Carlsbad salts. When the symptoms are more urgent, an occasional purge with mercury or podophyllin should be given; and euonymin in doses of 1 to 3 grains with a little rhubarb often will give much relief, especially where there is pain and weight in the side. The chloride of ammonium in doses of 15 to 20 grains three times a day should at the same time be given. When there are local symptoms and

enlargement, tincture of iodine should be painted over the side, and repeated often enough to keep up some irritation of the skin.

In the case of Europeans in a hot climate, if the symptoms do not readily yield to treatment, the patient should be sent to Europe, or should take a sea voyage, at the same time attending most carefully to diet and regimen.

CASE 8.—CHRONIC CONGESTION AND ENLARGEMENT OF THE LIVER, WITH HIGH TEMPERATURE AND RIGORS, BUT WITHOUT SUPPURATION. RECOVERY.

Private J. B., of active and temperate habits, five and a half years in India. Admitted into hospital at Dumdum in October 1891, on account of pain and "swelling" in his right side from enlargement of the liver. He had for a long time previously suffered from weight and uneasiness in the side, and occasional attacks of pain. The pain extended upwards to the right shoulder, and was for some time considered to be rheumatic. He felt sick after food, and his bowels were constipated. He had an evening rise of temperature up to about 102° F., and this persisted for a long time. He also had frequent rigors and fits of perspiration. He was put upon light diet, with occasional blisters and poultices to his side to relieve pain, and given a mixture of dilute nitro-muriatic acid. He was five months in hospital; and though his condition improved he did not get well, and was invalided home in March 1892, and admitted into Netley, May 2nd. His complexion was sallow, and the conjunctivæ muddy-looking but not jaundiced. Tongue furred in the centre, bitter metallic taste in his mouth, bowels confined and motions dark and pasty; had a dry, short, painful cough. The liver was considerably enlarged, both in upward and downward direction, and could be felt for 1½ inches below the ribs, nearly as far back as the axillary line. There was tenderness over the whole hepatic region, with a dull, aching pain.

Treatment.—Occasional mercurial purgatives with salines, chloride of ammonium, and dilute nitro-muriatic acid. Counter irritation by blisters and iodine. Plain non-stimulating diet, and later on, tonic preparations of iron. He quickly began to improve, and in a short time nearly all the symptoms subsided; but slight enlargement of the liver remained. He returned to duty in August. This was a case of chronic congestive enlargement of the liver due to climate, and probably partly of *malarial* origin.

CASE 9.—ACUTE HEPATITIS AFTER FOUR YEARS' RESIDENCE IN INDIA, FOLLOWED BY CHRONIC CONGESTION AND ENLARGEMENT, THREATENING FORMATION OF ABSCESS. RECOVERY; WITHOUT SUPPURATION.

J. C., aged 38, four years in India, a temperate man. Health generally good, but had syphilis at times from 1882 to 1885. In 1888 he suffered from ague, by which he was much pulled down. In March 1890 was sent to the hills on account of weakness, the effects of ague and syphilis. He greatly improved in health; but in January 1891 had a severe attack of acute hepatitis, which kept him in bed for three weeks. He was left weak and anæmic, with enlargement of the liver, and pain and tenderness. He continued to lose weight, so was invalided home. On the voyage he suffered much from pain in the liver, and from occasional fits of rigors. He arrived at Netley, May 1st. On admission his skin was very sallow and almost jaundiced. He was weak and emaciated, tongue furred, stomach irritable,

and no appetite. Bowels irregular, generally constipated, motions pale and clayey. There was great tenderness over, and considerable enlargement of the liver, which in the mammary line extended from the upper border of fifth rib to nearly 3 inches below the costal margin, and obvious bulging of the ribs and intercostal spaces on the right side, and pain in the hepatic region shooting up the back, and a constant dull pain in the right shoulder running down the arm. No fever. The urine loaded with lithates.

Treated by plain non-stimulating diet, and given chloride of ammonium, grs. x., and dilute nitro-muriatic acid, M. x. ter. die. Tincture of iodine was painted over the liver. After a short time the medicine was changed to small doses of perchloride of mercury and iodide of potassium, as he was suffering from periosteal pains which seemed to be syphilitic. He was then given quinine with iodide of potassium. After a month he greatly improved; the pain and tenderness in the hepatic region almost ceased, and he gained in weight, and the liver enlargement subsided, and soon afterwards he left hospital convalescent. The symptoms and condition of the liver at one time pointed strongly to the presence of an abscess, but after a time quite subsided.

Other Forms of Enlargement.

Besides the enlargement of the liver, the result of chronic congestion, we meet with other cases of slow enlargement of the organ, also associated with residence in hot climates, which present the characters of waxy or lardaceous degeneration, and which seem to be caused by the poison of malaria, or to be the result of the impaired and perverted nutrition to which malaria gives rise.

There are in the Museum at Netley many specimens of lardaceous livers taken from soldiers who have died from the effects of tropical disease. In some this condition of the liver has been associated with protracted ague and malarial cachexia, in others with chronic diarrhœa or dysentery, or other wasting disease. In some cases the liver is of enormous size. The enlargement is generally uniform and the surface smooth, but sometimes the liver is lobulated; on section its substance is firm, with hypertrophy of the fibrous connective tissue, and atrophy of the gland structure, but presenting no inflammatory deposits. Iodine gives the characteristic reaction and brown colour when applied to the cut surface. In some cases the enlargement is associated with fatty degeneration, but following similar causes, as malaria or wasting disease. In lardaceous enlargement there may be no symptoms beyond the general impairment of health, and some discomfort from the weight and size of the liver.

In some cases the liver is found to have undergone a form of pigmentary degeneration from the accumulation of black pigmentary matter in its parenchyma in and around the capillary vessels. This is generally the result of severe or prolonged malarial fever; and similar pigmentations are seen in the spleen and other tissues.

Functional Liver Affections.

Functional affections of the liver, giving rise to a variety of indirect symptoms, and not attended by any marked structural changes, are more common in hot than in cold climates. In addition to its *bile-secreting* functions, the liver is one of the organs mainly concerned in the processes of blood formation, and of nutrition and assimilation. It also largely helps in the oxygenation and the destructive metamorphosis of nitrogenous materials, and in the formation of urea.

Any derangement of these normal functions of the liver will often cause serious impairment of the digestive and nutritive processes, such as great emaciation, deficient formation of urea, diabetes, and sometimes abnormal development of fatty tissue and other forms of defective nutrition; and when the liver functions are imperfectly performed, there is always great tendency to congestion and enlargement, and to various other structural diseases. The causes of these functional affections, to which people are so liable in hot climates, are similar to those which have already been noticed in regard to the organic diseases, such as too free living, insufficient exercise, too sedentary habits, deficient supply of oxygen, especially where the result of overcrowding and insufficient fresh air, all of which conditions are greatly intensified by the influence of high temperature.

Jaundice, the presence of the colouring matter of the bile in the circulation and in the tissues, is a condition often met with in hot climates, and may often be considered a functional affection, and not necessarily due to any distinguishable organic change. The bilious, sallow skins, often brought on by residence in a hot climate, is a well-known appearance, though much less common now than in former years when people lived less carefully; still what is known as biliousness and torpidity of the liver is a frequent trouble of life in India.

The most common cause of jaundice in all climates is some mechanical obstruction to the passage of bile through the ducts, as may arise from stricture, from catarrhal inflammation of the ducts and gall-bladder or of the duodenum, from impaction of calculi, from accumulation of inspissated mucus or biliary products, from tumour or growths of any kind in the liver or neighbouring parts pressing on the ducts, or from enlargement of the liver itself; from the presence of round worms or other parasites in the bile passages, —a condition not unfrequent in India, where the *Ascaris lumbricoides* is so prevalent. Jaundice may occur in the course of hepatitis, cirrhosis, or any disease of the liver, and is always present in acute

atrophy. It is often seen in malarial fever and in some of the other specific fevers, and in septicæmia. Jaundice, again, may be due to *over secretion* of bile, as in hyperæmia of the hepatic tissues, which is present in the early stage of acute congestion and inflammation; or it may be caused by excessive destruction of blood corpuscles in the liver, leading to increased formation of bile. The bile that passes through the biliary passages into the intestines is partly excrementitious, but a portion of it is reabsorbed and taken up into the portal circulation, and under normal conditions it undergoes oxygenation and metamorphosis in the liver tissues; but when the amount is excessive, or when the oxygenating power of the blood is defective, the excess of bile that is reabsorbed from the intestines is taken up by the lymphatics; and this seems to be a common cause of jaundice in hot climates, where the high temperature and lessened respiration interfere with the complete oxygenation of the blood and the normal metamorphosis of the bile.

The same effect will be produced by undue and prolonged retention of bile in the biliary passages and bowels, as we see in habitual constipation. When the *through* passage of the bile is delayed there is excessive reabsorption, more is taken up than can be got rid of by oxygenation in the liver, and it accumulates in the system. Jaundice may also arise from suppressed secretion and non-elimination. This may be sometimes induced by mental emotions, as fear, grief, or anger, which, in some people, have a powerful influence on the formation and elimination of the bile.

In India we frequently see cases of slight jaundice associated with symptoms of impaired health and nutrition, the result of residence in a hot climate; and especially in subjects of malaria, but in which there is no evidence of actual liver disease. The symptoms are not always very definite; but in addition to the yellowness of the skin, there are generally loss of appetite and impaired digestion. The stomach is irritable, and often there is nausea and occasional vomiting after food, or in the early mornings. The vomited matter may be bilious, showing that secretion of bile is not arrested. The bowels are generally irregular, constipation alternating with diarrhœa; the stools are often pale and offensive, or are at times dark coloured and irritating. There is a tendency to flatulence and heartburn. The tongue is furred and yellowish, and there is a bitter or metallic taste in the mouth. There is generally some wasting and loss of strength, and often great depression of spirits and irritability of temper, amounting to hypochondriasis. There may be some hepatic pain and fulness, and perhaps bleeding piles or some other form of hæmorrhage from the bowels. There are often other symptoms

not exactly referable to the liver, as headaches, palpitation of the heart and irregular pulse, heaviness and drowsiness after meals. These symptoms are in great measure due to defective metamorphosis of the albuminoid matters in the liver, and imperfect formation of urea, and consequent excess of lithic acid and lithates in the blood and urine. The condition may be brought on or increased by want of exercise, by too free living, by neglect of the action of the bowels, and so on. When more food is taken into the system than is necessary for the nutrition of the tissues, or than can be readily got rid of by the skin, lungs, kidneys, and other excretory organs, it overworks the liver and causes functional derangement. In some people such derangements are very readily set up, especially so in a hot climate. Often, no doubt, the stomach is more in fault than the liver. Impaired digestive power, the "accursed hag dyspepsia," is one of the great troubles of hot climates, but generally the liver is primarily affected. At first the symptoms may be very slight and progress very slowly, but in time the patient's health becomes seriously impaired, and he is forced to return to a temperate climate and to undergo a long course of treatment. In such cases a sea voyage, combined with a restricted and careful diet, regular exercise, and keeping up the action of the skin and bowels, have a most beneficial effect. Exercise to be effective should be brisk, something more active than mere gentle walking. It should be of a nature to cause active movements of the chest and diaphragm. Horse exercise, when attainable, is especially good. Turkish baths, which makes the skin act violently, are often found efficacious; but care must be taken against the increased anæmia and exhaustion which may result from their use. The bowels should be regulated by occasional doses of such drugs as rhubarb and magnesia, podophyllin, euonymin, and the mineral salines, as Carlsbad salts, Vichy, Friedrichshall water, and so on; and when it can be managed the greatest benefit will result from a visit to Carlsbad, or one of the other watering-places, where a regular course of treatment can be carried out systematically; but in cases where there is much anæmia and impaired nutrition, great care is required not to still further impoverish the already watery blood. Abundant fresh air is all important; a residence at the seaside or among mountains is often just as good as at one of the special watering-places, provided that the same mode of life and regimen are strictly followed.

With return to a cool and bracing climate, the prognosis is generally favourable; and if the patient will persevere with the regimen and treatment his case requires, and will be careful not to over-stimulate and throw too much work on his exhausted liver,

his recovery will generally be complete and permanent, and he will again be quite able to stand a hot climate; but any want of care will easily lead to a return of the symptoms.

The history of tropical diseases, and the experiences we have gained by long connection with India and other hot climates, in which Europeans of all classes have lived for so long a period under every variety of condition, seem to point conclusively to the fact that the climate and long exposure to heat are the main factors in the production of liver disease; but that at the same time, by careful habits, and by adopting a mode of life suitable to the climate and the altered conditions of existence, much may be done to mitigate the injurious influence exerted upon the liver by such climates. The question has been already solved by the natives of India. The mass of the Hindus, the Aryan race, who come from the same Indo-European stock as the Europeans, and who, thousands of years ago, migrated from the cold northern regions of Central Asia into the burning plains of India, are no more liable to liver diseases than we are at home. They have, as we know, become almost vegetarians, and they indulge to a very slight extent in alcoholic drink. Again, the Mohammedan peoples of Upper India, who have mainly settled in India from more northern regions and from cold climates, are equally free from liver disease. They, as a rule, take much more animal food than the Hindus, but they absolutely eschew all alcoholic drink. There is no reason why Europeans in India should not enjoy an equal immunity with the people of the country from this class of disease, and all experience goes to show that Europeans can by careful habits spend the great part of their lives in India with very little risk of suffering from the common tropical liver diseases which have always been the cause of so much sickness and mortality among the English in India.

CHAPTER XVIII.

TROPICAL LIVER ABSCESS.

BY SIR JOSEPH FAYRER, K.C.S.I., LL.D., M.D., F.R.S.

Natural History and Etiology.—Tropical abscess of the liver has been so designated from the fact that it is almost entirely confined to residents in tropical or subtropical climates, or to those who have resided there, in whom suppuration has occurred after return to a temperate climate.

The comparatively rare cases of liver abscess which do present themselves in those who have never been in the tropics, are due to other though cognate causes. Such cases are pyæmic, and differ, with few exceptions to be subsequently noticed, from the large, single abscess,—it may be double or treble,—peculiarly characteristic of one form of the tropical disease, but are closely allied to those which depend upon dysenteric ulceration, and which are due to septic absorption from the ulcerated bowel, or to general septic poisoning. The liver in these cases may be found studded with abscesses, varying in size from a pea to an orange or larger.

Though the so-called tropical liver abscess is essentially an exotic disease, it is by no means determined to what particular climatic condition its genesis is to be attributed. The great disproportion of its occurrence in different parts of the world, where the same climatic conditions appear to exist, seems to suggest that it cannot be attributed to mere heat, malaria, moisture, or sudden alternations of temperature. It must be due rather to the combined effects of these, with varying food, habits and modes of life, and the different degree of strain imposed thereby on the functions of the liver. How far any one of these individually may be determinative, it is not possible to say; but be this as it may, some varying combination of these factors is apparently the explanation of its more frequent occurrence in India than in the West Indies or other tropical regions.

As Murchison[1] has pointed out, a high temperature favours functional derangements relating to sanguification and disintegration of albumen; whilst diet, which is suitable for cold or damp climates, in the tropics produces liver derangement. The breathing of a heated atmosphere gives a diminished supply of oxygen to the system; and further results of high temperature are to produce changes in the cells of the liver, which become filled with granular matter, and thus prone to further degeneration.

This much at any rate can be said as regards Europeans, that recent arrival is favourable to the occurrence of abscess; Waring showed that 50 per cent. of 114 cases occurred in people who had been less than four years in India.[2]

Murchison's statement, that liver abscess is chiefly met with between the ages of 20 and 45, is in accordance with experience.

Maclean suggests that the comparative rarity of liver abscess in the West Indies may be due in part to the tempering and purifying of the atmosphere by the sea-breeze, and to the fact that though the temperature there is very high during the day, the intense heat is not so continuous as it is in India, and the nights are cooler. Europeans in the West Indies can expose themselves much more to the sun than they can in India. In addition to the general influence of climate, he thinks that liver abscess is due to the effects of excess of nitrogenous food, intemperance, and sudden changes of temperature acting on systems weakened by cachexia induced by heat and malaria. Where alcoholic intemperance cannot be assigned as a cause, indolence and excess in eating, combined with the climatic influences already mentioned, are probably the most active factors.[3]

Admitting the validity of all this, it cannot be doubted that cases occur where no premonitory symptoms, and nothing in the mode of life or the general condition of the patient, would indicate the operation of these causes.

The frequency of liver abscess would seem to be diminishing; and it may be expected that it will still further diminish as improved hygienic conditions of living, occupation, food and drink obtain. The habits and modes of life in the tropics now are very different in their hygienic aspects to what they were in the past; and, as a consequence, not only liver abscess, but other causes of mortality and invaliding, are gradually becoming less frequent.

[1] Murchison, *Functional Derangements of the Liver.*

[2] Waring, *An Inquiry into the Statistics and Pathology of some Points connected with Abscess in the Liver.* Trevandrum, 1854.

[3] Maclean, *Diseases of Tropical Climates.*

The different forms in which liver abscess presents itself in the tropics may be briefly described as follows. They are not always readily to be distinguished and differentiated, though typical examples are tolerably distinct.

First, then, the large abscess, which is generally, though not always, single, is referable to the effects of climate combined with altered conditions of living, food and drink, and the excessive or injudicious use of alcoholic beverages; it must not be assumed, however, that intemperance is invariably a cause. It occurs even in the most temperate, though it is more frequent in those who live freely. Of forty cases in which the habits were noted by Waring, 67·5 per cent. were intemperate.[1] This abscess is often very large [the exceptionally large quantity of 80 oz., and again 50 oz. of pus, have been removed by the writer of this article from the same liver within a week]; but there may be more than one. Suppuration is generally preceded, especially in persons of sthenic constitution, by fever, pain, and other signs of active inflammation. Sometimes it assumes a more chronic form, and is insidious in its course, presenting no well-defined symptoms, or so slightly marked as to escape observation until its existence is revealed by bulging of the thorax, pointing in the epigastrium or below the ribs, or by sudden evacuation of the contents through the stomach, lungs, or bowels, or into the pericardium or peritoneum; or it may not be discovered until examination after death, perhaps from some other disease, when an abscess, sometimes old and encysted, may be found. Even a gnawing, deep-seated pain alone is in some cases the only symptom. The writer has recently had under observation a well-marked case of this kind. Liver abscess has frequently been detected where the symptoms were very obscure, indeed absent, and where it had not been suspected.

Prognosis of liver abscess of the simple form, if it be recognised and properly dealt with early, is not unfavourable. The evacuation of pus may take place spontaneously, through any of the channels before-mentioned; but if it can be detected and removed by puncture, the chances of recovery are enhanced.

It may be noted here that the few cases of single abscess that do occur in this country, to which no tropical origin can be assigned, are due to injury, ulcer of the stomach, of the gall-bladder or its duct, or to mischief resulting from embolism, phlebitis, or it may be to suppuration of the pelvic cavity, or of a hydatid cyst.

Secondly, liver abscess, following or coexisting with dysentery or tropical diarrhœa, regarded by Murchison and others as pyæmic, is

[1] Murchison, *Diseases of the Liver*, p. 198.

due to absorption of pus or septic matter from the ulcerated bowel, and therefore similar to the septic liver abscess of temperate climates. When multiple liver abscesses occur in a person who is or has recently been suffering from dysentery or chronic diarrhœa, complicated with ulcerated bowels, or fever with Peyerian ulceration, no better explanation of its origin could be given; such are very dangerous, though not necessarily fatal.

These abscesses begin as patches of necrosed tissue, embolisms, or infarcts, round which suppuration occurs, as with the core of a boil. This becomes the centre of a cavity, varying in size from a pea to an orange or larger, containing débris of tissue, puriform fluid, and probably staphylococci, the whole being surrounded by an area of congested and sometimes pus-infiltrated liver tissue. Such abscesses may occur also in the other viscera, lungs, spleen, or kidneys; they are septicæmic.

Of the 300 cases analysed by Waring, the number of abscesses was not stated in 12. Of the remaining 288, 177 had one abscess, 33 had two abscesses, 11 had three, 17 had four, 10 had from five to ten or more, and 40 had more than ten.

Thirdly, dysentery, malarial fever, hepatitis, and liver abscess may coexist with or supervene on each other, as results of climatic influences. In such cases it seems natural to ascribe the liver abscess to the dysentery; but it is probable that they are coincidences rather than consequences, and that the cause which affects the glandular structures of the large intestine may determine coincidently mischief in the liver. Such are obviously different from those before mentioned.

Fourthly, pyæmic abscesses which commence by necrosis of parenchyma, embolic deposits, or infarcts, and form cavities varying in size from a mere speck to a pea, a walnut, an orange, or larger. They contain débris of tissue, sanies, puriform matter, leucocytes, and micrococci. These abscesses are seen in various stages of development, and occur in other viscera as well as in the liver; they are truly septicæmic. Pathologically they are identical with the dysenteric liver abscess before mentioned.

This is the form that may originate in temperate climates as well as in the tropics, and is due to mechanical injury, to ulceration of the stomach or of the gall-bladder or duct, as a result of gall-stones or to ulceration in other parts of the body, and especially to phlebitis of the portal vein, or mischief in the pelvis or hæmorrhoidal region, as previously referred to.

The two following examples in the Pathological Museum, Calcutta Medical College, sufficiently illustrate the formation of pyæmic

abscesses in the liver, in one case as the result of amputation of the leg, in the other from injury to the bladder and rectum.

No. 1. Specimen showing several pyæmic patches in the lungs, *i.e.* portions of necrosed tissue, with puriform matter, surrounded by hyperæmic tissue, also the right lobe of liver occupied by large, so-called abscesses, and several small ones of similar character; enlargement of spleen, and firm fibrous coagula in right side of the heart. Taken from L. J., aged 22, who had sustained a severe compound and comminuted fracture of the right tibia and fibula, for which amputation was performed on 20th January 1868. Died of pyæmia on 2nd February.

No. 2. Portion of liver with numerous small pyæmic patches. Rectum and bladder showing recto-vesical fistula, from an Englishman, aged 44, admitted 16th December, with vesical calculus and fistula, the result of an injury, he having fallen on a sharp bamboo stake, which perforated the rectum and bladder, and was followed by calculus, for which lithotomy was performed. He died in a state of apnœa on 18th February. The pulmonary artery was plugged with firm decolourised fibrinous clots. Liver enlarged, with very numerous patches of dead liver tissue, with decomposed puriform fluid, around which suppuration was commencing. Lungs normal, spleen congested and softened.

Morehead says that, in malarious countries, liver abscess frequently occurs in association with remittent fever without any ulceration of the bowel. The combination of malarial poisoning with hepatic mischief is most frequent, and it is often difficult to decide how far certain rigors and chills may be due to malarial fever, how much to incipient suppuration in the liver, or to what extent both may be concerned. Of course, when there are physical signs of enlargement, pain, bulging, or pointing, there is less difficulty in diagnosis; but when these symptoms are absent, or only slightly expressed, as often happens, then the difficulty in diagnosing the true state of things is great, and exploration is needed to solve the doubt.

The coexistence of liver abscess with chronic diarrhœa should also be alluded to. In such cases the diarrhœa may depend directly on the liver mischief, and be kept up by pressure and irritation of the bowel. It may precede the formation of an abscess which sometimes takes place so insidiously as to escape notice until revealed by some of the symptoms previously described.

Liver abscess may, however, be unassociated with dysentery and diarrhœa, and yet be multiple; on the other hand, it may accompany or follow dysentery or diarrhœa, and yet be single. It is often difficult

to come to any decided opinion as to the true character, and each case requires careful study. The etiological relation of dysentery to liver abscess is always an interesting question. That they are dependent on each other has been held by high authority. Annesley considered that dysentery was due to hepatitis. Budd many years ago enunciated the opinion—which has received more support here than in India—that liver abscess is always due to purulent absorption from the dysenteric bowel; that, in fact, tropical abscess is pathologically identical with liver abscess in this country. Abercrombie thought they were coexistent, but independent of each other. Murchison, Macpherson, and others concur in this view. Sir R. Martin and others thought that liver abscess was intimately connected with disease of the cæcum; but this has not been altogether confirmed; for, of 72 cases where the cæcum was affected, in only 22 was there liver abscess. Moore says that eight observers out of twelve accord a percentage of 18 cases of liver abscess in 1532 of dysentery.[1] Of 44 specimens of liver abscess in the Pathological Museum at Netley, 11 were complicated by intestinal lesion.

Recent observations confirm the view of the relation of hepatic abscess to dysentery; and albeit in some cases of multiple abscess the condition is certainly due to septic absorption from a dysenteric bowel, yet frequently it is to be regarded as an expression of the general disease rather than as a direct consequence of dysentery. In temperate climates liver abscess is not always found connected with dysentery. Of many hundred cases of the latter which occurred in Millbank Prison during thirty years, Dr. Baly said that not one was complicated with hepatic abscess.

With reference to the relative proportion in which liver abscess and dysentery were coincident or combined in India, Dr. Waring, in 1854, made the following note:[2]—

Of 300 cases of fatal liver abscess in India, in only 82 cases, or in 27·3 per cent., was the hepatitis preceded by symptoms of dysentery. It would appear that in different hot climates liver abscess is more or less frequent according to circumstances, but that it is more prevalent in India than in any other country.

Brigade-Surgeon Lieutenant-Colonel Nash, Army Medical Staff, supplies the following information respecting the relative frequency of hepatitis, congestion, and abscess of the liver in European soldiers in foreign stations:—

[1] Moore, *Diseases of India*, 2nd edition.

[2] Waring, *An Inquiry into the Statistics and Pathology of Liver Abscess.* Trevandrum 1854.

Year.	Strength.	Disease.	Admitted.	Died.	Invalided.
		BENGAL.			
1888,	42,141	Hepatitis, Congestion, and Abscess of the Liver.	1026	63	43
1889,	42,083		979	55	63
1890,	41,147		826	51	72
1891,	Returns not received.				
		MADRAS.			
1888,	14,073	Hepatitis, Congestion, and Abscess of the Liver.	263	29	26
1889,	13,560		247	31	30
1890,	13,551		162	15	19
1891,	13,256		346	22	27
		BOMBAY.			
1888,	11,925	Hepatitis, Congestion, and Abscess of the Liver.	170	10	22
1889,	12,902		172	14	23
1890,	12,762		175	12	20
1891,	Returns not received.				
		CEYLON.			
1888,	1,133	Hepatitis, Congestion, and Abscess of the Liver.	20	3	...
1889,	1,146		16	1	...
1890,	1,028		15	...	1
1891,	1,251		15	...	2
		WEST INDIES (WHITE).			
1888,	1,164	Hepatitis, Congestion, and Abscess of the Liver.	2	...	...
1889,	1,061		6	...	...
1890,	1,019		5	1	...
1891,	1,096		1	...	...
		BERMUDA.			
1888,	1,346	Hepatitis, Congestion, and Abscess of the Liver.	6	...	...
1889,	1,320		1	...	1
1890,	1,546		3	...	...
1891,	1,601		3	...	...
		CANADA.			
1888,	1,329	Hepatitis, Congestion, and Abscess of the Liver.	2	...	...
1889,	1,422		6	...	...
1890,	1,381		2	...	1
1891,	1,424		2	...	...
		GIBRALTAR.			
1888,	4,736	Hepatitis, Congestion, and Abscess of the Liver.	...	...	...
1889,	4,761		7	1	1
1890,	4,659		10	...	...
1891,	4,682		12	1	1
		MALTA.			
1888,	6,353	Hepatitis, Congestion, and Abscess of the Liver.	31	3	3
1889,	6,516		34	4	...
1890,	7,055		64	3	...
1891,	7,697		53	2	3

Year.	Strength.	Disease.	Admitted.	Died.	Invalided.
		CHINA.			
1888,*	2,329	Hepatitis, Congestion, and Abscess of the Liver.	37	1	...
1889,	1,432		29	1	...
1890,	1,346		19	...	2
1891,	1,334		11	...	2
		STRAITS SETTLEMENTS.			
1889,	1,057	Hepatitis, Congestion, and Abscess of the Liver.	12	...	3
1890,	1,257		9	...	...
1891,	1,270		3	...	...
		MAURITIUS.			
1888,	405	Hepatitis, Congestion, and Abscess of the Liver.	2	...	...
1889,	524		3	1	...
1890,	516		10	1	1
1891,	580		7	1	...
		SOUTH AFRICA.			
1888,	3,717	Hepatitis, Congestion, and Abscess of the Liver.	26	2	4
1889,	3,397		14	...	3
1890,	2,961		10	1	2
1891,	3,185		10	...	3

* The figures in this year are for China and the Straits Settlements combined.

As to the extent to which hepatitis, which has an important bearing upon liver abscess, prevails in India, a late statistical report gives the following information. In the European army of all India, averaging 59,796 men, about 15,825 admissions and 803 deaths were recorded under the head of hepatitis during the ten-year period 1880–89. This gives an admission-rate of 26·8, and a death-rate of 1·34 per 1000. In the previous ten-year period there were 50 and 2·19 respectively.

In the army of Bengal the average admission-rate was 25·8 and the average death-rate 1·25 per 1000. In the preceding ten-year period the admission-rate was 47 and the death-rate 2·04 per mille. A considerable reduction has thus taken place in both the admission and the death rate.

In the European army of Madras the average admission-rate for the ten years 1880–89 was 36·9, and the death-rate 1·93 per mille; higher than those of Bengal. In the previous ten-year period (1870–79) the admission and death-rates were respectively 67 and 3·16 per mille; this, again, shows a great diminution in the frequency of the disease.

In the European army of Bombay the average rate of admission for the ten years 1880–89 was 21·4, and the death-rate 1·01 per mille; rather less than those of Bengal.

During the previous ten-year period (1870–79) these rates were respectively 41 and 1·71, showing that in this Presidency also there has been a considerable decrease in the frequency of the disease. Although these returns deal with hepatitis, it is fair to assume that the deaths were, as a rule, due to liver abscess.

In the last two Annual Reports of the Sanitary Commissioner with the army of India for 1889 and 1890, liver abscess uncomplicated with dysentery, and liver abscess associated with it, are dealt with separately from each other and from hepatitis.

In 1889 it appears that in the European army of India (strength 69,017) there were 125 admissions and 73 deaths from liver abscess, giving a rate of 1·81 admissions and 1·05 deaths per mille. Of these, 71 admissions and 40 deaths were in the Bengal army, giving an admission-rate of 1·65 and a death-rate of ·94 per mille; 26 admissions and 19 deaths were in the Madras army, that is, an admission-rate of 1·84 and a death-rate of 1·39 per mille; 28 admissions and 14 deaths were in the army of Bombay, giving an admission-rate of 2·17 and a death-rate of 1·08 per mille.

In the same year, from liver abscess associated with dysentery, there were 14 admissions and 13 deaths, giving an admission-rate of ·20 and a death-rate of ·18 per mille. Of these, 13 admissions and 12 deaths were in the army of Bengal, that is, an admission-rate of ·35 and a death-rate of ·27 per mille; 1 admission and 1 death were in the army of Madras, or an admission and death-rate of ·07 per mille.

In 1890 it appears that in the European army of India (strength 67,823) there were 97 admissions, or 1·42 per mille, and 43 deaths, or ·63 per mille, from liver abscess. Of these, 50 admissions, or 1·29 per mille, and 23 deaths, or ·55 per mille, were in the army of Bengal; 22 admissions, that is, 1·60 per mille, and 11 deaths, that is, ·80 per mille, took place in the army of Madras; and 25 admissions, or 1·95 per mille, and 9 deaths, or ·71 per mille, were in the army of Bombay.

In the same year, from liver abscess associated with dysentery, there were in the European army of India 24 admissions and 28 deaths,[1] giving an admission-rate of ·35 and a death-rate of ·41 per mille. Of these, 24 admissions, that is, ·58 per mille, and 26 deaths, that is, ·62 per mille, took place in the army of Bengal; and 2 deaths, that is, ·14 per mille, took place in the army of Madras. From Bombay there were none recorded. These were probably cases of liver abscess occurring during the course of dysentery, and proving fatal; but it is not likely that they represent all the cases of liver abscess due to dysentery, some of which must be included in the foregoing list; that is, in cases which are secondary to or

[1] Some of these deaths must have occurred out of hospital.

have been traced to dysentery, but in which that disease was not prominently present when the liver abscess occurred.

The death-rate in the European army from liver abscess (with and without dysentery) was, in 1889, 1·24 per mille; in 1890, 1·05 per mille, showing a decided decrease in mortality.

The Twenty-seventh Annual Report of the Sanitary Commissioner with the army of India for 1890 says: "The percentage of hepatic abscess in the total causes of death was much the same in the three Presidencies, that in Bengal being slightly the highest. The mortality per mille of strength from abscess of the liver was highest in Bengal, lowest in Bombay. Bengal proper had a mortality of 2·64 per mille from hepatic abscess, and 30 per cent. of all its deaths were from that disease; the corresponding figures for Quetta being 1·98 and 21·1, and for Burma 1·70 and 8·2." In the previous year Bengal proper had a mortality of 2·57 per mille, and 19 per cent. of all its deaths were from that disease. Burma had a mortality of 2·49 per mille, and 8·5 per cent. of all its deaths were from the same disease. From this it will be seen that the incidence and mortality from this disease vary considerably in the same locality in different years.

In 1889, in the native army of India (strength 128,642), there were 6 deaths from hepatic abscess, or ·05 per mille.

In 1890, in the Native army (strength 127,746), there were 4 deaths from hepatic abscess, or ·03 per mille.

The Annual Report of the Sanitary Commissioner with the army of India for 1890 says: "On comparing the statistics of the Native army with those of the European, it will be found that in the year under review the death ratio of the Europeans from hepatic abscess was about thirty-five times higher than that of the natives of India, it having been twenty-five times higher in the two preceding years; also that hepatic abscess contributed 7·6 per cent. of all the deaths of European soldiers, but only 0·2 per cent. of the deaths of native soldiers. In both the ratio of mortality to strength was reduced."

The relative position of liver abscess to some other diseases as a cause of death is shown in the following returns:—

DEATHS PER MILLE AMONG THE TROOPS FORMING THE EUROPEAN ARMY OF INDIA IN 1889 AND 1890.

Year.	Fevers.	Hepatic Abscess.	Cholera.	Dysentery.	Smallpox.	Diarrhœa.
1889,	6·92	1·24	1·13	·81	·25	·09
1890,	5·58	1·05	·72	·74	·06	·04

A return of hepatic abscess in the General Hospital of Calcutta during five years shows the following results (Birch):—

Twenty-three admissions—4 or 5 of which, however, were moribund when admitted—gave only 5 recoveries; 4 were punctured between the ribs—all died; 5 were punctured below the ribs—4 died; 1 recovery out of 9 operated on. The ages varied from 20 to 59 years; 18 were men, 5 were women, all Europeans or Eurasians. Here it may be noted that as regards the proportion of the sexes, liver abscess seems much more frequent among men than women; for instance, out of the 300 cases given by Waring, only 9 were women.

Of the 5 cases returned as cured, in 3 there was a history of dysentery. In 12 out of the 18 deaths, post-mortem examination was made; 5 showed dysenteric ulcers; and of the 6 cases in which post-mortem examination was not made, 4 had dysentery. In 3 cases the dysentery had immediately preceded or was intercurrent with liver symptoms; in the rest, dysentery had preceded at intervals of from a week to nine years.

These cases were apparently all of a severe character, probably not admitted until the disease was far advanced, and when septic conditions in the hospitals were more rife than they are now.

The following cases of tropical liver abscess treated at the Royal Victoria Hospital, Netley, were furnished by Surgeon Lieutenant-Colonel Stevenson:—

Year.	Remained.	Admitted.	Total.	Died.	Returned to Duty.	Invalided.	Discharged and Readmitted for Other Diseases.	Total.	Remaining.
1880, . . .	...	9	9	3	...	5	...	8	1
1881, . . .	1	6	7	1	...	5	...	6	1
1882, . . .	1	6	7	2	...	3	...	5	2
1883, . . .	2	...	2	1	1	...	...	2	...
1884, . . .	...	1	1	...	1	...	...	1	...
1885, . . .	...	7	7	3	...	1	...	4	3
1886, . . .	3	5	8	1	1	5	...	7	1
1887, . . .	1	11	12	2	2	7	...	11	1
1888, . . .	1	17	18	...	7	6	1	14	4
1889, . . .	4	21	25	1	11	12	...	24	1
1890, . . .	1	7	8	2	...	2	2	6	2

The following statement of deaths from acute hepatitis in the Madras General Hospital was calculated by Surgeon-Captain Youngerman:—

Year.	Admissions.	Deaths.	Percentage of Deaths on Total Annual Deaths.
EUROPEAN.			
1878, ...	18	3	3·9
1879, ...	18	2	4·3
1880, ...	23	3	5·2
1881, ...	11	3	5·1
1882, ...	11	3	5·2
Total, . .	81	14	...
NATIVES.			
1878, ...	14	3	1·5
1879, ...	10	3	1·8
1880, ...	31*	...	...
1881, ...	11	1	0·65
1882, ...	10	2	1·12

* The percentage mortality for 1880 is unreliable, because "hepatitis" was used in a more comprehensive sense than usual, and 2 cases in a dying state were removed by their friends.

Semeiology.—The early symptoms of hepatic abscess are those of acute congestion, *i.e.* enlargement and increased dulness of the liver, pain, and tenderness in the hepatic region, increased on pressure, defective movements of the ribs, and high temperature. There is cough, pain in the right shoulder, inability to lie on the left side owing to the weight of the swollen and dependent liver, nausea or vomiting, from pressure on the stomach or irritation of the vagus; a yellow tinge of the skin and conjunctivæ (sometimes, but not often, jaundice), and high-coloured urine, containing lithates. The pulse is rapid, the breathing hurried, the tongue coated, and in advanced stages sometimes red and dry. As the case progresses, rigors or chilly feelings make their appearance, followed by high temperature and sweating. These suggest, if they do not certainly indicate, suppuration.

These symptoms, together with bulging or pointing, in which the intercostal spaces are sometimes, though not always, obliterated, with increase of the area of dulness either upwards or downwards, leave little doubt of the presence of abscess. But there are cases in which the symptoms are not so well marked, and yet an abscess may have formed and escape detection until sudden bulging and fluctuation become apparent, or until it bursts into the bowels, stomach, or lung, or it may be into the pericardium or peritoneum; in the last two cases with fatal consequences, in the former with

great danger from shock or suffocation, but, happily, not often with immediately fatal results; for such not unfrequently recover.

The amount of pain in liver abscess depends a good deal on the part of the organ implicated. If the pus be deeply seated, or at a distance from the peritoneal covering, it is comparatively slight, or even absent. If it approach the surface, the pain is severe; and perihepatic abscesses may be the most painful of all. There is in any case a sense of distress and uneasiness, felt most distinctly when the patient turns on the left side,—with stitch in the side on taking a deep inspiration,—but also when on the right; decubitus is dorsal. In some cases there is continuous severe pain, like a neuralgic pain, in the side, even when other symptoms are wanting. Tension of the recti abdominis muscles, with a sense of nausea on pressure, is also a characteristic symptom.

It often happens that the patient is suffering at the same time from malarial poisoning, and is liable to recurrences of febrile paroxysms, which make it difficult to decide whether they indicate fever or suppuration; and where there has been no previous malarial fever, the rigors or chills of incipient suppuration are sometimes so slight as to have escaped notice, and the patient denies having had them at all.

Again, if the abscess be small and central, the pressure is so slight on neighbouring parts that the stomach is neither directly nor sympathetically affected, nor is there pain in the right shoulder, or in the hypochondrium in decubitus on the left side; these signs being either absent or so faintly expressed as to give little aid to diagnosis. The patient may be thought to be suffering from malarial cachexia and climatic deterioration of health only, till suppuration reveals itself, as before described; or it may not be discovered till after death, when an abscess, which may be encysted, is detected, or a contracted cavity containing a certain amount of caseous matter,—which reveals the former presence of pus,—the contents of which have undergone absorption. Such cases are rare, but doubtless they do occur. In these, after long quiescence, inflammation may light up renewed suppuration.

The value of the thermometer here is great, but cases do occasionally occur in which no rise of temperature has been observed; it is perhaps, however, the most reliable indication we have of the change going on in the liver, the absence of other symptoms making diagnosis so obscure. All one can do in these circumstances is to wait, and watch the temperature and pulse, with the state of the skin and excretions. Occasional sweatings, which may be far from well marked (sometimes they are even absent), will tend to establish

the probability of suppuration; and exploration, which should always be resorted to when there is uncertainty, may confirm it.

On the other hand, the matter may approach the surface insidiously, and reveal itself by bulging or pointing. Such are of the nature of the so-called cold abscesses, and are sometimes discovered accidentally.

With reference to the site of the abscess, it may occupy the right, sometimes the left lobe. It occasionally lies close to or upon the surface of the liver, in which case it is known as perihepatic abscess, immediately under the peritoneal surface of the liver, or between the layers of the peritoneum; or it is sub-diaphragmatic, and is limited by inflammatory exudation.

Again, there may be parietal abscesses, which, bulging in the hypochondriac or epigastric region, may be mistaken for true liver abscess.

Other symptoms, which are not generally characters of tropical liver abscess, are œdema of the lower extremities, enlargement of the superficial veins of the abdomen, and hæmorrhoids. Their occurrence is rare and accidental.[1]

Here may be noted the possibility of mistaking a distended gall bladder, a hydatid cyst, or malignant growth of the liver, for liver abscess (*vide* descriptions of these diseases). Empyema may also be mistaken for liver abscess, or the converse; in any case, evacuation of the purulent matter will be indicated.

Though liver abscess so frequently points either between the ribs or in the hypochondriac or epigastric region, when it is amenable to surgical interference, yet it does not always do so, and the cases are numerous in which, as before remarked, it takes a different course, and opens either into the lung, the stomach, the intestine, the hepatic veins, the cavity of the peritoneum, or even into the pericardium. In the three last cases the result is probably fatal, whilst in the former, the pus finding free exit, recovery is not unfrequent; but in any case the external pointing is preferable, and to be hoped for; and if the matter can be got out by surgical operation, the risk of internal opening should not be incurred.

Morbid Anatomy.—On examination of the ordinary tropical abscess after death, the cavity is found to be ragged, with shreds of tissue hanging from the surface. It appears as though the substance had been broken down and disintegrated. In some cases there may be signs of condensation, infiltration, and hyperæmia of the surrounding tissue, but frequently these are absent; and in others, where the abscess has been large, the lobe (generally the right) is flaccid and

[1] Murchison, *op. cit.*

collapsed, like the walls of an emptied cyst, with but little true liver substance remaining; and in cases where the liver has formed adhesions with the diaphragm, abdominal wall, or peritoneum, the parts are fused together. In the chronic form especially, the cavity of the abscess seems to be lined with a cyst of exudation.

Some of the specimens in the Pathological Museum at Calcutta well illustrate the morbid changes and also the various directions in which liver abscess opens spontaneously. The following are examples:—

1. *Single Abscess.*[1]—Abscess of the left lobe, destroying almost all its structure, except a lamina of its inferior surface. The wall of the abscess is irregular and ragged.

2. *Multiple Abscess, probably Dysenteric.*—A liver containing a number of small abscesses varying in size from a hazel-nut to a hen's egg. Walls shaggy and ragged.

3. *Large single Abscess.*—A large encysted abscess of the right lobe of the liver, forming a cavity large enough to hold a small human head. Complete destruction of all the tissues of the right lobe; left lobe, lobus Spigelii, and lobus quadratus unaffected. Walls of abscess strengthened by adhesions to surrounding parts. Interior of cyst lined by albuminous and fibrinous exudation, more or less ragged in parts.

4. *Two Abscesses communicating by a Sinus.*—An irregular superficial abscess of the liver, fixed to the neighbouring parts by adhesions forming the external opening of a deeper seated abscess in the interior. A canal led from these irregular ulcerations to the interior of the organ. The abscess reached the peritoneum, which was almost a line in thickness from organised exudation. The breaking down liver tissue round the cavity was nearly three-quarters of an inch thick, and the irregular opening in the centre, communicating with a deep-seated abscess, was about an inch across.

5. *Abscess opening into the Lung.*—Abscess of the right lobe of the liver opening into the corresponding lung. Appears to have begun in posterior part of liver; did not implicate that organ much, but burst into the chest through the diaphragm, forming with the lung a large cavity. There was an opening into the chest visible, and deposit of lymph on the pleura.

6. *Abscess opening into the Pleura and also into the Peritoneum.*—An abscess about the size of an orange on the convex surface of the liver opening through the diaphragm into the right pleural cavity

[1] These cases are recorded in the catalogue of the Pathological Museum, Medical College, Calcutta.

set up pleurisy; lung compressed, much lymph lining the diaphragmatic pleura. Abscess subsequently opened into the peritoneal cavity and caused death.

7. *Abscess opening externally, and also into the Pericardium.*—An abscess in the liver which was found to communicate with the cavity of the pericardium; it had pointed and been opened at the epigastrium, and 40 oz. of pus had been evacuated. There was about a pint of matter in the left pleura.

8. *An Abscess opening into the smaller Curvature of the Stomach.*—The abscess occupied the greater portion of the left lobe of the liver, and opened through the smaller curvature of the stomach, a large part of the walls of which had been completely destroyed. The portion of the parietes of the stomach supplemented by the contracting wall of the abscess was uneven and somewhat roughened. The right lobe was studded with small abscesses.

9. *Abscess opening into the Duodenum and Stomach.*—In this there were three abscesses; the first, an old abscess, had strong adhesions to the false ribs on the right side. These gave it the appearance of an aneurismal cyst, projecting on the right of the gall-bladder. This old abscess was empty, containing only a little yellow fluid mixed with flocculence. It was lined by a sort of mucous membrane (?). This had been evacuated by puncture. Secondly, another abscess of more recent date, not larger than an orange, had opened into the duodenum just below the pylorus, immediately to the left of the gall-bladder, to which last the duodenum was intimately adherent. Thirdly, another abscess formed in the centre of the left lobe, and then extended upwards, causing absorption of all the structures till it reached the diaphragm, where it adhered strongly, and seems to have directed its course as if to open at one point into the pericardium, for it perforated the diaphragm. The pericardium and lungs were both strongly adherent to the diaphragm. It also extended downwards, opening into the stomach about 3 inches below its cardiac orifice. The mucous coat of the stomach was ulcerated, and its outer cellular coat was reduced to a sloughy state and hung about the opening.

10. *Abscess opening into the Duodenum and Vena Cava prior to its passage through the Diaphragm.*—The cavity of the abscess, immediately behind the vena cava, would admit an orange. Its margins were ragged. About half an inch nearer the convex surface there was a small encysted abscess having no communication with the former. The parenchyma of the liver had undergone cellular or lobular degeneration, and presented a spongy appearance.

11. *Abscess communicating with the Colon.*—A large abscess of the

right lobe of the liver opening into the transverse arch of the colon.

12. *Opening of a Blood Vessel into a Hepatic Abscess.*—Part of a large hepatic abscess in which death occurred from the opening of a blood vessel, which is shown in the preparation.

13. *Multiple Abscess from Dysentery.*—Liver of a patient who died from dysentery. Isolated portions of gangrenous tissue are seen, and in some places this has become enucleated during maceration.

14. *Dysenteric Multiple Abscess.*—Liver of a European who died of dysentery, displaying remains of two old, small, centrically situated hepatic abscesses. There is a considerable amount of fibrous deposit around both the small cavities. There are no recent abscesses in the liver.

15. *Cicatrisation of Liver Abscess.*—A portion of the liver demonstrating the cicatrix of an old abscess on the upper surface of the right lobe, near the longitudinal ligament and the anterior margin. The cicatrix has been adherent to the contiguous parts, and in the process of detachment the adhesions have been torn. The puckering of the liver is well manifested.

16. *Single Abscess bursting into the Peritoneal Cavity.*—Abscess in the left lobe of the liver. It pointed at the umbilical region, burst into the peritoneal cavity, and caused death.

17. *Single Abscess.*—A large abscess in the left lobe of the liver endeavouring to make its way through the diaphragm, which is agglutinated to the wall of the abscess on the abdominal, and to the pulmonary pleura on the thoracic side.

18. *Double Abscess.*—A large abscess in each of the large lobes of the liver, causing almost entire destruction of the hepatic structure. Liver completely agglutinated to the stomach.

19. *Cavity of Abscess showing Portal Vein passing through.*—A large non-encysted abscess in the right lobe of the liver with honeycombed and ragged margins, through which a large portal vein passes; and a smaller encysted abscess, in the same lobe, the interior of which is puckered from the emptying of its contents.

The ragged walls of the cavities, when exposed, are covered with a layer of pus, stained and discoloured by blood, often of a chocolate colour, or sometimes tinged with bile, which has a peculiar faint, but not putrid, odour. It contains pus corpuscles, disintegrated liver cells, shreds of cellular tissues, and probably micro-organisms.

Septicæmic abscesses are often near the surface, and when laid open the contents are the same as already described.

The following interesting memorandum by Professor Macfadyen,

gives an account of the micro-organisms found in connection with liver abscess, and throws much light upon its etiology.

Notes on the Bacteriology of Tropical Abscess of the Liver.

It is now an established fact that the production of pus in the human organism is due to the action of bacteria.

It is true that under certain conditions pus may be formed in the tissues without the direct intervention of bacteria. The injection of certain chemical irritants under the skin gives rise to local abscesses. Thus oil of turpentine or ammonia, when subcutaneously injected into animals, produce pus. This result is, however, produced artificially, and may be described as merely an interesting laboratory experiment. Under natural conditions, the formation of pus is due to the direct action of bacteria on the tissues.

The pyogenic or pus-forming bacteria have been specially studied by J. Rosenbach and Passet, to mention only two names amongst a host of investigators. They isolated the pyogenic organisms from pus, and cultivated them on various nutrient soils, such as peptone, beef broth, gelatine, agar-agar, etc. In this way the morphology of these organisms was carefully worked out. Their pathogenic action was also investigated by direct experiment on the lower animals. Suppuration was produced in animals inoculated with pure cultures of the organisms. The causal relation of the organisms to the process was thus clearly proved.

The majority of the pyogenic micro-organisms belong to the group of the staphylococci. The spherical cells are arranged in a characteristic fashion, somewhat resembling clusters of grapes, and hence the name "staphylococci." The organism most commonly met with in pus is the *Staphylococcus pyogenes aureus*. It is the most virulent of the pyogenic bacteria. The growth of this organism on agar-agar or potato has an orange-yellow colour.

Other staphylococci are also met with in pus. The next in importance to the *aureus* is the *Staphylococcus pyogenes albus*. As the name implies, its growth on nutrient soils has a white instead of a yellow appearance. It is less virulent than the *Staphylococcus pyogenes aureus*. The following names have been applied to other sub-varieties, which are occasionally met with:—

Staphylococcus pyogenes citreus.
Staphylococcus cereus albus.
Staphylococcus cereus flavus.

It will suffice merely to mention here another important pyogenic organism, the *Streptococcus pyogenes*. The cells of this organism are grouped together so as to form chains, hence the name "streptococcus." It closely resembles the streptococcus of erysipelas, and many observers believe the two to be identical.

The *Staphylococcus pyogenes aureus*, as already stated, is the most virulent member of the group of the staphylococci. It is capable of producing the most varied morbid processes in the body. When subcutaneously inoculated into rabbits and guinea-pigs it produces local abscesses. It causes death when injected directly into the blood stream of a rabbit. On post-mortem examination the appearances are found to be those of a general purulent inflammation. The joints are affected, and metastatic purulent deposits and infarctions are found in the organs, especially the kidneys. The rabbit succumbs to the pyæmia in two to three days. Before

death, convulsions may occur. In the muscles there are yellow deposits of pus which contain the staphylococci. They are found in the myocardium. The pericardium may be filled with fluid. The kidneys contain a number of yellow spots, which usually have a wedge shape. The micrococci form emboli, and the tissues in their immediate neighbourhood become necrotic. The multiple abscesses so formed contain the cocci, and they can be re-cultivated from the pus.

As regards their action in the human body, a few words must suffice. The *Staphylococcus aureus* is generally found in boils and carbuncles. Garré, by rubbing the organism into the skin of his forearm, produced a purulent inflammation of the tissues of the skin. In this instance the cocci probably made their way into the deeper layers of the skin through the glandular ducts.

The *Staphylococcus aureus* is further found in acute abscesses, impetigo, sycosis, acute infectious osteomyelitis, purulent glands, empyema, pus in joints, tonsillar abscesses, mammary abscesses, strumitis, idiopathic cerebro-spinal meningitis, etc.

"Mixed infections" sometimes occur, the cocci appearing in the course of some other affections, *e.g.* in typhoid fever and the variola pustule, with suppuration as a sequel.

The *Staphylococcus aureus* is also found in cases of ulcerative endocarditis. Orth and Wyssokowitsch succeeded in producing an acute ulcerative endocarditis in rabbits with cultures of the organism. Previous to injecting the cocci into the blood stream, they artificially produced a lesion in one of the valves of the heart of the rabbit.

To sum up, the staphylococci are usually found in circumscribed abscesses, whilst the streptococci are generally to be met with in spreading suppurative processes. The pus streptococci are also found in puerperal pyæmia.

It will be seen, then, that the pyogenic cocci may produce at one time a strictly local formation of pus (boil, etc.), and at another time a general formation of pus throughout the body. The result varies according to the channel of infection. Thus Garré, by rubbing the staphylococci into his arm, produced local abscesses. The subcutaneous inoculation into rabbits had the same result; whilst the injection into the blood stream gave rise to metastatic pyæmic abscesses.

Some interesting researches have been made with regard to the causation of abscess of the liver.

I will first mention the investigations of Netter. This observer found that after ligature of the bile duct, the gall-bladder did not remain sterile—micro-organisms appeared in it. They were a bacillus, and the well-known *Staphylococcus aureus*.

The bacteria, after ligature of the bile duct, did *not* remain localised in the gall-bladder. They penetrated into the liver and the blood. When the infection was due to the *Staphylococcus aureus* alone, abscesses were produced in the liver and other organs.

Netter also found the "aureus" in the blood of a patient after an attack of colic due to gall-stones. The cocci were found in the blood and liver in a case of icterus gravis. This patient had an alcoholic cirrhosis of the liver. In cases of *obstruction* of the bile duct, the cocci were present in the bile channels.

In morbid conditions of the bile channels, the *Staphylococcus aureus* undoubtedly plays an important *rôle*. And having gained entrance to the

bile channels, the danger is always present of its migrating to the liver and inducing there suppuration.

Kartulis states that in dysenteric liver abscesses he constantly found amœboid organisms — "dysentery amœbæ." He did not detect these amœboid organisms in "idiopathic" liver abscesses, though he found pyogenic bacteria. Cultivations were made by him from the pus of these abscesses. In one instance the *Bacillus pyogenes fœtidus* was found, and in another the *Staphylococcus pyogenes albus*. In two cases the results were negative.

Kartulis's investigations were made on Egyptian dysentery, complicated by liver abscesses. He divides tropical abscesses into two kinds—

1. The idiopathic.
2. The dysenteric.

The first form, he thinks, is probably due to bacteria alone. The second form is probably due to amœboid organisms, which gain entrance to the portal vein system through a lesion of the intestinal wall. Kartulis believes that these amœbæ may carry with them the pyogenic organisms to the liver. In short, the dysenteric liver abscess is due to pyogenic bacteria, carried thither by amœbæ from the digestive tract.

Animal experiments gave negative results, and attempts to cultivate the amœbæ outside the body have not yet been successful.

Osler records two cases of dysentery with liver abscess. In the pus he found amœboid bodies. They were about twelve times as large as a white blood cell, and showed active amœboid motions. He also found them in the fæces, which were no longer distinctly dysenteric.

Kartulis likewise found amœboid organisms in the intestines in cases of Egyptian dysentery. Their causal relation to the disease has not yet been proved. Indeed, it may be that more than one organism can produce diarrhœa or dysentery. Any morbid condition which weakens the resistive power of the intestinal mucous membrane will favour the penetration of microbes from the lumen of the intestine into its walls.

The writer was able to make a bacteriological examination of a case of tropical abscess of the liver. A gentleman invalided from India was operated on in London. A large, single abscess was found situated in the right lobe. Samples of the pus were collected in sterilised glass tubes, and examined immediately after the operation. Under the microscope the fresh pus was found to contain pus cells, fat cells, blood cells, and liver-tissue débris. The pus likewise contained large numbers of staphylococci. No other bacteria were present. Stained specimens yielded the same results—only staphylococci were visible. A careful search was made for amœboid organisms, but none were found. The only organisms then found, on microscopical examination, were the staphylococci.

Cultivations were next made from the pus on agar-agar and potatoes. A small portion of the pus was smeared over the surface of the agar or the potato. The tubes were then placed in an incubator at blood heat. At the end of twenty-four hours there was a bacterial growth in all the tubes. This growth had an orange-yellow colour, and consisted entirely of staphylococci. No other organism grew on the agar or potato. The pus therefore yielded a pure culture of the *Staphylococcus aureus*.

Gelatine-plate cultures were also made from the pus. The gelatine plates likewise yielded pure cultures of the *Staphylococcus aureus*. In both instances, therefore, the *Staphylococcus aureus* was the *only* organism found in the pus.

Control experiments were made on animals. The subcutaneous inoculation of pure cultures obtained of the staphylococcus, produced local abscesses in rabbits. There was therefore no doubt that the organism isolated was the *Staphylococcus pyogenes aureus.* In this case, then, the writer was unable to find the amœboid organisms of Kartulis and Osler. It was a typical tropical abscess, and contained only the *Staphylococcus aureus*, which had undoubtedly produced the suppuration. There are two ways in which the cocci might find their way from the intestine into the liver. (1) By absorption from an ulcerated intestine; (2) through the bile channels when obstructed or diseased, as Netter's experiments prove.

It is not therefore necessary to suppose that the predisposing cause must in every case be some dysenteric lesion of the intestine. The bile channels would afford a convenient channel for the migration of the staphylococci to the liver.

Treatment.—The treatment in the outset of the acute form is that of congestion; rest and restricted diet, free purgation by occasional mercurials and salines; chloride of ammonium and ipecacuanha; hot fomentations; and where the pain is severe and the temperature high, a few leeches on the side or to the anus may be useful. The former modes of treating acute inflammation of the liver by general bloodletting and mercurialisation are obsolete. When pus has formed, endeavour to procure its evacuation as soon as possible. When any doubt exists as to its presence, explore with an aspirating cannula needle; there is little danger in this proceeding if carefully performed. Slight inflammation may follow, but it will soon subside; and even if no pus be detected, it sometimes seems to do good by relieving tension and abstracting a certain amount of blood.

In the Annual Report for 1891 on the Civil Medical Department of Hyderabad, Dr. Laurie gives a table showing the method of treatment and the result in twenty-six cases of liver abscess which were aspirated antiseptically. Of these four died.

These show the value of aspiration in liver abscess. The rule, then, is to aspirate all cases of abscess of the liver unless the pus is pointing at the surface; and many cases are cured in this way without exposure to the risks of an incision. If incision subsequently becomes necessary, the previous aspiration of the abscess is not found to prejudice the patient's chance of recovery. Dr. Laurie adds that tartar emetic is used very freely with excellent results, in large and small doses, in all cases of liver abscess which are not of specific or septic origin.

The writer of this article is a strenuous advocate of puncture for explorations, and has occasionally seen one aspiration followed by recovery; but the more common event is for the abscess to fill up again if not opened by incision immediately pus is detected, and

later the exploratory puncture of aspiration is followed by incision and free opening.[1]

In exploring with the aspirating needle or in making incisions, it is sometimes an anxious question as to whether adhesion has taken place between the liver and the parietes or not. As a general rule, little or no danger is to be feared in puncturing the liver if the patient be not the subject of cachexia from splenic leucocythæmia, in which case there is a certain amount of danger of hæmorrhage, and of the blood finding its way into the peritoneum or pleura. Such, indeed, has been known to occur; and after death, which quickly followed the puncture with an aspirating needle, blood was found extensively extravasated and coagulated in laminæ in the peritoneal cavity. Generally, however, even though adhesion may not have occurred between the liver and the parietes, there is little or no danger, and the operation should be resorted to when doubt exists as to the presence of pus; profuse hæmorrhage, if a large vein has been opened, may rapidly fill the aspirating bottle; but either there is no escape of blood into the cavity, or if there be any, no evil results; whilst in the puncturing of the viscus itself and the withdrawal of blood, there is often benefit, for a swollen, painful liver is not unfrequently relieved by this proceeding, and it is followed by subsidence of the symptoms; indeed, it has been recommended for this purpose on the high authority of Dr. G. Harley. In any case,—in one of cachexia, splenic especially,—though it would be subject of anxiety, if it be deemed essential for exploratory purposes it should be resorted to, and it would in such a case be well not to withdraw the needle for some time until exudation and coagulation shall have prevented danger of extravasation.

Should the necessity for the removal of the pus be urgent, and should adhesion not have taken place, the pus should be withdrawn through a large cannula, which should be retained *in situ* and allowed to act as a drainage tube. It is, however, well to wait as long as possible to allow of consolidation; but interference should not be too much deferred, lest fever, exhaustion, and septicæmia render the case hopeless.

As the abscess increases in size, more hepatic tissue is destroyed; constitutional irritation, fever, and hectic, with danger of pyæmia increase; the ribs are eroded, and the liver may be reduced to a mere sac containing pus (and débris), adjacent organs being compressed and adherent.

In cases where the pus lies deep with a quantity of liver substance intervening, it sometimes becomes a difficult question to

[1] *Lancet*, August 20, 1892.

decide when the opening should be made; but the sooner it can be done with any prospect of safety from hæmorrhage, the better.

A considerable quantity of liver substance may be cut through, yet the hæmorrhage, which may be very profuse, soon ceases; this, however, cannot always be relied on, especially in persons who are already weak, exhausted, and anæmic. In such cases, therefore, it is better to puncture the liver with a large grooved trocar and cannula, and leave the latter in for a day or two or more, allowing the pus to drain through it. After that time the opening can be increased by dilatation and a larger tube introduced. For this purpose a grooved cannula has been devised by the writer of this article, which can be fitted on to the aspirator, and serves the purpose indicated. Along the groove can be passed, either a narrow knife or a pair of dilating forceps, by which the opening can be enlarged as they are withdrawn, in the latter case without cutting and causing hæmorrhage. In a large proportion of cases this danger of hæmorrhage does not occur.

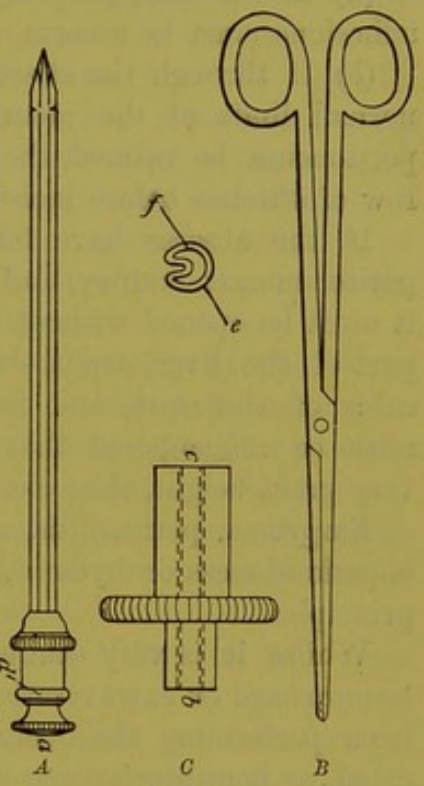


FIG. 50.

A, Grooved trocar and cannula (½ nat. size).
B, Dilating forceps (½ nat. size).
C, Section of the piece of metal into which the trocar fits (nat. size).
a, The head of the trocar. When this is withdrawn the aspirating tube can be fixed into the cannula by means of a small piece of metal *b*, *c*, of which one end *c* fits into the cannula at *d*, and the other end *b* fits into the aspirating tube. *e*, Section of cannula. *f*, Section of trocar in cannula.

The bulging indicates the point at which the incision has to be made; it is generally free from danger, adhesion having probably formed.

Possible risks from the extravasation of pus, should such be found, when the liver is punctured, have to be considered. Upon this subject Murchison says with reference to the escape of pus into the peritoneum, and consequent fatal peritonitis:[1] "In most cases, however, when the abscess is near the surface, the adhesions would prevent the entrance of pus into the peritoneum. Morehead speaks of absence of adhesions as quite exceptional (in only three out of seventy-six fatal cases). Moreover, if desirable, it is always possible to produce adhesions; and in exceptional cases, where none exist, it will be prudent to produce them by the local application of caustic

[1] Murchison, *Diseases of the Liver*, 3rd ed. p. 208.

potash before puncturing; but when the puncture is made in an intercostal space, this proceeding is unnecessary."

Mr. Godlee, who has had much experience in the surgical treatment of liver abscess, makes the following judicious remarks (*vide British Medical Journal*, Jan. 1890) as to the precautions to be adopted:—

(*a*) If the abscess present at the epigastrium, the presence of adhesions must be ascertained before incising the liver.

(*b*) If through the chest wall, a spot must be chosen below the normal limit of the pleura; but if, by chance, either pleura or peritoneum be opened, the opening must be closed with a double row of stitches before incising the liver.

If the abscess have burst into the lung, pleura, pericardium, peritoneum, or kidney, and its position can be clearly determined, it must be opened without delay. Hydatids of the upper and back part of the liver are to be treated upon the same lines; but in cases of this sort, and in those of sub-diaphragmatic abscess, it must be remembered that the diaphragm may be pushed up to a very great height, thus closely simulating intra-pleural suppuration.

Empyema, pericarditis, and peritonitis, caused by rupture of a hepatic abscess or hydatid, must be promptly dealt with on general principles.

Whilst it is very desirable to avoid risks from intra-peritoneal hæmorrhage or extravasation of pus, these risks must not deter one from performing the operation when the necessity for it is indicated, as non-interference would involve the patient in even greater danger. It is a choice of evils, and the least should be chosen. This is illustrated by the fact that of 203 cases collected by Rouis where the abscess was not opened, 162 (or 80 per cent.) died.[1]

The apparent proximity of pus to the surface being somewhat delusive, in cases of doubt as to the extent of liver-substance intervening, it is well to introduce the grooved cannula after an incision has been made through the parietes. As before said, the bistoury or dilating forceps can subsequently be used to enlarge the opening sufficiently to admit of the entrance of a large drainage tube; this will obviate any danger of hæmorrhage, and free exit will then be given to the pus; nor should the tube be removed, except occasionally for purposes of cleansing, until the discharge has diminished to a serous oozing, and the tube itself has been gradually extruded by the contraction and granulation of the cavity.

Here it may be noted that as the cavity contracts, the tube is

[1] Murchison, *op. cit.*

apt to get twisted upwards or downwards, displaced, even extruded, when it may be very difficult to replace it. In such cases, should there be any lodgment of pus, the opening must be enlarged, and it is absolutely necessary that the tube should be reintroduced. It may even possibly be necessary to make another opening.

The tube may be kept *in situ* by a flange or safety pin, to which two silk threads are attached, which may be secured to the side by sticking plaster.

During the process the tube should from time to time be shortened by cutting, in order to prevent any irritation arising from its pressure against the walls of the contracting cavity.

It occasionally happens that an abscess may open into the bowel or lung, and yet also point at the surface; and the question of adding an external opening to that which already exists internally may arise.

Should it so point, and especially if the discharge of pus internally be restricted or intermittent, and a high temperature or sweating indicate its reformation and lodgment, a counter opening should be made through the bulging. The extent of mischief and the probability of the abscesses being multiple, renders the prognosis in such cases very doubtful, though not quite so hopeless as it would be if the opening had not been made.

In short, in dealing with liver abscess, the following principles should be observed:—

1. That directly it has been ascertained that pus has formed, an opening should, if practicable, be made to give exit to it.

2. That a drainage tube sufficiently large to give free exit should be kept in the wound.

3. That strict attention should be paid to ensure the free exit of pus, and the prevention of decomposition in the cavity.

4. That whilst sedulous attention should be given to perfect cleanliness, and usual antiseptic precautions in the dressings, it is not desirable, so long as the pus is untainted, to wash out the cavity with any fluid, antiseptic or otherwise; though, in the event of decomposition having taken place, it may be expedient to do so with a weak solution of some non-mercurial antiseptic.

As regards the dressing of the wound after the opening of the abscess, the indications are to insure a free discharge and no lodgment of pus; to change the dressings sufficiently often to obviate uncleanliness or decomposition of the discharges, and the entrance of septic matter. The margins of the wound and its vicinity should be carefully washed with solution of carbolic acid, 1 to 40 or 60, or solution of bichloride of mercury, 1 to 1000

or 2000, or boracic acid, eucalyptus, or other antiseptic. If decomposition should occur, wash out the cavity with a weak solution of carbolic acid, peroxide of hydrogen, or eucalyptus, but not the mercurial solution.

For the external dressing, various forms of gauze may be used; the carbolic acid, eucalyptus, salicylic wool, or other forms of antiseptic, according to the fancy of the surgeon. These should envelop and cover the wound sufficiently to absorb all discharge, and keep the parts perfectly free from taint. It should be changed only just as often as circumstances require. In the outset, whilst the discharge is free, the dressing may have to be changed twice a day, for some time daily, and later at longer intervals.

As regards the site of puncture, it may be made in any part of the hypochondriac region, between the ribs, below the cartilages, or in the epigastrium, according to the position in which the abscess points. It not unfrequently happens that it does not obviously point anywhere; but if bulging of the side and other indications of matter having formed be present, and the exploring needle, whether introduced between the ribs or below the cartilages, have confirmed the formation of pus, if firm pressure of the point of the finger between the ribs localise the pain in one particular spot, that point must be selected as the one through which the opening is to be made. This indication, from deep pressure of the finger between the ribs causing pain, is a valuable diagnostic symptom of the presence of pus in the liver. Here it may be noted that the intercostal spaces frequently do not bulge, when abscess has formed.

It is recommended by some authorities to make a counter opening in liver abscess. When the abscess has not pointed posteriorly, no doubt such may occasionally be necessary, and if the drainage from the first opening is not free and effective, it should be made in the most depending spot. But if the first opening be free, and the tube sufficiently large, no difficulty, as a general rule, will be experienced; and it must be remembered that a second opening would not always be free from danger. Nevertheless, it is a sound surgical principle, applicable here as elsewhere, and should be resorted to if any difficulty of drainage or reformation of pus suggest its expediency.

After opening between the ribs, portions of necrosed bone may come away; such cases though dangerous are not always fatal, and an instance in which recovery occurred is recorded by the writer.

As the tube is likely to be obstructed by inspissated pus or flocculent matter in the cavity, the greatest care should be taken to keep the passage through it clear, so that the pus may have free exit, as its retention from any cause is certain to be followed by

recrudescence of febrile symptoms indicated by rigors, rise of temperature, and sweating. Such symptoms arising when the tube appears to be clear and the discharge has ceased, may indicate the formation of another abscess. The tube must then be completely withdrawn, fresh exploration must be made with the needle, or it may be a long probe, which, perhaps breaking down an intervening septum of liver tissue, will give exit to a fresh collection of pus, and so evacuate the contents of another abscess, when renewed attention must be directed towards securing free discharge therefrom.

Throughout the whole of the surgical treatment, the greatest care must be taken as to antiseptic precautions, by washing the surfaces with a view to preventing the access of septic matter or micro-organisms. No special treatment beyond attention to the diet and the state of the bowels is, as a general rule, necessary; restlessness, sleeplessness, or pain may be relieved by chloral, morphia, or some other sedative. Some simple tonic with quinine, especially if there be any febrile tendency, may be useful. Quiet and care in avoiding chill are essential until convalescence is well advanced.

If any symptoms of dysentery or chronic diarrhœa continue, it would be better to keep the patient on a strictly milk diet, administered in small quantities of 3 to 4 oz. every hour; and if depression should indicate necessity for stimulants, a little brandy should be added to the milk, whilst doses of compound ipecacuanha powder may be administered to the extent of 5 or 10 grains two, three, or four times a day, as circumstances may seem to require.

The previous history of the case will serve to some extent as a guide to the nature of the abscess; if it be single, and dealt with early, and if no complications arise, the prognosis is favourable; another abscess may form, and still recovery occur. But in cases where multiple abscesses are of the septicæmic character before alluded to, the prospect of success after puncture is very small; indeed, in such the operation would seldom be performed unless one of the abscesses, having attained considerable development, point; when relief, though it may be only of a temporary character, would be given by emptying it of its contents.

The question whether liver abscess be single or multiple is always difficult, and one about which it is almost impossible at first to form an exact diagnosis. Generally, the evacuation of pus is followed by an immediate fall of temperature, which remains at or even below the normal standard, provided the discharge be free and the pus protected from decomposition. A rise in the temperature, or its remaining high, with recurrence of pain, fever, and sweating, gives cause for apprehension, either that pus is being reformed and lodged

in the original cavity, that it is decomposing, or that a fresh abscess is forming. In any case, a strict watch must be kept on the symptoms, and one must interfere as early as possible to give exit to newly formed pus, or to remove decomposing discharge from the cavity. If the symptoms of suppuration continue, although no physical sign may betray the presence of pus, explore the liver freely with the aspirating needle. As the wound heals the diet may be increased, but the patient must be protected from all excesses or errors in food.

But it must be remembered that if it be impossible to diagnose the situation of the pus, operation should be withheld so long as the patient is not losing ground.

It may be here well to note the necessity for caution in using the exploring needle. In the struggles of the patient under the influence of the anæsthetic, he may roll over and break the needle, incurring the risk of a portion being left in the wound, or the liver substance being torn. The breaking of the needle has so happened in the experience of the writer of this article.

The method of operating for hepatic abscess at the Medical College Hospital, Calcutta (according to Dr. McConnell), is as follows :—

Always explore first, either with a hypodermic syringe (with long needle or cannula) or aspirator. Finding pus, use either a large sized trocar and cannula, or more commonly, simply cut down on abscess (guided by the exploring needle) and having reached it thus, withdraw the knife and push in a long probe. Run in an india-rubber drainage tube (the largest size, usually as thick as a finger), and drain the abscess.

The practice of washing out the cavities has been given up, unless the pus is fœtid. It is found that cases do better without washing out, even by antiseptic lotions.

The tube is fixed in by a safety pin, which is secured by strips of sticking-plaster. Iodoform dusted over the wound in the skin, then boric lint applied over a space of about 6 inches square, surrounding the tube, and over the latter, loosely packed, perchloride (of mercury) gauze. If discharge is likely to be copious, carbolic tow is placed over the gauze; finally, a broad bandage completes the dressing.

Large abscesses have, for the first few days, to be dressed twice, others only once a day. As the abscess contracts the tube is shortened, and the dressings changed every second or third day. Then as further contraction occurs, the tube is replaced by a narrower one, and so on till complete healing and cure is effected.

Opening is preferred through the abdominal wall, and not between the ribs, unless the abscess decidedly points there. When it does so necrosis of one or more ribs has often already taken place, but it is not found (at the hospital) that this occurs *as a consequence of drainage*, for soft rubber tubes are used, as they do not cause any marked pressure on surrounding parts. But the objection to opening between the ribs is that the latter fall together as the abscess contracts and the tube gets nipped, or the liver contracting, the channel or track of the abscess changes and

becomes tortuous, and so drainage becomes difficult and a counter opening necessary.

Resection of one or more ribs is also resorted to (at the hospital) for freer drainage, but hitherto cases have not done well with this procedure. Perhaps this may be because only very large abscesses require such resection, and probably most of these cases are bad ones, *i.e.* low vitality and exhaustion from size of abscess and extent of suppurating surface.

The fact remains that (in the hospital) resection of ribs has not been found useful in dealing with hepatic abscesses, and simple drainage by means of india-rubber tubes is preferred.

As regards the duration of tropical abscess in the liver, it may terminate fatally, or evacuate within three weeks from the outset of the symptoms. It is less rapid, as a general rule, than the pyæmic abscess. Murchison has pointed out that it may extend over two, or three, or even six months; and cases occur where a small tropical abscess, with thick organised walls, has existed for months or even years in a cold or quiescent form, and subsequently enlarged and burst.[1] Cases are occasionally met with in this country where a large abscess has formed in the liver of a person years after return from India.

Here it may be noted that abscesses may be absorbed. Symptoms during life pointing to suppuration have in a few exceptional cases subsided and health been restored, whilst in other cases post-mortem appearances have revealed contracted cicatrised cavities which indicated the former presence of pus. In any case a liver abscess is a most serious affair, and the result is uncertain; but by observing the principles and practice inculcated, considerable success may be hoped for.

A liver abscess should never be allowed to point and cause gangrene of the abdominal wall from pressure, and consequent inevitable death from exhaustion; cases have not unfrequently occurred where a simple incision, made earlier, would in all probability have relieved suffering, and might have saved life.

The question may arise, after recovery from liver abscess, whether the subject of it should return to a tropical climate. No doubt, were considerations of health alone concerned, it would be better not to do so; but in many instances of officers and others in the public service, whose interests are centred in localities where they contracted the disease, it may be satisfactory to know that such persons have returned to India and other foreign stations, and have not suffered from any return of hepatic disease. Past experience would naturally impress upon such persons the necessity for extreme caution in their mode of living,

[1] Murchison, *op. cit.*

and in avoiding whatever might tend to provoke a recurrence of the disease.

Illustrative Cases.

The following cases illustrate the chief points of etiological, pathological, and medical and surgical interest of tropical liver abscess:—

No. 1.—Liver Abscess. Opened below the Ribs. Recovery.

An English officer, aged 29, was exposed to the sun in July, in India, during the greater part of an afternoon. This was followed by severe headache and general malaise, and a few days later by severe pain in the right side. The pain increased, and there was fever; leeches were applied over the liver; in the evening there was a rigor. During the next few days the pain increased; more leeches, aperients, and salines were administered, and the pain became diffused, with feeling of weight in the hepatic region. Blisters were then applied. The patient was weak and anæmic. The liver was large, and just below the last rib there was a fluctuating swelling. Pulse 100, skin cool and moist, no pain, but uneasiness and sense of fulness in the side; breathing slightly embarrassed. Tongue clean, bowels costive, appetite fair. The abscess was opened, and 18 oz. of thick, dark-coloured pus withdrawn through a cannula. The cavity was washed out with a solution of carbolic acid, one drachm to a pint of water. The cannula was left in the wound.

The next day it was removed, as it caused irritation, and the wound kept distended with a plug of lint soaked in glycerine and carbolic acid. Quinine and sulphuric acid were ordered, and the bowels kept open with sulphate of magnesia. The appetite was good; pulse 88; no fever. The discharge of pus gradually diminished, the patient continued to improve, and sailed for England six days after the operation. On reaching Ceylon he was able to take a walk on shore.

This is a good example of simple abscess of the liver in a young man of temperate habits. There was no previous dysentery or diarrhœa; it apparently began by congestion, terminating rather insidiously in suppuration, which probably commenced about twelve days after the first symptoms of congestion. As inflammation approached the surface the pain increased, and perihepatitis caused the adhesion of the right lobe to the parietes. There was reason to believe that the abscess was solitary, and not due to septic absorption from dysenteric ulceration. The prognosis was favourable, as latterly the abscess had caused little constitutional disturbance. The rapid contraction of the cavity showed that repair was vigorous. Recovery proceeded rapidly under the influence of change of climate and sea air.

No. 2.—Abscess of the Liver. Opened between the Ribs. Necrosis and Exfoliation of the Ribs. Recovery.

This is the case of a native of India, in whom an abscess was punctured between the ribs behind the axillary line, and exit given to a large quantity of pus. After some days there was a large sloughing opening, and necrosis of a considerable portion of one or two ribs. His general condition then improved, and the wound subsequently closed. There was a deep cicatrix

over the exfoliated ribs, but otherwise the wound had healed well, and the man was stout and in perfect health.

This case is exceptional; for where the ribs necrose and the opening becomes large from ulceration or sloughing, the patient generally succumbs to exhaustion, if not to some other complication. An abscess pointing between the ribs, especially towards the back, is very prone to cause necrosis; if punctured, the earlier the operation is done the better.

No. 3.—Liver Abscess. Opening through the Stomach. Recovery.

A young Englishman, aged about 22, of healthy, vigorous frame, rather irregular habits, after three years in Bengal complained of anorexia, hepatic fulness, general sense of failing health, disordered bowels, tongue loaded. He was anæmic, and the skin had a yellow tinge; pulse quickened. There had been neither rigors, chills, cough, nausea, nor vomiting. The liver was enlarged downwards, but there was no bulging, nor much tenderness on pressure. Saline aperients were ordered, and he was sent to sea for a few days.

Shortly after his return from on board ship, when romping one evening, a friend accidentally gave him a squeeze which compressed his body. This was followed immediately by symptoms of collapse, with coughing and vomiting a large quantity—a basinful—of purulent matter. A liver abscess had opened into the stomach and been evacuated. On recovering from the shock he soon began to regain strength, and before long was restored to health.

This case shows how insidiously the liver may suppurate without more marked symptoms than may be seen in cases of malarious poisoning, in which the general deterioration of health is expressed by occasional feverish attacks, attended by some hepatic congestion; the first evidence of formation of matter being its sudden evacuation in the manner described.

No. 4.—Liver Abscess. Opening through the Lung. Recovery.

In 1866 a young officer in camp at Agra, who had been a great deal exposed to heat and fatigue during hot days and cold nights, appeared to be suffering from liver abscess complicated with pneumonia of the right lung. Three weeks later, when he came to Calcutta, he was fearfully emaciated and hectic, with a very rapid pulse and diarrhœa. He was expectorating large quantities of pus, and appeared to be rapidly sinking. A few days later he was put on board a steamer in the hope that change of climate and sea air might benefit him. At Galle he was so low that he was landed, but he soon revived a little and went to Singapore. He rapidly regained strength even in the tepid climate of the Straits, and when he returned to Calcutta soon resumed his duties, and subsequently completely regained his health. He was alive and well years afterwards.

In this case there was probably only one abscess, though a very large one, which had formed insidiously and found its way into the right lung. The lung regained its normal condition, and did not suffer permanently from the suppuration in which it had been involved.

Such a termination could not generally be expected in so severe a case. It shows that even where a large abscess has opened into the lung, recovery is possible.

No. 5.—Liver Abscess. Opened below the Ribs. Recovery.

A young man, aged 23, neither intemperate nor irregular in habits, who had been in Calcutta for three years, had fever in March 1867. In April there were well-marked indications of the formation of pus, though not much pain, and neither jaundice nor diarrhœa.

The liver was enlarged downwards, and there was a prominence below the ribs into which a trocar and cannula was introduced on 19th April, and exit given to a large quantity of pus. The cavity was washed out with a weak carbolic solution, and the cannula left in, that more pus might be drawn off. He was much relieved, and soon improved. He was put on board steamer with the cannula still in the wound, but improved so much on the way that it was removed in about fourteen days. He recovered within three months, and subsequently returned to India. Years afterwards he was there in good health, and is now in good health.

No. 6.—Probable Liver Abscess. Recovery either by Absorption or possibly by Opening into the Bowel, though no Discharge was seen.

An officer, who first went to India in 1868 and had subsequently spent twenty-one months in England, began to feel ill in February 1877. He had pain on the left side of the back of the head; skin yellow; fever; spleen enlarged, and bulging of the left side; this was blistered, and soon subsided. On 27th February he embarked for England. In Bombay he got worse, and on the voyage had restless nights, intense weakness, loss of appetite, hands tremulous, shivering fits, and night sweats. Arrived in England on 2nd April.

The liver was enlarged downwards; the skin tinged with bile; face pinched and anxious; urine occasionally loaded with lithates; bowels constipated; great debility and nervous depression; slight tenderness on pressure over the lower margin of the liver; no pain nor enlargement of spleen; other organs apparently normal. Temperature rose in the afternoon to 102° or 103°. General aspect pointed to hepatic suppuration. Gradually strength returned, there was less depression, and an improvement took place in the condition of the urine and secretions. The temperature fell steadily, especially under the influence of five grain doses of quinine and hydrochlorate of ammonia three times daily,—a valuable remedy, for the use of which, in hepatitis, we are indebted to Dr. Stewart, B.M.S.

It is impossible to say whether suppuration had taken place or not. A small abscess may have formed, that either had become quiescent and was gradually drying up, or had discharged into the bowel, or might subsequently do so.

No. 7.—Liver Abscess with Sloughing of the Integument. Large Opening. Death from Exhaustion.

An officer, aged about 50, who had been twenty-four years in India, spare and of sallow complexion, in August 1882, when he had been home for about three weeks, presented himself with symptoms indicating an abscess of the liver, pointing below the ensiform cartilage.

From the history he gave, the formation of the abscess had been preceded by the usual symptoms of fever and sweating.

It was very painful on pressure, and the skin was already gangrenous;

about 3 inches' area of skin had sloughed from pressure. The slough was laid open as soon as he came under observation, and from 8 to 10 oz. of thick liver pus evacuated. It was kept open, and treated and dressed with carbolic acid. The patient was much wasted, and had occasional diarrhœa. Prognosis unfavourable on account of bowel complication, perhaps multiple abscess, and size of sloughing opening.

On 1st September his temperature was 99°; pulse, 100. Thick matter and pieces of the sloughing integument came away. The surface was of a dark reddish-brown, like cotton soaked in dark stained pus; tenderness and hardness round the edges rather less, more on the upper and right margin. Pus oozed on pressure; bowels better. Fear of extension of mischief in the liver, and septicæmia.

Died on 20th September, apparently from exhaustion. Signs of another abscess.

It appears that the patient, previous to coming under observation, had been advised by some one to allow the abscess to open of itself. Had an opening been made earlier, it is possible that the result might have been different.

No. 8.—Single Liver Abscess, forming with very little constitutional Indication of Suppuration; early Detection of Pus; Opening of deep Abscess; second Opening. Recovery.

An officer, aged 30, a healthy, vigorous young man of careful mode of life, went to India in November 1887, to Bangalore. He got a chill at Ootacamund in June 1889. Was laid up more or less for three months, but did not then leave India. Recovered, and kept well till 1891. At Poona got fever. Went to Australia for two months. Returned to India in May 1891. Caught cold after galloping; had fever.

About this time some hepatic mischief seems to have come on. He went to Bombay on leave, and sailed for England. Was fairly well when he left Bombay; had pain in the liver on the way home. Had no rigors, and apparently very slight fever on board ship. Reached England on the 28th of June 1892.

It appears that when in Egypt some years before he had a dysenteric attack, but had quite recovered long before he went to India. Up to the present never had any suspicion that his liver was affected. He was in India altogether about five years.

When he came under observation on 29th June he had pain, but no sweating and no fever. The symptoms continued much the same, and on the 30th July the liver was punctured in the ninth intercostal space, and 8 to 10 oz. of pus evacuated; pain diminished. On 2nd August a free incision was made in the same spot, 6 oz. of pus evacuated and a drainage tube inserted. On the 8th he was still doing well; the discharge was free but diminishing, and the tube was shortened. The temperature had not been above 98°·3. On the 15th he was not so well; tube extruded some days before by contraction of the cavity. Temperature rose to 100° the day before; more pain, slight sweating, loss of appetite.

On 16th August the abscess was reopened and a large grooved cannula was introduced. The aspirator drew out about 1 oz. of thick pus. The temperature fell, but rose again (to 103°) on the 19th, and there were sweats. On the 23rd he was better, and from that date continued to improve. There was very little discharge, and what there was was quite healthy.

The tube was gradually shortened; the tongue was clean; the bowels regular; he was quite convalescent by 1st September. He had quite recovered by the end of October, and was to return to India.

No. 9.—Liver Abscess recurring. Opened. Recovery.

An officer, aged 32, of sanguine temperament and moderate and active habits, who had been in India nearly nine years, had an attack of dysentery and congestion of the liver in September 1889. In October 1890 he had fever and dyspepsia, and a small swelling was observed in the epigastric region. On 12th October he was admitted into the General Hospital, Rangoon. The swelling increased in size, his skin was dusky, tongue thickly furred, pulse rapid and weak. He had fever; fluctuation of the swelling in the epigastric region was perceptible, and the skin over it was reddish. On 17th October the abscess was opened at its most prominent part in the median line, and about 8 oz. of pus were evacuated. The wound closed, and the patient was discharged on 10th November. He left for England, but the abscess reformed, and was opened again in Cairo on 17th December. Towards the end of the month it had nearly healed again, but on 29th December it was found that a fresh abscess had burst through the original opening. This was laid freely open, and an abscess as large as an orange was found lying behind the first one, from which a quantity of chocolate-coloured pus was withdrawn. The temperature at once fell, and did not again rise. Free drainage was maintained, the cavity washed out daily with weak carbolic lotion, and the tube gradually shortened as it contracted. All enlargement of the liver disappeared. Chloride of ammonium and nitro-muriatic acid were administered latterly. On 25th January the patient left the hospital well. Patient went on to England, recovered, and returned to India in August 1892.[1]

No. 10.—Liver Abscess complicated with Dysentery and Diarrhœa. Two Openings. Recovery.

An officer, aged 32, who had been eight years in India, had been sent to England in 1887 on sick leave for spleen and fever. In January 1890 he had fever, pain in the region of the liver, and vomiting, for about a week. In June he had diarrhœa, following a chill; this passed into dysentery, but returned, and continued till December of the same year. There was return of fever and pain in the side in October.

On 24th December 1890 he complained of pain in his right side, over the posterior hepatic region. He was sallow, much emaciated, in a flabby condition, tongue coated, appetite impaired, region of the liver prominent posteriorly, intercostal spaces of affected side masked, much flatulence, three or four watery and offensive stools daily, which, under chloride of ammonia and nitro-muriatic acid, became normal. Temperature, 102°, but soon decreased; fever occasionally, sweating of head when asleep. Pain became somewhat less, and swelling decreased appreciably.

On 22nd January 1891 he was transferred to the General Hospital, Madras. Symptoms much the same. Swelling most prominent about the axillary line, and in the tenth intercostal space. On 24th the liver was aspirated, and a free incision made about the right mammary line in the eighth intercostal

[1] *Records of the India Office.*

space, and about 40 oz. of pus evacuated. A long probe was introduced, and a counter opening made on its point in the posterior axillary line in the eleventh intercostal space. Two large drainage tubes were inserted. He was at first slightly collapsed, but afterwards progressed well. Granted leave for a year in February 1891. When he arrived in England the openings were healed, but he was suffering from dysenteric diarrhœa, which began in the Red Sea. This soon disappeared under treatment. In October 1891 he was recommended to apply for further leave. Returned to India in robust health in August 1892.[1]

No. 11.—Liver Abscess. Opened in the Ninth Intercostal Space. Recovery.

An officer, who had been about two years in Afghanistan, had fever in January 1890, which lasted for three months, and hepatic congestion in October of the same year. In March 1891 he left Quetta with his regiment for Poona, and suffered a good deal on the way from the hot sun of Sinde. On the voyage from Kurrachee to Bombay he had a cough, spit blood, and noticed a swelling in his side. On 7th April he entered the General European Hospital, Bombay; he had no fever, his tongue was furred, his bowels constipated, and his skin sallow. A swelling was found between the seventh and tenth ribs in the posterior axillary line, and the liver was moderately enlarged. The abscess was opened on the 8th April, and about 12 oz. of pus evacuated. He remained in hospital for three weeks, and embarked on 29th April, by which time the discharge had nearly ceased. It recurred owing to the fatigue of packing, etc.; but by the time Gibraltar was reached it had again ceased, and shortly after landing the wound healed entirely. He experienced no further trouble.[2]

No. 12.—Liver Abscess. Opened. Recovery.

An officer, aged 28, who had been in India for five years, had suffered from occasional attacks of fever, one of which was followed by a severe outbreak of boils. He went on leave to Naini Tal early in October 1891. He rapidly recovered, and the boils and fever disappeared. On 14th October he was chilled, and when he reached home felt severe pain in the region of the liver. Dysentery supervened; it yielded to treatment, but was followed by fever, the temperature frequently rising to 104°·4; there were occasional profuse perspirations. Enlargement of the left lobe of the liver was apparent, with a distinct prominence in the epigastric region. This was explored and opened on the 5th of November; about a pint of matter was evacuated and a tube introduced, which was removed on 3rd December.

The patient left for England in December 1891. In June 1892 he had a severe attack of fever, with pain over the region of the liver. At the end of August he was progressing well, had a good appetite, and weighed his full weight.[3]

No. 13.—Liver Abscess. Opened in the Eighth Intercostal Space. Recovery.

An officer, aged 34, who had served eleven years in India, was admitted into hospital at Bangalore suffering from malarial cachexia early in June 1892.

[1] *Records of the India Office.* [2] *Ibid.* [3] *Ibid.*

He had previously contracted fever and dysentery, followed by hepatitis, in Burma, while there in the cold weather of 1891. His liver was found to be much enlarged upwards; there was increased area of dulness, and pain in the right side. He had a troublesome cough and obstinate constipation, no rigors, no night sweats; the temperature twice went up to 101° while he was in hospital, but was generally normal. Leave to England was recommended; but when in Madras he was admitted to the General Hospital there on 8th July for pain and swelling over the region of the liver. A very tender spot was found in the eighth intercostal space; an exploratory puncture was made, and pus discovered. A free incision was then made and 4 oz. of pus evacuated; a drainage tube was introduced, and the wound dressed antiseptically. Recovery was uninterrupted, and he was discharged on 9th August 1892 to proceed to England.

The patient presented himself in London on 31st August 1892; the wound was quite healed, all symptoms of hepatic mischief had passed away, and he was rapidly regaining strength.[1]

In the following cases the notes of the surgical part of the treatment are due to Mr. Godlee, by whom it was carried out:—

No. 14.—Multiple Liver Abscess. Opened in two Places. Death.

A merchant, aged 36, had lived in Kimberley for eight years. In 1883 he had intermittent fever, which returned in May the next year, accompanied by pain in the liver. The patient was stout, flabby, and of a bad colour; foul tongue, weak pulse, and temperature rising to 102° or 103° in the afternoon. An abscess was obviously pointing at the epigastrium; it was opened on 7th May, and about half a pint of offensive yellow pus was evacuated. There was a good deal of bleeding. For the day after the operation the temperature was normal, but it then began to rise, and the condition of the pulse, bowels, and tongue pointed to the presence of another abscess. On 14th May the liver was punctured just below the ribs, a little outside the first opening; but as pus was not found, the needle was passed through the seventh or eighth space at the lower part of the axillary, and 3 or 4 oz. withdrawn from a considerable depth. The wound was then enlarged, and, using the cannula as a guide, dressing forceps and the finger were passed into the abscess. This caused much hæmorrhage, and the wound had to be plugged; but two days later the plug was removed and a large tube introduced, which seemed to drain the abscess satisfactorily for a time. There was no improvement in the general state, and ten days later a fine trocar was passed into the wound, and a large quantity of pus was evacuated. On 3rd June the patient died. There was no post-mortem, but probably there were many abscesses.

No. 15.—Sub-diaphragmatic Liver Abscess. Opened. Recovery.

A young man, aged 30, a resident of Calcutta, in 1888 showed symptoms of liver abscess, the liver being extended far down, with an increased area of dulness in front and in the axilla, with tenderness in the latter. A trocar, introduced through one of the lower intercostal spaces, detected pus, and when an opening was made, 60 oz. of matter were evacuated from

[1] *Records of the India Office.*

between the upper surface of the liver and the diaphragm. This was a sub-diaphragmatic abscess.

In this case no opening into the liver could be found; a large drainage tube was inserted. The liver at once resumed its normal position, and the abscess took a typical aseptic course.

No. 16.—Large Liver Abscess. Opened in the Epigastric Region. Recovery.

A lady, aged 30, went to India in January 1888. She suffered from boils, dysentery, and hepatic congestion. The liver became very much enlarged and tender, and the temperature at times reached 103°. In August 1889 she was much emaciated and slightly jaundiced. The liver was large and tender; there was prominence at the epigastrium, and a sense of nausea on pressure. An opening was made in the epigastric region, and a large quantity of pus evacuated. In a few days the liver resumed normal proportions, and in about two months the abscess healed. The discharge was trifling after the first week or ten days.

This case was under the care of Dr. Mair, seen in consultation by Sir Joseph Fayrer and Mr. Godlee.

The following cases from Madras were furnished by Dr. Keess:—

No. 17.—Multiple Abscess. Opened in the Eighth Intercostal Space. Death.

A young man, aged 31, of temperate habits, felt in August 1882 a severe stabbing pain below the right nipple, and on 1st September was admitted into the General Hospital, Madras. The liver was extended below the costal arch, in the mammary line 2 inches, in the axillary line 1 inch. There was slight bulging, much pain on pressure over the liver, aching pain in the right shoulder, and occasionally dry cough. He was much depressed, bowels loose, temperature 99°. The temperature increased, respiration became thoracic, bulging between the ant. axillary and mammary lines became obvious, fluctuation was detected about the eighth intercostal space. On the 19th he was aspirated at this spot, and 22 oz. of pus were drawn off. The temperature immediately fell, but the improvement was very temporary. On the 24th he was aspirated again, and 14 oz. of dark red pus drawn off. For a few days he had symptoms of peritonitis: but though these passed away his condition did not improve, and, on the 27th, a free incision was made over the seat of the punctures, and 30 oz. of pus evacuated. A drainage tube was inserted, and the abscess continued discharging from 10 to 16 oz. daily; but soon the pus became extremely offensive, the wound sloughed and opened out, diarrhœa set in, and he died exhausted on 24th November.

Post-mortem examination showed that the right half of right lobe of liver was adherent to parietes; the floor of abscess was very extensive, but not deeply seated. Deep in the substance of the right lobe there was a second abscess, containing about a pint of pus; a third, very small, on the upper surface, and a fourth, about the size of a goose's egg, in the under surface of the left lobe.

No. 18.—Threatened Suppuration of the Liver. No Puncture. Recovery.

An officer, aged 37, resident nine years in India, subject to malarial fever, was admitted into the General Hospital, Madras, with acute hepatitis, in September 1882. The temperature was 103°·2; the tongue was furred, bowels relaxed; increased area of hepatic dulness, and great tenderness over the right hypochondriac area; the spleen also was enlarged and tender. The fever continued, temperature rising in the evenings to 102°·6; these exacerbations were accompanied by stabbing pain over the region of the liver. Chloride of ammonium and weak nitro-muriatic acid were substituted for the aconite previously given. These symptoms continued for some days, and it was thought an abscess had formed. On 9th October the patient began to improve, progress continued, liver and spleen returned to their normal size, and by the middle of November recovery was complete.

No. 19.—Liver Abscess. Opened. Large quantity of Pus evacuated. Recovery.

A Hindu, aged 30, was admitted into the General Hospital, Madras, on 6th January 1883, for liver abscess. He had previously suffered much from malarial fever and diarrhœa, and had a cachectic appearance. The liver was tender and enlarged, and there was bulging in the right side and pain, and occasionally pain in the right shoulder; temperature, 101°; pulse, 120; slight cough. On 9th January the abscess was aspirated, and 27 oz. of creamy pus evacuated. Relief was experienced for some days, but on the 15th pain returned; fluctuation reappeared, and on 21st and 22nd there were rigors. On the 24th he was reaspirated in the same spot, and 34 oz. of very thick pus evacuated. But the temperature remained high, and on 1st February a large trocar and cannula were introduced, and 36 oz. of pus evacuated. A drainage tube was inserted, and the abscess continued discharging profusely; on the 21st the discharge became offensive, but local application relieved this; on the 28th the discharge was scanty, the wound gradually closed, and the patient was discharged cured on 21st March.

No. 20.—Liver Abscess. Opened. Subsequently opened into the Right Pleura. Death.

A Eurasian, aged 39, of intemperate habits, was admitted into the General Hospital, Madras, on 21st February 1883, with fever, cough, pain over the region of the liver, tongue loaded and tremulous, conjunctivæ yellow, temperature 100°; increased area of dulness, special tenderness in the region of the left lobe; slight bulging in the epigastrium. On 1st March this had increased, and there were fine crepitations at base of right lung. On 13th March fluctuation was detected; the swelling was aspirated, and 8 oz. of pinkish matter drawn off. A free incision was made on the 16th; 16 oz. of matter were evacuated, and a drainage tube inserted. On the 19th there was pneumonia of the right base, and temperature rose to 104°; on the 22nd both knee joints became swollen and tender; on the 23rd severe pain in right side came on at 3 P.M., and patient died at 6 P.M. The right chest had suddenly become absolutely dull right up into the axilla.

There was no post-mortem; but from the continuance of high temperature

after the incision, it was surmised that there was a secondary abscess of the right lobe, and that death was caused by its opening into the right pleural cavity. The wound had remained quite healthy all the time.

No. 21.—Liver Abscess soon after Child-birth. Puncture. Death.

A lady, aged 25, was reported as suffering from fever on 31st March 1883. Two days previously she had been delivered prematurely of her fifth child. For the first few days she had continued fever, temperature in the morning 102°, in the evening 103°. On 2nd April there was severe pain in the right hypochondriac region, but no hepatic enlargement; the pain disappeared under the use of linseed-meal poultices. On 5th April she had a severe attack of bilious diarrhœa, fever continuing; the liver was slightly enlarged, but not tender. Bowels had been regular, and tongue clean all the time; temperature now began to fall, but circulation was very excited. Hepatic symptoms disappeared, except that liver continued slightly enlarged. On 27th May the liver was more enlarged, and there was bulging below the false ribs. The superficial veins over the hypochondria and mammary regions were enlarged, and there was slight œdema of the feet; the liver was tender on pressure, with a firm elastic feeling and an indistinct sensation of fluctuation; temperature rose again, and she was losing ground. Vomiting seemed to point to an abscess being about to discharge into the stomach. It was decided to puncture on 1st June; she was then extremely weak, temperature 100°·4, voice feeble, respiration thoracic and weak; had passed no urine since 2 P.M. the day before. When the liver was aspirated, about 28 oz. of thin reddish-brown pus were evacuated. She rallied a little after the operation, but towards evening she failed, and died at 9·30 P.M.

The following case from the Royal Victoria Hospital, Netley, is contributed by Professor Cayley:—

No. 22.—Deep-seated Abscess of the Liver, following Chronic Hepatic Congestion and slight Dysentery. Aspiration and Incision in the Seventh Intercostal Space. Recovery.

A private, of temperate habits, who had served four and half years, chiefly in Burma and India, had an attack of ague in June 1891, after which he never felt quite well. In August he was admitted into hospital for hepatic congestion; this was followed by slight dysentery, from which he completely recovered. As he did not regain his health, he was sent home, and reached Netley on 1st May 1892, having had slight return of hepatic congestion on the way home.

He was weak and emaciated; sallow; tongue pale and flabby; bowels constipated; dull, aching pain in the hepatic region; temperature rising in the evening to 100° or 101°; occasional profuse night sweats; slight cough. There was slight enlargement of the liver, increased area of dulness, tenderness on percussion.

After three days, as the symptoms continued, the liver was aspirated in the seventh intercostal space, and 5 oz. of thick chocolate-coloured pus withdrawn. The patient was greatly relieved, and temperature fell to 99°. Two days later, as the symptoms recurred, he was reaspirated in the same place, and 3 oz. of pus withdrawn; the enlargement greatly subsided, and

temperature fell to normal. The symptoms returned, however; and as the third aspiration, a fortnight later, withdrew only a few ounces of pus, a free incision was made in the seventh intercostal space, about 5 oz. of thick flaky pus withdrawn, and a drainage tube inserted. From this time the man steadily improved, the discharge gradually decreased, and ceased in six weeks; and soon afterwards he had completely regained strength and health.

The following cases from the Medical College Hospital, Calcutta, are contributed by Professor McConnell, Physician to the Medical College Hospital, Calcutta:—

No. 23.—Multiple Abscess of the Liver. Death from Dysentery and Exhaustion.

A Hindu, aged 40, was admitted into the Medical College Hospital, Calcutta, on the 20th April 1891. Had been addicted to drink for many years. Was very emaciated and weak. Had suffered from pain in the hepatic region for six months previously, fever for two months, and a fortnight previously bulging had appeared and had gradually increased. Bowels loose and contained slime. Area of liver dulness extended. Whole of right hypochondrium and epigastrium occupied by a soft elastic swelling, with distinct fluctuation; temperature, 99°·4 F. On 21st April (temperature sub-normal) an incision was made over the most prominent part of the swelling, in the right hypochondrium, 2 inches below the costal arch, and 12 oz. of thick pus evacuated. A large drainage tube was introduced and antiseptic dressings applied. These were renewed in the evening; the temperature was then 98°.

The condition of the bowels did not improve; six to eight stools were passed per diem, loose and thin, containing a little feculent matter and some shreddy mucus.

Temperature, which had been normal since the operation, began to rise on 29th April, and was 100°·4 in the evening and 99° the next evening. Stools less frequent and free from mucus. Complained of pain in the abdomen generally, and particularly over the liver. Not gaining strength; pulse small and frequent; tongue inclined to be dry.

On 1st May the stools were more frequent, and contained mucus; pain continuing; temperature, 98° in the morning, 99·4° in the evening.

On 2nd May the temperature was subnormal, stools loose; patient very low, complained of tightness across the chest and a sense of suffocation.

On 3rd May there was scarcely any pulse at the wrist; extremities cold; breathing hurried, shallow, and forced; temperature 97°·6. Patient gradually sank and died.

In the post-mortem examination, only the liver and large intestine were allowed to be examined. A large abscess cavity was found in the right lobe of the liver, involving the anterior two-thirds of the lobe. It had evidently contracted, and was about 3 inches in diameter. About thirty small secondary abscesses, varying in size from a pea to a pigeon's egg, were found scattered through the rest of this lobe and also in the left lobe. The majority seemed to be of very recent origin. There was dysenteric ulceration of the descending colon, sigmoid flexure, and rectum.

No. 24.—Liver Abscess in right Lobe. Opened in the Tenth Intercostal Space. 150 oz. of Pus evacuated. Recovery.

A Hindu, aged 40, was admitted into the Medical College Hospital, Calcutta, on 30th June 1891. A month previously he had been attacked with fever which lasted a fortnight. It recurred a week before his admission into hospital, and was then accompanied by pain and feeling of distension in the region of the liver. The area of dulness was extended; there was tenderness on pressure over the whole of the area, and a feeling of resistance to the finger when pressed into the intercostal spaces, but neither localised bulging nor fluctuation. Temperature, 101°. He was treated with quinine in the mornings when the temperature was normal or subnormal (rising in the evenings to 101° or 102°), and with aloes pill for constipation and coated tongue.

On 5th July a puncture was made in the tenth intercostal space; pus was found, a large-sized trocar and cannula passed in, and 150 oz. of pus evacuated. The patient was much relieved, and the temperature fell to 98°.

The discharge was profuse for about a week, but it gradually lessened, and after a week was found to be bloody in character. The cavity of the abscess began slowly to contract, and on 2nd August a drainage tube of smaller calibre was introduced, which was shortened every three or four days. The patient gradually recovered, and was discharged on 12th August.

The chief points of interest in this case are the enormous size of the abscess, coupled with the absence of distinct signs.

No. 25.—Multiple Abscess of the Liver. Death from Dysentery and Exhaustion.

A Hindu, aged 30, was admitted into the Medical College Hospital on 12th September 1891. Had suffered from fever, pain in the liver, and dysentery for the previous three and a half months, passing six or seven stools per diem, containing mucus and blood.

Was emaciated and weak. Temperature, 100°. Liver enlarged, and tender on pressure. The lower right ribs, from the seventh downwards, unduly protruded, and intercostal spaces widened. No actual fluctuation, but elastic feeling, particularly in the eighth intercostal space. Liver treated with outward application.

On 14th September an exploratory puncture was made in the eighth intercostal space, and pus being found, an incision was made, a large-sized trocar and cannula introduced, and 15 oz. of thick pus evacuated. A drainage tube was introduced and antiseptic dressings applied. Evening temperature 99°·4.

The pain over the liver was much relieved, but the condition of the bowels remained much the same, six or seven mucoid stools in twenty-four hours; the pulse improved, and the temperature the next day was 98° in the morning and 99°·8 in the evening. The dysentery continued; the discharge from the abscess went on freely; the temperature for the next three days was 97° or 98° in the morning, rising to 100° or 101° in the evening. The discharge became blood tinged, the patient lost ground, and visibly emaciated. On 20th September the temperature was subnormal all day. On 21st the pulse became very small, the extremities cold, heart's action weak, stools passed involuntarily. Died at midnight.

Post-mortem examination twenty-two hours after death. Body extremely

emaciated. Slight rigor mortis in the lower extremities. An incision with sloughy, unhealthy-looking margins, about 1 inch long and 1½ inches wide in the eighth intercostal space in the mid-axillary line.

Brain and membranes anæmic, but otherwise healthy.

Lungs somewhat collapsed and anæmic except at the bases, which were œdematous and slightly congested. Recent pleuritic adhesion between the base of the right lung and diaphragm.

Heart.—Small decolourised clots, with fluid blood in both right and left cavities; the latter contracted, the former flabby.

Abdomen.—About 50 oz. of dark greenish coloured serum in the peritoneal cavity.

Liver weighed 3 lbs. 12 oz. Was everywhere strongly adherent to the diaphragm and abdominal parietes. At the posterior and back part of the right lobe was an abscess cavity the size of one's fist, containing offensive, thick, reddish pus. It communicated with the external opening made between the ribs. Closely surrounding this cavity were a series of small abscesses in the substance of the right lobe, some separate, others confluent, also filled with reddish-yellow pus. Many appeared to have recently ruptured into the principal abscess cavity. Scattered irregularly throughout the rest of this lobe were small abscesses (in size from that of a pea to that of a walnut), surrounded by bright red zones of congestion. The left lobe was free from suppuration, but the hepatic tissue was very soft and friable and fatty looking. The gall-bladder was full of viscid, orange-yellow coloured bile.

Spleen enlarged (weight, 12 oz.). Capsule much thickened in parts, almost cartilaginous in appearance and consistency. Substance soft and pale.

Kidneys healthy, somewhat pale.

Intestines.—The whole of the colon enormously thickened, and covered with large, sloughy, or bare ulcers, which in many places involved the whole thickness of the mucous membrane.

No. 26.—Abscess of the Liver. Opened in the Ninth Intercostal Space, in the Mid-axillary Line. Recovery.

A Hindu, aged 27, was admitted into the Medical College Hospital, Calcutta, on 6th April 1892. About a month previously he began to suffer from fever and colicky pain in the epigastrium, which after twelve days shifted to the right side and changed in character, becoming dull and aching. The area of hepatic dulness was enlarged, and tender on percussion. The spleen was not enlarged; pulse small and soft; bowels constipated for the previous few days; temperature normal in the morning, reaching about 101° in the evening. Urine free from albumen, but showed a trace of biliary colouring matter; conjunctivæ yellow.

The patient was relieved by free evacuation of the bowels the next day. But as the hepatic tenderness and the fever continued, the former was treated externally with chloroform and belladonna, the latter with ammon. chloride and a saline diaphoretic mixture. The symptoms, however, remaining the same. On the 9th April puncture was made by means of a long-needled hypodermic syringe in the ninth intercostal space, and pus was discovered. A small bistoury was then passed in, and 2 oz. or 3 oz. of reddish curdy pus evacuated. A drainage tube was inserted.

The same evening the temperature was 99°·6, the next morning was

normal, and never rose again. The abscess cavity gradually contracted, the drainage tube was shortened, the conjunctivæ regained their normal colour, and the patient was discharged on 13th May 1892.

This is a good illustration of the value of puncture in doubtful cases.

No. 27.—Abscess of the Liver. Incision in the Hypochondrium. Recovery.

A Hindu, aged 32, was admitted into the Medical College Hospital, Calcutta, in May 1892; he had been suffering from fever for the previous three months, associated recently with pain in the right infra-axillary region, and swelling in the same part.

No previous history of dysentery or alcoholism. Bowels constipated; liver enlarged and tender; seventh to tenth ribs unduly prominent, and ninth and tenth intercostal spaces widened; indistinct fluctuation could be felt in the ninth intercostal space and below the costal arch. Pulse was small, but regular; tongue coated, but moist.

On the morning of admission the temperature was 99°·4, rising to 102° in the evening. The next morning it was 99°. Exploration was made with a long hypodermic syringe in the right hypochondrium and in the ninth intercostal space, and pus was found in both directions.

An incision was then made in the hypochondrium, in the most prominent part of the swelling, and 16 oz. of thick reddish pus evacuated. A large sized drainage tube was introduced, and antiseptic dressing applied. Temperature fell to normal in the evening.

The next day the morning temperature was 96°·6, the evening 98°·2; the discharge was very copious, and continued so for the first week. It then began to diminish, the cavity gradually healed, and the patient gained weight and flesh. The drainage tube was removed on 1st August, and by the 5th the wound had perfectly healed, and the patient was discharged.

No. 28.—Case of Abscess of the Liver. Opened in the Eighth Intercostal Space. Recovery.

A Hindu, aged 37, was admitted into the Medical College Hospital, Calcutta, on 4th July 1892. Had lived in Eastern Bengal for the previous five years, and enjoyed good health till December 1891, since when he had suffered from malarial fever, with pain and heaviness over the region of the liver. For the ten days previous to admission the pain was so severe as to prevent decubitus on either side, and the right hypochondrium was tender to the touch. Temperature normal.

No previous history of dysentery or alcoholism; patient somewhat reduced, but all organs, except the liver, healthy. Area of liver dulness extended, and tender to the touch; bulging of the lower ribs, and indistinct fluctuation in the eighth intercostal space in the mid-axillary line. There was slight pointing in the eighth intercostal space; exploration revealed the presence of pus; an incision was made, and 30 oz. evacuated. A large sized drainage tube was introduced; the discharge for the first week was copious; it then began to diminish, and the cavity gradually healed. The temperature never rose above 98°, and in the mornings was often 97°. The patient improved, however, in health and strength from the date of the operation. On 29th July a sinus of only 2 inches remained, and on 6th August this had healed and the man was convalescent.

The rapidity of healing and the complete absence of all fever were the chief characteristics of this case.

Remarks by Dr. McConnell.

Cases 27 and 28 had no previous history of dysentery or alcoholism, showing that single or solitary tropical abscess may occur without preceding bowel troubles or alcoholic irritation. Both these men were in fairly good health, *i.e.* not much reduced or emaciated, and with their other organs in good working condition. Their uninterrupted recovery after operation must be largely attributed to this. Unfortunately, it is exceptional to receive patients, and especially native patients, in this condition in Indian hospital practice. Generally a native will try a hundred remedies, and procrastinate from week to week until he becomes so weak and exhausted that, when he does seek admission, his state is most unfavourable for operative interference. He has no vitality, the abscess has assumed enormous proportions, or secondary suppurative foci have already formed in other parts of the liver, and such cases die in the proportion of quite 50 per cent., probably more. But where admission is sought fairly early in the disease, and the general vitality is not greatly impaired, we have, I should say, a success of quite 75 per cent. by incision and free drainage, with antiseptic precautions. And the same holds good as regards private patients, consultation here being early, and the chances of recovery thereby greatly enhanced.

We use no special probes, cannulæ, or apparatus of any kind, nor find the need of them.

An abscess of the liver is treated like an abscess of any other part of the body, on general surgical principles, the most important of which are free drainage and antisepticism.

The following cases, from the Medical College Hospital, Calcutta, are contributed by Dr. Birch, Principal and Senior Physician :—

No. 29.—Liver Abscess, followed by Diarrhœa. Recovery.

A European, aged 48, was admitted into the Medical College Hospital, Calcutta, on 22nd April 1890; he had had pain and swelling in the right hypochondrium for about eight days. When the liver was punctured, 56 oz. of pus were evacuated, and the discharge continued very freely for a long time. Diarrhœa supervened, and continued for a long time. The temperature was generally normal, once subnormal, and once up to 100°. This patient was discharged cured on the 4th July, after gaining some strength and flesh.

No. 30.—Liver Abscess. Opened in the Mammary Line below the Arch, and again in the Tenth Intercostal Space. Death.

A Mohammedan, aged 60, was admitted into the Medical College Hospital, Calcutta, on 15th September 1890, very prostrate and emaciated. The liver was much enlarged, and there was a prominent swelling below the costal arch, and the tenth intercostal space bulged. The first opening was made on the 16th in the mammary line below the costal arch, and 20 oz. of pus evacuated. On the 17th an opening was made in the tenth intercostal

space, and 6 oz. of pus evacuated. The discharge from both openings was healthy; the temperature rose every evening. The hepatic enlargement did not subside, the wound in the tenth intercostal space began to slough, purging came on, and death ensued on the 2nd October.

No. 31.—Multiple Abscess of the Liver. Opened towards Median Line in front, ¾ inch anterior to Lower Border of Eighth Rib. Discharged also into Abscess Cavity in the Lung. Death.

A Hindu, aged 26, a hard drinker, was admitted into the Medical College Hospital, Calcutta, on 3rd November 1890. There had been pain below the right nipple for four months previously. Both lobes were enlarged below the arch, and there was a diffused painful swelling occupying the middle of the abdomen. The patient was very weak and emaciated, and had diarrhœa. The abscess was opened anterior to the lower border of the eighth rib on 4th November, and 32 oz. of pus evacuated. The discharge continued free; the temperature often went up to 100° or 101°, and frequently became subnormal. Diarrhœa was constant, there were profuse night sweats, a cough developed, the abdomen became tympanic, and the patient gradually sank, and died on the 3rd of December.

A post-mortem examination was made two hours and three-quarters after death.

There was great emaciation, and general rigor mortis.

The *abdominal walls* were very thin, and the muscular substance pale.

The *heart* was small.

The *lungs* were pale. The left was free from adhesions, and collapsed on opening the chest. The right lung was large, and adhered to the diaphragm and ribs. On breaking down the adhesions at the base, an abscess cavity was opened, which communicated through a ragged rent in the diaphragm with another abscess cavity on the upper surface of the right lobe of the liver. A thin, incomplete layer of granulations lined the walls of the lung abscess, and the surrounding lung tissue was consolidated and non-crepitant.

Diaphragm.—On its upper surface and to the right was a circular opening, 2½ inches in diameter.

Abdominal cavity.—The opening through the parietes, in which the drainage tube was inserted, was found to penetrate the liver substance. The tube reached the cavity.

Liver.—The lower border was within 2 inches of the umbilicus. The upper surface of the right lobe was adherent to the diaphragm, and to the parietes over the entire hypochondrium. The whole of the upper and posterior portion of the right lobe constituted an abscess cavity of 2 pints capacity, and contained a thick, white, curdy pus; the walls were rough, firm, and lined with a layer of granulations. In the lower portion of the right lobe was another abscess cavity with a 3 oz. capacity; the roof of this formed part of the floor of the other, and this partition between the two cavities was about ⅛ inch in thickness, and consisted of a layer of granulation tissue above and below, with a small amount of liver substance between. Another small cavity, containing about 1 oz. of thick pus, occupied the upper and anterior portion of the liver, immediately above the gall-bladder. The rest of the liver was in a nutmeg condition.

Kidneys and intestines pale; gave no reaction with iodine.

Spleen enlarged.

No. 32.—Abscess of the Liver discharging through the Lungs. Recovery.

A Mohammedan, aged 30, was admitted into the Medical College Hospital, Calcutta, on 17th November 1890, with fever and hepatic pain. Liver dulness began from the nipple; there was much tenderness on pressure, and there was a distinct fulness of the right side, and pain in the right shoulder. He had a troublesome cough. He coughed up large quantities of hepatic pus of a chocolate colour. The fever and expectoration diminished; he did well, and was discharged on 18th December.

No. 33.—Liver Abscess. Recovery.

A Hindu, aged 25, a total abstainer, was admitted to the Medical College Hospital, Calcutta, on 29th April 1891. An opening was made the same day, and 5 oz. of pus evacuated. He had diarrhœa at first, but there was no fever. The tube was removed on the 11th May, and he left the hospital on 8th June, a small sinus still remaining.

No. 34.—Liver Abscess bursting into the Lung. Recovery.

A Hindu, aged 40, was admitted into the Medical College Hospital, Calcutta, on 1st May 1891. He had been expectorating blood-stained pus for nine days previously. The liver was enormously enlarged, and there was slight dulness below the right clavicle and right interscapular regions. Evening temperature was usually 101° or 102°. Expectoration ceased on the 4th, but was renewed the next day. Breath became fœtid, and discharge lost much of its red colour. Left hospital at his own request on 8th June, as he considered himself well.

No. 35.—Liver Abscess bursting into the Bowel. Opened also from the Exterior. Probable Death.

A Hindu, aged 60, was admitted into the Medical College Hospital, Calcutta, on 25th May 1891. Had suffered from fever for eighteen days previously. Was very weak. There was a swelling about the size of a Bael fruit in the epigastrium. Upper margin of liver reached the fifth rib. Suffered much from hiccough; tongue dry, red, and cracked. Stools contained clots of blood and pus. Exploration was made into the swelling, and a few drops of pus evacuated; the end of the cannula was discoloured by sulphuretted hydrogen. An incision was made, but nothing more was obtained from the wound. Stools continued of the same character; he became weak and covered with perspiration, and was removed by his friends on the 27th.

No. 36.—Liver Abscess. Opened about the Inner Third of the Costal Arch. Recovery.

A Hindu, aged 26, was admitted into the Medical College Hospital, Calcutta, on 29th May 1891. He had had dysentery six months previously, and had been exposed to rain just before the attack. There was increased area of liver dulness. The abscess was pointing at the sixth intercostal space in the axillary line, but the opening was made about the inner third

of the costal arch, and 30 oz. of pus evacuated. The tube used was 8 inches long. The temperature rose twice after the operation to 100°. The patient gradually recovered, and was discharged on 23rd July 1891.

No. 37.—Liver Abscess. Opened a little below the Costal Arch in the Mammary Line. Recovery.

A Hindu, aged 22, who had been a hard drinker for five years, was admitted into the Medical College Hospital, Calcutta, on 1st June 1891. There was an increased area of dulness. Lower part of right chest bulged a little, and eighth, ninth, and tenth intercostal spaces were obliterated. An exploration was made, and pus found. The abscess was opened a little below the costal arch and in the mammary line, and 12 oz. of thick pus evacuated. The recovery was uninterrupted. On third day after operation temperature rose to 100°, but otherwise there was no fever. Marked improvement in general health. Discharged recovered on 16th July.

No. 38.—Liver Abscess. Opened to the Right of the Linea Alba. Recovery.

A Mohammedan, aged 30, was admitted into the Medical College Hospital, Calcutta, on 13th June 1891. He had suffered for one month previously from a saucer-shaped swelling about 3 inches in diameter below the ensiform cartilage. The abscess was in the left lobe, and there was bulging on either side of the median line. Incision was made to the right of the linea alba, and 6 oz. of pus evacuated. The cavity began to contract on the fourth day; he was discharged on 13th July.

No. 39.—Liver Abscess. Recovery.

A Mohammedan, aged 60, an occasional drinker, was admitted into the Medical College Hospital, Calcutta, on 27th June 1891. There was a swelling about 2 inches in diameter in the epigastric region, to the right of the median line. There was increased area of dulness, but not much pain. Temperature 101° on admission. The abscess was opened on the 30th June, and 2 oz. of pus evacuated. It was at first thick and greenish, and did not present hepatic characters till two days after the operation. He was discharged well on the 25th July 1891.

No. 40.—Liver Abscess. Opened 1½ inches below the Costal Arch, and 2 inches to right of Median Line. Recovery.

A Hindu, aged 59, a total abstainer, was admitted into the Medical College Hospital, Calcutta, on 11th November 1891. Had had fever for six months. There was increased area of dulness. The right lobe did not seem enlarged below the arch, but there was slight pain on pressure. There was swelling or fulness of the epigastrium to about 1 inch above the umbilicus, and extending to 1 inch to the left of the median line. An incision was made 1½ inches below the arch, and 2 inches to the right of the median line, and 25 oz. of pus evacuated. The temperature rose only once to 100°. Was discharged on 10th December.

No. 41.—Liver Abscess. Opened in the Ninth Intercostal Space, and through the Diaphragm. Recovery.

A Hindu, aged 42, a total abstainer, was admitted to the Medical College Hospital, Calcutta, on 21st November 1891. He had had dysentery, with fever, six months previously, and bleeding piles for a year, which had ceased five months previously. There was a globular tumour over the posterior hepatic region, which distended the lower ribs and dilated the intercostal spaces, and which had appeared two weeks before admission. The aspirator revealed pus, and an incision was made through the ninth intercostal space and the fibres of the diaphragm, when a large quantity of pus was evacuated. Temperature was subnormal for about four days after the operation, and perspiration profuse. However, the patient was discharged cured on 20th December.

No. 42.—Liver Abscess. Opened about 2½ inches below the Costal Arch, near the Median Line. Subsequent Death from Smallpox.

A Hindu, aged 30, was admitted into the Medical College Hospital, Calcutta, on 22nd January 1892. Had had fever and pain in the right hypochondriac region for six months. There was a large globular swelling on the right side of the abdomen, 8 × 5 inches, which was resilient and slightly tender on pressure. Increased area of dulness. Exploration revealed the existence of pus, and an incision was made 2½ inches below the arch, near the median line, and a large quantity of pus evacuated. The discharge continued profuse for some days, and the temperature went up to 100°. On the twenty-ninth evening, profuse perspiration was followed by general convulsions, succeeded by delirium. There was a second attack, but milder, of the same kind at night. After this the fever diminished, the discharge lessened, and the patient was doing well, when he got an attack of small-pox, of which he died.

No. 43.—Liver Abscess, complicated with Dysentery. Opening into the Peritoneal Cavity. Death.

A Mohammedan, aged 30, a total abstainer, was admitted into the Medical College Hospital, Calcutta, on 29th January 1892. He had had dysentery and fever a month previously. He was much emaciated, and could not lie down. The area of dulness extended from the fifth rib to 2 inches below the costal arch in the mammary line, and there was a prominent swelling below the right arch. The bowels were costive, and there were marked signs of peritonitis. The liver was explored twice, but no pus detected. The patient gradually sank, and died on the 4th February.

Post-mortem.—Abdomen.—The surface of the viscera was covered with a thick layer of lymph. The peritoneal cavity contained a considerable quantity of fluid. Parietal and visceral peritoneal layers adherent. The peritoneal surface corresponding to the puncture showed no abnormal signs differing from the rest of the peritoneum. The liver did not project below the costal margin.

The *intestines* were adherent, soft, and covered with lymph. No ulceration of the small intestines. In the large intestines there were several irregularly shaped ulcers, with thickened edges and sloughing bases, and there were a few sloughing ulcers in the cæcum.

Liver and stomach removed together weighed 4 lbs. 12 oz. At about the

middle of the stomach there was an adhesion with the wider surface of the left lobe of the liver. At this point of adhesion there was an opening the size of a crow quill, from which pus issued into the peritoneal cavity. That this opening had nothing to do with the puncture was evident, because it was covered by the lower margin of the left lobe of the liver, which was imperforate. A probe passed through the opening entered the left lobe of the liver, on opening which an abscess cavity as large as an orange was found lying close to its under surface, and opening as described into the peritoneal cavity. From the cavity of the stomach it was separated only by the thickness of the stomach wall. The right lobe was merely congested.

Lungs congested. All other organs healthy.

No 44.—Liver Abscess complicated with Pleurisy. Rib resected. Pleural Cavity opened. Diaphragm incised. Drainage Tube inserted through Pleural Cavity and Diaphragm. Recovery.

A Hindu, aged 30, a hard drinker, was admitted into the Medical College Hospital, Calcutta, on 27th February 1892. He had had pain in the right mammary and infra-axillary region for two months. Dulness commenced posteriorly about 1 inch below the angle of the scapula; over dull area respiration was feeble, and in front commenced at the fourth rib, and extended to 2 inches below the costal arch. Exploration was made below the arch with no result, and the next day at the sixth interspace in the axillary line; about 16 oz. of pus were evacuated, it was thought, from the pleural cavity. Fever, pain, cough, and diarrhœa continued. On 5th March 1¾ inches of the seventh rib in the post-axillary line was excised. About a pint of slightly greenish, serous fluid was evacuated from the pleural sac. The diaphragm at once filled up the wound; it was explored, a free incision made, and about 30 oz. of pus evacuated. The discharge was free. Evening fever continued for some days, but it gradually abated, the diarrhœa ceased, and the patient was discharged recovered on 4th April 1892.

No. 45.—Liver Abscess. Opened externally 1 inch below the Arch in the Mammary Line. Opened internally into a large Bile Duct. Death.

A Hindu, aged 26, a hard drinker, was admitted into the Medical College Hospital, Calcutta, on 26th March 1892. He had suffered for two months previously from fever and pain. Extended area of dulness; fulness and tenderness below the right costal arch. Pain was very great, bowels constipated. Opening was made 1 inch below the arch in the mammary line. Discharge continued free the whole time; diarrhœa set in, motions containing clots of blood. Patient rapidly emaciated, and died on 12th May.

Post-mortem.—Body extremely emaciated. Liver found adherent to abdominal walls round the wound. The whole of the right lobe was found to be evacuated, only a shell of liver-substance remaining, and this gave way as the liver was separated from the surrounding structures. The abscess cavity communicated with a large bile duct.

No. 46. Liver Abscess. Opened about 1½ inches below the Arch and a little to the right of the Mammary Line. Recovery.

A Hindu, aged 38, a hard drinker, was admitted to the Medical College Hospital, Calcutta, on 21st April 1892. He had noticed the enlarged liver

for a month previously. There was a globular bulging 4 × 6 inches, tender on pressure, below the costal arch. The bowels were constipated. Exploration detected pus; an incision was made 1½ inches below the arch, and a little to the right of the mammary line, and 10 oz. of pus evacuated. The patient made a rapid recovery, and was discharged on 2nd June.

No. 47.—Liver Abscess. Opened below the Arch, a little to the right of the Mammary Line. Tube removed and afterwards re-introduced. Recovery.

A Hindu, aged 26, was admitted into the Medical College Hospital, Calcutta, on 3rd May 1892. He had suffered from fever for one month. There was a large baggy swelling in the right hypochondriac region; the lower intercostal spaces were obliterated, and there was distinct fluctuation about 1 inch below the costal arch. After exploration had detected pus, a free incision was made below the arch, a little to the right of the mammary line, and 30 oz. of pus evacuated. The patient did well, and the tube was removed on the 26th; but from the 29th fever came on again, the discharge began again, and the tube was reintroduced on the 31st. Pain came on 3 or 4 inches to the right of the opening. On puncture no pus was found. The fever ceased for a short time, but came on again, and the pain became intense. On 21st June a solid resilient mass was observed to occupy the right lumbar region. This was punctured in three directions, but no pus found. Fever and discharge still continued. Between the 26th June and 14th July five large masses of slough were removed from the cavity of the wound, fever continuing all the time. The nature of the swelling in the right lumbar region was never satisfactorily explained. The patient recovered, and was discharged on 11th August.

This case illustrates the necessity for a drainage tube of a large calibre.

No. 48.—Liver Abscess bursting into the Lung. Puncture. Death.

A Mohammedan, aged 20, was admitted into the Medical College Hospital, Calcutta, on 5th May 1892. He had pain for two months, and a cough for a fortnight previously. There was large expectoration of pus, and for some time the case proceeded favourably. Puncture was made when it was clear that the case was not going on so well. The patient died exhausted on 28th July.

Post-mortem twenty-three hours after death.—Rigor mortis nil. Lividity nil. General œdema. Subcutaneous fat scanty. Muscles pale and wasted. Intestines and stomach much distended; 16 oz. of thick dark brown fluid removed from abdominal cavity and 2 oz. from the left pleural cavity, of a sanguineous nature. Lower border of liver ½ inch above the arch and 1 inch below the xiphoid cartilage.

Thorax.—On dissecting the skin and muscles from the right side of chest, a cavity between the sixth and seventh rib was opened, and from this 24 oz. of thick hepatic pus came. The ribs above and below were eroded for 2½ inches. An abscess was evidently tending to burst here in the mid-axillary line.

Pericardium contained 2 oz. of dark port-wine coloured fluid.

Heart = 5 oz.; healthy.

Pleura in front very firmly adherent to chest wall.

Right lung.—On endeavouring to determine the level of the liver, a cavity above the liver was opened into, the same as was opened from outside the thorax. The finger passed easily through the diaphragm, which had apparently sloughed away. On removing the right lung, liver, and ribs together, a shallow abscess cavity with sloughy walls was found on the surface of the liver, and this was continuous with the cavity through the diaphragm and right pleura, and the large suppurating cavity in the base of the right lung which replaced almost the whole of the lower lobe. Its walls were ragged and sloughy.

Left lung = 13 oz.; very much decomposed. A quantity of dark brownish liquid exuded from it.

Great omentum does not cover the distended intestine. At the lower end of the large intestine, some shallow punched out ulcers varying in size from a sixpence to one-third that size.

Spleen = 4 oz.; decomposed, soft, and black.

Stomach and *duodenum.*—Large patches of slate colour, apparently due to decomposition.

Liver dark brown, and friable from decomposition; no other abscess; substance apparently normal.

Gall-bladder contains a small quantity of bright yellow bile.

Kidneys.—Medullary part of both pale. No reaction with iodine. Both weighed 3 oz.

No. 49.—Liver Abscess. Opened in the Tenth Intercostal Space, anterior Axillary Line. Recovery.

A Hindu, aged 32, was admitted into the Medical College Hospital, Calcutta, on 15th May 1892. He had suffered from fever for three months, and liver pain for two months previously. There was a large swelling over the right infra-axillary region, soft and tender. This was punctured in the tenth intercostal space, and pus found. Free incision was made in a transverse direction, and 32 oz. of pus evacuated. The temperature only rose once over 100°, the patient progressed well, and was discharged well on 10th June 1892.

No. 50.—Liver Abscess. Opened below the Arch, ½ inch to right of Median Line. Death.

A Hindu, aged 25, was admitted into the Medical College Hospital, Calcutta, on 19th May 1892. He had had pain in the liver for two months previously. He was emaciated, but tongue clean and bowels regular. Area of dulness extended from lower edge of fifth rib to 2 inches below the costal arch, the latter place being very tender; right intercostals obliterated. Fever in the evening. On the 31st the liver was punctured below the arch, ½ inch to right of median line, and pus detected. A 5-inch drainage tube was inserted, and 12 oz. of pus evacuated. The case progressed favourably till shortly after the 14th, when fever returned. A few days later the discharge was found to be offensive. A double drainage tube was introduced, and the cavity washed out daily with perchloride, and sloughs came away. Signs of septicæmia continuing, a counter opening was made in the axillary line, between the ribs; but the patient gradually sank, and died on the 4th July.

No. 51.—Liver Abscess. Opened ½ inch below the Arch, and a little to the left of the Mammary Line. Recovery.

A Hindu, aged 22, was admitted into the Medical College Hospital, Calcutta, on 1st June 1892. He had had fever and pain in the hepatic region for eighteen days previously. There was slight bulging on the right side at the lower part of the chest; there was increased area of dulness, and the spleen was enlarged. On 2nd June the pleura was explored in the seventh intercostal space, and some clear serous fluid evacuated. On the 4th the liver was punctured below the arch, and pus detected. A free incision was then made about ½ inch below the arch, and a little to the left of the mammary line. After the evacuation of about 10 oz. of pus, the discharge ceased suddenly, but the fever still continued. The probe accidentally discovered another cavity, from which about 6 oz. of pus of quite another character were evacuated. The fever continued for several days, and multiple abscess was feared. The fever stopped on 19th June, and the patient was discharged recovered on 12th July.

No. 52.—Liver Abscess. Opened 2 inches below the Costal Arch, a little to the left of the right Mammary Line. Death from Disease of Rectum.

A Hindu, aged 30, was admitted to the Medical College Hospital, Calcutta, on 15th July 1892. Had been suffering from pain in the hepatic region for 1½ months previously. There was a large, fluctuating tumour under the right costal arch. An incision was made 2 inches below the costal arch, a little to the left of the right mammary line, and 20 oz. of pus evacuated.

The patient progressed favourably for a time. On 23rd July he had piles and much rectal pain. On 4th August a portion of the rectum was excised, and a large neoplastic tumour, with much ulceration, discovered. The liver wound still continued to do well. The patient died on 13th August of malignant disease of the rectum.

The writer cannot conclude this paper without expressing his grateful acknowledgments to the distinguished medical authorities who have contributed so many cases illustrative of the clinical aspects of hepatic suppuration.

CHAPTER XIX.

SUNSTROKE.

BY SIR JOSEPH FAYRER, K.C.S.I., LL.D., M.D., F.R.S.

Natural History and Etiology.—Under the designation of sunstroke, coup de soleil, insolation, erythismus tropicus, ictus solis, sun fever, thermic fever, heat apoplexy, heat asphyxia, calenture, are included certain pathological conditions and their concomitant symptoms, which are due to the effects of exposure to excessive solar or artificial heat, generally, though not invariably, to the former, and which occur most frequently in tropical or subtropical climates, especially under peculiar atmospheric and meteorological conditions; but also in temperate climates during seasons of exceptional heat, or under other circumstances where the body is exposed to an inordinately high temperature, whether solar or artificial.

The morbid conditions, though mainly due to heat, are liable to be modified by climatic conditions, but are likely to occur whereever and whenever the individual is exposed to a high temperature, especially if the heated atmosphere is contaminated by moisture and gaseous impurities, or when individual peculiarities render the sufferer more susceptible to noxious influences.

Under the general designation of insolation are included—

1. Syncope or heat exhaustion.

2. A condition resembling shock, caused by the action of the direct rays of the sun on the head or spine, when the nervous centres may be affected, and death may result from failure of respiration and circulation.

3. A condition in which the blood and tissues are overheated, either by the direct action of the sun or a high temperature in the shade. Vasomotor paralysis, intense pyrexia, with failure of respiration and circulation, often ensue, and induce fatal results; or if recovery take place, it may be incomplete, owing to molecular change, which causes a variety of symptoms indicative of lesion of

the nerve centres—it may be death. All these conditions are well exemplified in India, and in other hot and tropical climates.

To the above may be added certain cases which are complicated by cerebral hæmorrhage and its consequences; these come appropriately under the head of heat apoplexy. The symptoms in such cases are varied, depending upon the portions of the cerebro-spinal centres, which are the seat of the lesion.

The last two Annual Reports of the Sanitary Commissioner with the Government of India for 1889 and 1890 give the following information:—

In 1889 the European army in India numbered 69,266 men. Of these there were 179 admitted for heat apoplexy, of which 86 died, and 49 for sunstroke, of which 8 died, making a total of 228 admissions, or 3·3 per mille, and 94 deaths, or 1·36 per mille.

In the same year, of 3166 women with the European army in India, 2 were admitted for heat stroke, and 2 died, or ·63 per mille.

Of 6041 children, 2 were admitted for heat stroke, or ·3 per mille, and 1 died, or ·17 per mille.[1]

In the same year the Native army numbered 128,642, and of these there were 35 admissions for heat apoplexy and 8 for sunstroke; from the two together 20 died, making a total of 43 admissions, or ·3 per mille, and 20 deaths, or ·16 per mille.

The jail population for 1889 was 96,121. Of these there were 127 admissions from heat stroke, or 1·3 per mille, and 42 deaths, or ·44 per mille.

In 1890 the European army in India numbered 67,823. Of these there were 7 admissions for heat stroke, of which none died, 13 for sunstroke, of which 4 died, and 104 from heat apoplexy, of which 46 died, making a total of 126 admissions, or 1·9 per mille, and 50 deaths, or ·74 per mille.

In the same year, of 3130 women with the European army in India, 2 were admitted for heat stroke, and 2 died, or ·64 per mille.

Of 5912 children, 4 were admitted for heat stroke, or ·7 per mille, and 3 died, or ·51 per mille.

In 1890 the Native army numbered 127,746, and of these there were 19 admissions from sunstroke and 7 from heat apoplexy, and 11 deaths, giving a total of 26 admissions, or ·2 per mille, and 11 deaths, or ·09 per mille.

The jail population for 1890 was 96,610, and of these 60, or ·6

[1] This shows a remarkable immunity, if compared with reports from America; *vide* p. 698.

per mille, were admitted for heat stroke, and 23, or ·24 per mille, died.

It will be observed from the above that insolation caused the admission in 1889 of 3·3 per mille, in 1890 of 1·9 per mille, of European soldiers; in 1889 of ·63, in 1890 of ·64 per mille of European women; in 1889 of ·3 per mille, in 1890 of ·7 per mille of European children; in 1889 of ·3 per mille, in 1890 of ·2 per mille of native soldiers. The women and children being subject to much less exposure and predisposing causes of the disease, suffered much less than the men, while natives seem from their racial peculiarities to enjoy greater immunity than Europeans.

These returns show also how the effects of heat influence certain classes of persons whose circumstances and conditions of living are under direct observation and control. Reliable data are thus afforded on which to determine this element in vital statistics, whilst they show that the effects of heat form an important item in the general death-rate of India; no doubt it is similar in other tropical countries, presenting similar climatic and thermal peculiarities.

These returns also show how the incidence of the disease varies from year to year.

The influence of age and length of residence in a hot country in determining the incidence of this disease is shown in the following figures, taken from the same reports:—

Deaths in the European Army from Apoplexy and Heat Stroke, according to Age.

	1889.	1890.
Under 24, . . .	49, or 1·48 per mille.	23, or ·70 per mille.
25-29, ...	34, or 1·44 ,,	20, or ·81 ,,
30-34, ...	13, or 1·83 ,,	6, or 1·01 ,,
35 upwards, . . .	8, or 2·52 ,,	5, or 1·90 ,,

Deaths in the European Army from Apoplexy and Heat Stroke, according to length of Service.

	1889.	1890.
1st and 2nd years, .	47, or 2·15 per mille.	23, or 1·10 per mille.
3rd to 5th, ,, . .	37, or 1·21 ,,	21, or ·66 ,,
6th to 10th, ,, . .	14, or 1·17 ,,	6, or ·54 ,,
11th to 15th, ,, . .	5, or 2·69 ,,	3, or 1·91 ,,
15th and upwards, .	1, or 1·23 ,,	1, or 1·58 ,,

In these last returns, apoplexy and heat stroke are bracketed together, all being attributable to heat. They indicate the severe forms of the disease. No doubt many simpler cases in which complete recovery follows, as well as some which exemplify other effects of heat combined with climatic influences, are not included.

Admissions and deaths from hemiplegia are not included under the head of heatstroke in the official returns, but as many of them were probably due to insolation, it is as well to mention them.

In 1889 there were 4 admissions and no deaths in the European army from hemiplegia, 27 admissions and 2 deaths in the Native army, and 21 admissions and 5 deaths in the jail population.

In 1890 there were 11 admissions and 1 death in the European army from hemiplegia, 26 admissions and 1 death in the Native army, and 25 admissions and 1 death in the jail population.

The foregoing returns represent the average incidence of sunstroke in ordinary years in India among a class of persons whose conditions of life being officially cared for, are hygienically fairly correct. No doubt, in others less protected, as in the scattered European, Eurasian, and immense native population, the incidence of the disease is often greater. In different seasons, when there are greater accessions and waves of heat, the disease and the mortality from it increase. A most remarkable example of this has occurred this year (1892) in the United States, within certain geographical limits, east of the Mississippi; insolation has been most prevalent and fatal, and children have suffered severely, probably far beyond what is usual. Such waves of high temperature appear to recur at uncertain intervals.

The effects of high temperature on the general health of Europeans or other residents in hot climates is a question of much hygienic importance, especially when it is considered in connection with other causes of disease, such as malaria; but this can only be glanced at as connected with the more striking and special action of heat on the constitution.

Of all animals, man appears to possess the greatest power of adapting himself to changes of climate and of maintaining health under extreme variation of heat and cold, provided always that his constitution be unimpaired by disease or exhaustion, and that his viscera be in a normal state of functional activity, his diet sufficient in quantity and appropriate in quality. His body has the inherent power of maintaining and regulating an equable temperature under extremes of heat and cold; his state of health, idiosyncrasy, and racial peculiarity contributing towards this object.

Vigorous, healthy Europeans have the power of tolerating, nay,

of thriving, in great heat; those who have become accustomed to the climate, and have learnt how to live judiciously and to protect themselves against noxious influences, are more tolerant than recent arrivals, whilst a negro or a native of India can not only sustain, but bear even with pleasure on the bare head and body, an amount of heat which would prostrate a European; but if the temperature rise above a certain point, they too suffer, as is well illustrated in India, where many natives die from the effects of heat alone, especially during the hot winds.

Heat is so intimately associated with other climatic conditions, such as electric, hygrometric, and barometric states of the atmosphere, telluric and other miasmata, that it would be difficult to say how far it alone is concerned in affecting the health of those who are exposed to their combined operations. It seems, indeed, probable that the deteriorating effects of hot climates are due more to these accessories than to heat itself. It is with the action of heat alone, however, that we are now chiefly concerned.

The effects of the combination of those conditions which characterise hot climates are not yet sufficiently determined, and it is to colonial, naval, and military medical officers that one may naturally look for further enlightenment. Though the human body in a state of health possesses the autonomous faculty of preserving an equilibrium of temperature under varying conditions, yet continued exposure to great heat cannot be long endured with impunity. The observations of Parkes, Becker, and others show its deteriorating effects on the nervous system, on the secretory and eliminative functions, and on those of digestion and assimilation. It also causes fever in all degrees of intensity, from the simplest to the most ardent, when it is often combined with miasmatic poisoning, extreme cases passing into those dangerous conditions which are known as sunstroke.

The degree to which heat operates is modified by the hygrometric state of the atmosphere. Dry hot winds, such as those of Upper India, are better tolerated than the moist, though relatively cooler, air of Bengal and Southern India, with a temperature above 85° or 90° F. Dry heat favours perspiration, thus keeping the body cool; damp air, though it may be at a lower temperature, impedes evaporation, and thus diminishes the natural refrigerating power of the body.

Toleration of heat depends also on the state of health, vigour of constitution, and temperance in all things, especially in the use of food and alcohol. Healthy persons, as before said, bear a high temperature well, if transpiration from the surface of the body be

unimpeded by atmospheric moisture or other causes, and if the air be pure. But when, from any cause, perspiration fails, or the natural eliminative functions be interfered with, suffering soon ensues, and the danger of ardent fever or insolation and cerebral congestion and hæmorrhage is great, and is enhanced when the temperature of the moist and impure air rises above that of the normal heat of the body. That these evil consequences are not due to the direct action of the sun alone, is shown by the fact that many of the fatal cases take place in rooms, tents, or hospitals, or even at night or in the early hours of the morning before sunrise, especially if the air be vitiated as well as hot, and among those who are predisposed by disease, debility, or irregular and intemperate habits.

These affections are most prevalent in the hottest parts of the year; in India, the months of April, May, June, and July give the largest returns of cases.

It is hardly possible to assign any particular degree of temperature as one of excessive danger; because, as before remarked, the tolerance of heat is very great in the case of persons in perfect health in a pure atmosphere, and also in the dark-skinned races; but under the conditions before stated the danger increases when the temperature is equal to or higher than that of the body.

The following extracts from the *Times and Observer* of August 1892, illustrate in a remarkable manner the effects of an unusually high temperature, such as has been experienced in the United States during one of the waves of excessive heat which periodically pass over that part of the world, also in Europe, in France, and Germany:—

Times, August 1, 1892:—

New York, *July* 30.

The extraordinary heat which has prevailed here during the week still continues. Yesterday the thermometer at the Reform Club registered 107° in the shade, and 98 sudden deaths occurred here during the day, which were directly or indirectly due to the heat. This is a record which was not exceeded even during the memorable years of 1847 and 1876. The total number of deaths in New York yesterday was 223, of which 111 were those of children under the age of five. In the poor tenement districts, where the population is denser than in any other city in the world, the death-rate among the children has been simply appalling. Much sickness, indeed, prevails among all classes, as sleep has been almost impossible since last Saturday, the heat at night being unendurable. The coroners, the hospital staffs, and the ambulance corps are alike unequal to the unusual demands made upon them.

At Chicago the morgue is overfilled, and no more bodies can be received there. Meanwhile, the drain on the water service there has been so great that the supply has given out.

This terrible heat, which has now lasted seven days, is general in all the country east of the Mississippi.

July 31.

The temperature is at length beginning to decline, although the death-rate is still very high. Ninety deaths due to the effects of the heat occurred here yesterday, while the number of cases of sunstroke and prostration is quite extraordinary. The mortality among horses has not as yet shown any signs of decrease. In addition to the many factories and offices which have been closed during the greater part of the past week, a considerable number of shops remained unopened yesterday.

The heat is still terribly oppressive, and it is estimated that the atmosphere contains 79 per cent. of moisture. This state of affairs, continuing as it has done both day and night, has affected everybody, and sickness of a more or less serious character is almost universal. The total number of deaths which have occurred from the heat in New York up to the present is 296, and the total mortality for the week up to noon to-day is 1434, which is the largest figure that has been reached for twenty years past. According to the latest reports, great relief has already been experienced by the approaching cold wave in the Western States, where almost as much suffering has been caused by the heat as in the Eastern States.

Times, August 20, 1892:—

PARIS, *Aug.* 19.

Tropical heat continues to prevail here, and the temperature in Paris yesterday was again 96°·8 in the shade. Fresh cases of sunstroke occurred, and one of those terminated fatally. When the men employed in the meat market at La Villette went to unload the cattle trucks this morning they found about 100 oxen and 300 pigs dead from suffocation, or in a state of complete collapse. Advices from the departments report that the heat is everywhere abnormal.

Evening.

This evening the weather has become sensibly cooler, and rain has begun to fall heavily. The telegrams from the provinces also announce an abatement of the intense heat, accompanied by storms of rain.—*Reuter.*

TOURS, *Aug.* 19.

During the military manœuvres here to-day there were four cases of sunstroke among the troops, and besides these several men were taken back to barracks seriously ill.—*Reuter.*

LAON, *Aug.* 19.

Owing to the great heat the military manœuvres here have been suspended, and the troops have returned to garrison by night marches. There has been one death and about thirty cases of illness from the heat.—*Reuter.*

BERLIN, *Aug.* 19.

The heat prevailing here is almost unbearable, the shade temperature at 5 P.M. registering 89°·6. At the Imperial Ammunition Factory at Spandau work is suspended owing to the heat. On the Emperor's instructions, his Majesty's Aide-de-Camp-General to-day telegraphed to the civil authorities

here requesting them, until the great heat abates, to suspend afternoon instruction in the schools. The city magistracy adopted a resolution in accordance with his Majesty's request.—*Reuter.*

VIENNA, *Aug.* 19.

The heat in Vienna continues to be excessive. To-day seven persons were conveyed to the hospitals suffering from sunstroke, two of them dying shortly after admission. The cases of heat prostration are innumerable, and three have had a fatal issue. Orders have been given for the suspension of the manœuvres of the Vienna garrison.—*Reuter.*

Times, August 22, 1892:—

VENICE, *Aug.* 21.

The whole of Northern Italy has been visited by a protracted period of unusual heat, the maximum readings ranging above 90° F., with unusually high minima. There is no present indication of a change in the weather. Many cases of sunstroke have occurred among the troops now assembling in preparation for the manœuvres; but, owing to the excellent precautions taken by the military authorities, fatal results are extremely rare.—*Our Own Correspondent.*

TRIESTE, *Aug.* 21.

The heat during the past few days has registered 110° F. in the shade, and sea water shows a temperature of 90°. A telegram, received from Venice, reports that the 36th Italian Infantry Regiment has undergone fearful sufferings while on the march from Venice to Monselice. When near Dolo the infantrymen succumbed in large numbers, and upwards of 100 of them were lying exhausted by the roadside. Twenty of them were taken to the hospital at Dolo suffering from sunstroke.—*Reuter.*

VIENNA, *Aug.* 21.

The weather is excessively hot, although the thermometer at noon to-day registered 3° less—namely, 102° F. in the shade. Eight deaths have occurred through the heat since Thursday last.—*Reuter.*

BERLIN, *Aug.* 21.

The intense heat has caused great loss of life all over Germany. Telegrams from Halle state that the soldiers of the 93rd Regiment have suffered terribly on the march. One man dropped down dead, and forty were rendered unconscious by sunstroke. During the brigade manœuvres at Posen many cases of sunstroke occurred. The soldiers of the Durlacher and Grenadier battalions at Carlsruhe were forced to manœuvre under the burning sun, and many of them dropped down in the field overcome by the heat. Four men of the Durlacher Regiment and two Grenadiers have since died. At Stuttgart there have been numerous cases of sunstroke, several of which terminated fatally. The heat is causing great loss to shippers, the waters of the Spree, Elbe, Havel, and Oder having fallen so low that the navigation of barges and other craft is interrupted.

At the Meteorological Bureau over 95° F. were registered yesterday. The Press expresses much indignation that the military authorities should persist in carrying out the manœuvres in such hot weather.—*Reuter.*

Times, August 23, 1892 :—

TRIESTE, *Aug.* 22.

The tropical heat has had most fatal results for the troops manœuvring on the Karst Hills, near Senosece, Carniola. Two hundred cases of sunstroke occurred among the men, and eleven of them terminated fatally.—*Reuter.*

VIENNA, *Aug.* 22.

For the past eight days almost unbearable heat has prevailed here, and although a cooler atmosphere is reported from France and Switzerland, no fall in the thermometer has occurred here. Early this morning the sky was overcast and a thunderstorm appeared imminent, but the weather became clear again, and the heat remains excessive. This evening a violent thunderstorm broke over the city.—*Our Correspondent.*

VIENNA, *Aug.* 22.

Reports continue to be received of the sufferings experienced by the troops in consequence of the heat. At Agram last Wednesday, while the 71st Brigade was engaged in an evolutionary march, it is computed that at least one-third of the men dropped out of the ranks overcome by the heat. The officers also suffered severely, and a first lieutenant named Vollman was found dead on Friday last by the roadside. The total number of deaths directly attributable to the heat which has occurred here during the last five days amounts to fourteen.—*Reuter.*

BUDAPEST, *Aug.* 22.

Two persons died from the excessive heat here to-day.—*Reuter.*

PARIS, *Aug.* 22.

So many soldiers have been overcome by the heat, owing to marches not having been countermanded, that M. de Freycinet has issued a circular requiring reports on all such cases in order that the responsibility for them may be ascertained.—*Our Own Correspondent.*

Times, August 25, 1892 :—

BERLIN, *Aug.* 24.

To-day has brought no alleviation of the oppressive heat in Berlin, and that it still continues in the provinces is shown by the telegrams announcing deaths from sunstroke. Yesterday the chief tramway company in the capital lost seven horses from this cause. In some cases the morning school hours were either shortened or omitted, the thermometer showing over 84° F. in the shade at 10 o'clock in the morning. A telegram from Thorn, in West Prussia, states that the thermometer reached the height of 95° F. in the shade, and that from the country round come complaints of the destruction to crops of all sorts occasioned by the scorching heat. The exercises of the troops there were shortened and carried out in the early morning.—*Our Own Correspondent.*

BERLIN, *Aug.* 24.

Five fatal cases of sunstroke occurred to-day in the vicinity of Thorn. At Dantzic fifty men of the 125th Regiment were rendered unconscious to-day by the heat, and at Bromberg a bandsman of the 129th Regiment

died from the same cause. At Homburg the shade temperature is well over 100° F., and three deaths have occurred. The situation in Berlin and Charlottenburg is rendered very serious by the fact that the water supply is failing and becoming undrinkable. At Rinteln, in Brandenburg, twelve houses were burned to ashes to-day, owing to the absence of water to quench the flames.—*Reuter.*

SOFIA, *Aug.* 23.

The wave of intense heat which has passed over Western Europe has arrived in Bulgaria, and has been accompanied by thunderstorms and violent showers of hail, which have done serious and, it is feared, irreparable damage to the maize crops in some of the southern districts. In several places the plants have been entirely denuded of their leaves. The vineyards have also suffered severely.—*Our Correspondent.*

VIENNA, *Aug.* 24.

The *Neue Frei Presse* states that no fewer than 180 men of the 32nd Regiment fell out of the ranks overcome by the heat while on the march from Budapest to Fünfkircken, and that one of the soldiers succumbed. Similarily, while the 34th Regiment was marching from Kaschau to Borsod, 250 men were prostrated, and three died. The latest telegram from Budapest announces that rain is falling this evening in Hungary.

Eighteen deaths from sunstroke are officially reported to-day.—*Reuter.*

Observer, August 21, 1892:—

PARIS, *Aug.* 20.

Yesterday's storm has greatly refreshed the atmosphere. Several cases of sunstroke have been reported in the suburbs, two of which have proved fatal.

BERLIN, *Aug.* 20.

The heat here still continues intense. Several fatal cases of sunstroke have occurred, and similar fatalities are reported from various other parts of Germany.

VIENNA, *Aug.* 20.

There were two more deaths this morning from the effects of the heat, which continues as fierce as ever. Many cases are reported of people swooning in the streets.

Observer, August 28, 1892:—

TRIESTE, *Aug.* 27.

A suicidal mania has been prevalent here for the past ten days, during which no less than sixteen persons have taken their lives. Most of the suicides were due to love affairs or to mental derangement caused by the the great heat.

The above, which relate to lat. 41° N. in America, and to various parts of the Continent of Europe, show that sunstroke is not exclusively a tropical disease, but may happen wherever the temperature rises above a certain point.

All who suffer do not die; some perfectly recover, but many are permanently injured and become invalids for life, and unfit to return to a hot climate.

It has been remarked that people on board ship seldom suffer from insolation; but those who have had experience in the Red Sea, Indian Ocean, or Persian Gulf know that this alleged immunity is not universal.

Semeiology and Pathology.—In addition to the general disturbance of health depending on functional derangement, the morbid conditions which are to be attributed to the effects of a high temperature alone, and with which this article is concerned, are—

1st. Syncope from exhaustion, caused by either the direct rays of the sun or a heated atmosphere in the shade, especially when the physical or mental powers are depressed, *e.g.* in the case of engine-room men in steamers, in hot climates when the air temperature rises to 120° or more; men on parade or marching, if oppressed with clothes or accoutrements; labourers or artificers, men in hay-fields in England or in heated rooms and factories, and people in barracks, hospitals, tents and ships may suffer in this way, or may pass into the dangerous state of asphyxia or cardiac apnœa, especially if exhausted by dissipation and intemperance. The condition is one of depression; the skin is cold and pale, the pulse feeble. Death may occur from failure of the heart, but recovery is usual.

2nd. An analogous condition caused by the action of the direct rays of a powerful sun, with their intense heat and glare.

The centres may be affected, circulation and respiration fail, and death result. Recovery frequently occurs, but this may be incomplete or retarded by structural central changes, sometimes the result of cerebral hæmorrhage or meningeal inflammation. The first symptoms of exhaustion having passed, reaction may assume the form of high fever, or may indicate lesion of the cerebro-spinal system. When death occurs suddenly or rapidly, as it occasionally does, from cardiac failure by inhibition, it is sometimes attributed to the coagulation of cardiac myosin; but this is more probably a post-mortem change. The heart's action may be brought suddenly to a close by tetanic contraction of the ventricles, as shown in the experiments of C. Bernard and Lauder Brunton, as the result of the action of great heat, or death may be due to cerebral hæmorrhage. The condition of syncope or shock is liable to be followed by reaction and consecutive symptoms of varied character, affecting the nervous system; and it has been frequently observed that these do not supervene for some time, it may be days, after apparent recovery from the first urgent symptoms.

3rd. Overheating of the whole body, either by direct exposure to the solar rays or to a high temperature in the shade, causing vasomotor paralysis, intense pyrexia, failure of circulation and respiration, resulting in asphyxia, and often in death.

This form of insolation may arise from direct action of the sun's rays on the head and body. The blood, nerve and other tissues become overheated, in addition to the shock which results from the first impression of heat; there is ardent (thermic) fever; the temperature of the body rises to 106° F., or even higher; cardiac and respiratory failure soon result.

But it occurs frequently also in the shade or at night, as before said, and especially in persons who are suffering from exhaustion, fatigue, dissipation, illness, or over-indulgence in food or alcohol, or excessive obesity; and in those who have weak, fatty, or dilated hearts, especially when the air is impure from overcrowding or insufficiency of cubic space. Such, indeed, form a considerable part of the fatal attacks of the so-called heat apoplexy or asphyxia in hot climates.

Many die, some recover, but not unfrequently the recovery is imperfect; there are often sequelæ, the result of structural change, which impair health or intellect, and destroy life at last.

In the cases due to heat exhaustion and shock, the primary symptoms are those of depression,—the person becomes unconscious, or nearly so; the skin is cold, pale, and moist, with feeble pulse and, it may be, sickness. The condition is that of collapse, and in this state, if reaction be not soon established, death from syncope may result, though this is rare; recovery is generally complete, but when the direct application of intense solar heat and light have caused it, secondary lesions may result, and vertigo, twitching, muscular tremor, and temporary loss of power, indicating perhaps even more than functional derangement.

The premonitory symptoms of insolation may appear for some hours, even days, before the dangerous conditions set in. There is general malaise, restlessness, insomnia, apprehension of impending evil, precordial anxiety; hurried, gasping, shallow breathing; a feeling of constriction round the chest; vertigo, headache, nausea or even vomiting; anorexia, great thirst, a desire to micturate frequently, and fervent heat of the skin. These symptoms, one or other of them, becoming aggravated, the temperature rises to 104°, 106°, or even 110° F.; dyspnœa and restlessness increase; the head, face, neck, and skin of the body generally become red and livid, sometimes dry, sometimes moist with perspiration; pulse full and labouring, carotid pulsation very perceptible; pupils contract closely, to dilate

again widely before death; unconsciousness passes into complete coma, stertor, convulsions—often epileptiform; relaxation of sphincters and suppression of urine may precede death.

The symptoms indicate a profoundly disturbed state of the cerebro-spinal centres; the intense heat is incompatible with the due performance of their functions, and death will rapidly result unless prompt aid be given; indeed, it frequently does so despite all treatment.

Death is caused by apnœa, asphyxia, or cardiac failure; there may be cerebral hæmorrhage and effusion, whilst petechial patches appear on the body in some cases.

Though some recover, or rather do not die, a large proportion are permanently injured, and become more or less invalids for life, which, as before said, is frequently shortened by obscure cerebral or meningeal changes, which affect the sufferer in varying degrees of intensity, producing irritability, impaired memory, headache, mania, or dementia, partial or complete paralysis, partial or complete blindness or deafness, intolerance of the sun's rays, even in the mildest cases, rendering a person incapable of serving in hot climates, or of enduring exposure to the sun in temperate climates. This may end gradually in fatuity, dementia or epilepsy, or both, or chronic meningitis; with thickening of the calvaria, and intense cephalalgia; the patient may suffer in a lesser degree from disordered innervation and general functional derangement.

Morbid Anatomy.—When death has occurred from syncope or shock, there is no very obvious morbid change; the brain with its membranes, and the lungs, are sometimes, not always, congested; the venous trunks, especially those of the abdomen and the right cavities of the heart, may be full of blood, which is dark, grumous, often imperfectly coagulated, and deficient in oxygen; the abdominal viscera are congested; lividity of the body comes on rapidly after death.

In animals killed by exposure to a high temperature, the blood and tissues having been heated to 110° F. or higher, the heart has been found in some cases contracted, in others it remained flaccid.

In death from thermic fever and the more intense form of insolation, the lungs are often deeply congested, the heart in some cases is contracted, it may be from coagulation of the myosin, and the venous system is engorged.

The body may be marked with petechial patches or livid ecchymoses which began before death. The blood is dark, grumous, and acid in reaction; the globules are crenated, and do not readily form into rouleaux.

The body for some time after death retains a high temperature; in early, post-mortem examination, necessary in hot climates, the interior and viscera, when opened, feel pungently warm, and dark blood drips freely from the incisions. Rigor mortis comes on rapidly, the brain and membranes are congested, and there may be cerebral hæmorrhage, or effusion of serum into its substances or cavities.

Treatment.—Preventive treatment is most important; careful protection of the head and spine from the direct rays of the sun by proper solah (pith) hats is enjoined; light clothing—fine woollen is the best—should be worn, and cotton pads over the spine.

Precaution against over-fatigue or exertion, either physical or mental, and freedom from excitement on the one hand, or depression from fatigue or want of food on the other, is most desirable. Rooms should be well ventilated, and the atmosphere kept as pure as possible by constant ingress and egress of air.

The punkah, thermantidote, and tattie, or other means of cooling the air, should be freely used; and exposure to the sun or exertion during the hotter parts of the day should be avoided. During the hot, still nights—a most dangerous time—punkahs should be kept freely going, windows open; the body should be protected by light woollen clothing to guard against sudden alternation of temperature.

Moderation is essential; excess of animal food and alcoholic drinks should be scrupulously avoided; the diet should be light and nutritious but unstimulating, while iced water should be freely drunk. A moderate amount of physical exercise and mental occupation should be encouraged. Regularity in living, and careful attention to the condition of the bowels should be enforced. No one is more likely to suffer from insolation than he who has undergone mental or physical exhaustion, or who has indulged in the abuse of alcohol or excess of food.

Healthy men who lead regular lives withstand heat and preserve health in a measure that would be regarded as remarkable if contrasted with the effects of the same degree of heat on those who do not observe these precautions.

In simple heat exhaustion, remove the patient to a cool place, in the shade if possible. Douche with cold water on the head and chest, not too prolonged, so as to depress. Remove all tight and oppressive clothing, apply ammonia to the nostrils, and give a slight stimulant. If depression be prolonged or profound, administer stimulants and restoratives; let the patient avoid exertion or exposure to heat as much as possible.

In the steamers in the Red Sea and Indian Ocean, stokers,

usually Africans, are occasionally brought up from the furnaces unconscious from heat exhaustion, but are quickly restored by fresh air and cold douching.

When a man is struck down by the hot sun, use the cold douche freely; the object is twofold, to rouse by reflex action and to reduce temperature.

At the capture of Rangoon in 1853, in the month of April, numbers of men were struck down by the fierce sun. They were brought to the field-hospital quite unconscious, clad in thick red coats with leather stocks. They were treated with free douching (with the bhistee's mussuck), and all recovered except one or two who had been bled. In certain cases flagellation with a sweeper's broom was effective in rousing; in others, mustard poultices to the body and legs, and stimulating enemata, with a diffusible stimulant by the mouth were useful.

When it is said that they recovered, the reaction at the time is referred to. In some there were consecutive symptoms of fever, cephalalgia, etc.; and could their subsequent history be traced, it would probably be found that complete recovery never took place, and that in others chronic intracranial mischief remained.

Exposure to the sun should be carefully guarded against; and unless recovery has been complete and rapid, the sufferer should be removed to a colder climate, when he must be protected from excitement, mental or physical; and continued care must be taken to prevent excess of every kind.

In severe cases of thermic fever or insolation, the object is to reduce temperature as speedily as possible before tissue changes take place. The hyperpyrexia is due not only to the direct action of heat on the tissues, but to vasomotor disturbance, and therefore quinine used hypodermically may produce good results by reducing temperature, and should be used. For the same purpose morphia has been suggested; but its benefit seems questionable. Bleeding has been abandoned except in rare and exceptional cases, where it may be necessary to relieve an overdistended and labouring right heart. In ordinary cases, though temporary relief may have been afforded by it, the improvement has been transient, and followed by relapse into a more dangerous condition. No absolute rule about bleeding can be laid down in this or in any other disease; it may, for reasons above mentioned, be the lesser evil, and if so, should be adopted. Treatment generally consists in cold affusion and the application of ice, care being observed not to reduce the body temperature too low; a thermometer must be the guide. The bowels should be relieved by calomel, colocynth, and saline purgatives

and enemata; whilst antipyrin, aconite, acetate of ammonia, quinine, or other antipyretic may be given till the temperature be reduced. Sinapisms to the chest may be useful.

Blisters are sometimes applied to the neck, but rarely, in the earlier stages at all events, with any benefit. In the epileptiform convulsions which sometimes occur, inhalation of chloroform may be useful, but it must be carefully administered.

The earliest and most severe symptoms having subsided, the fever which follows must be treated on ordinary principles by continuance of quinine and antipyretics as already mentioned, whilst the nourishment must be light and unstimulating. As the case progresses, if symptoms of intracranial mischief supervene, indicating meningeal lesion, iodide and bromide of potassium and counter-irritation may be of service.

It is essential in the treatment of these cases that perfect rest and tranquillity of mind and body should be enjoined. When insomnia is distressing, hypnotics may be useful; but they should always be administered with great caution. The patient should be protected from excitement and fatigue, and restriction should be imposed upon the use of alcoholic stimulants. These precautions should be continued for some time after apparent recovery has taken place. The sufferer should not be permitted to return to a hot climate for a considerable period after health has been restored, if ever; and he should be guarded even in this country against all exposure to the sun or to a heated atmosphere.

The sequelæ of sunstroke are often very distressing, rendering the patient a source of much anxiety to himself and to his friends.

The less severe symptoms, such as those of the slighter form of meningitis or of cerebral mischief, occasionally pass away after protracted residence in a cold climate; but they are frequently the cause of suffering, danger, and shortening of life, and offer some of the saddest proofs of the evils of residence in tropical countries.

The treatment of these sequelæ will depend upon the indications of the extent of the mischief and the part of the nervous system which has suffered. These may vary so much as to render it impossible to give more definite directions here. For further details, reference should be made to treatises on cerebro-spinal and mental diseases generally.

This article should not be concluded without reference to the valuable monographs on sunstroke by Professors Longmore, Maclean, Aitken, the late Drs. Hill and Barclay, and especially to a most valuable work by Dr. H. C. Wood, of Philadelphia, which deals exhaustively with the etiology, pathology, and treatment of all the forms and phases of insolation.

Illustrative Cases.

The following cases illustrate the immediate and consecutive phenomena in their varying degrees of intensity, attending the effects of heat:—

No. 1.—Case of Heat Exhaustion.

A young officer, tall, powerful, and vigorous, probably a somewhat irregular and careless liver, but not intemperate, was on duty during the month of January at an open-air church parade in camp at Delhi. The sun's rays were powerful, and the atmosphere somewhat hot at that time of the day. While standing at the head of his men he was seen to look pale, stagger, and fall over; being carried into a tent, it was found that he was in a state of syncope from exhaustion. His uniform was loosened, cold douches applied, and a mild stimulant administered. After a brief period of unconsciousness, and some long-drawn, gasping sighs, he gradually returned to consciousness, and after a day or two to duty, no evil result ensuing.

No. 2.—Case of Sunstroke producing transient but recurring Cerebral Disturbance.

An officer of the Survey, aged 27, when in Beluchistan, was struck down by the sun in October 1889, after having been out some three or four hours. He did not absolutely lose consciousness, but remembers feeling faint and giddy, with confusion of ideas. After an hour he recovered sufficiently to walk a distance of two miles. He was recommended to wear tinted glasses, and returned to work a week or ten days afterwards. He was unable do much without feeling faint, and finally had to give up work.

Early in December he was recalled to Calcutta. At different times he still experienced the feelings of giddiness and faintness,—sometimes when out in the sun in the morning, sometimes when working by an assay furnace, sometimes for no apparent reason.

During the rains he became comparatively well, and afterwards could go out sometimes in the sun, especially in the evening, without being much distressed, though it had the same effect once or twice.

After his return to England in October 1890, heat on his head or glare occasionally produced feelings of distress, and made him half-unconscious of his surroundings.

Besides these symptoms he suffered from nervousness,—such as a desire to jump down if he found himself on a height,—and occasional inability to control his thoughts. These disappeared, however, when his attention was well occupied.

The last report was favourable, but no recent information has been received.

No. 3.—A simple Case of Sunstroke. Recovery.

An officer, aged 24, of sanguine temperament and regular habits, who had been rather more than four years in India, had suffered from fever in Burma and Quetta previous to January 1891. He was then attending a

musketry class in Poona, where he was much exposed to the sun. One day towards the end of the course, after work in the open air, he became faint, and lost consciousness to a slight extent. In April he was again much exposed to the sun, and on 26th May he was placed on the sick list for ague, but returned to duty on 1st June. On 22nd June he was again invalided with symptoms of shock to the nervous system. He had intense headache, aggravated by exposure to light or any mental work, very nervous, pain in the spinal region, and most intensely in the cervical portion; pains in the front of the chest, pulse slow and irregular. Giddiness when suddenly standing up, pupils dilated, for some days suffered from double vision if he attempted to read. He improved under nervine tonics and rest, and during the cloudy weather attempted to work on 18th July, but was placed on the sick list again on the 22nd.

Patient was sent to England on leave for one year in July 1891.

This officer regained his health in England, but was not permitted to go to India till the cold season of 1892.

No. 4.—Case of Insolation complicated by previous Concussion of the Brain.

An officer, aged 27, of nervous temperament and temperate habits, who had served rather more than three years in India, was shooting in the Terai from 18th April to 2nd June 1891. During the next fortnight he suffered from fever and delirium. He went to another station on the 18th June, and when he came under observation soon after, he was completely delirious; temperature subnormal, pulse moderate; under the hallucination that he had committed some terrible crime. Remained in the same condition for a week, sleep being obtained only by means of hypnotics; food administered with great difficulty. On 24th June symptoms of cerebral effusion showed themselves, which were relieved by a blister to the scalp. Amendment then began, and convalescence was established by 1st July.

It should be mentioned that this officer had a fall from his horse in January 1891, resulting in a broken collar-bone and some concussion of the brain, from which he soon recovered. The treatment, besides that already mentioned, consisted of purgatives, and subsequently tonics.

In July, leave to England for one year was granted. He recovered, and was allowed to return to India.

No. 5.—Case of Sunstroke, ending in Dementia.

A young man, aged 22, after exposure to the sun when shooting in India, soon after his return to his quarters became insensible, with stertorous breathing and convulsions. He gradually became conscious, but with loss of power in the lower extremities, loss of speech, and defective intelligence. He was sent to an asylum in 1878, suffering from dementia; he had then recovered the use of his limbs, but mental power was weak. He improved considerably, but tonic contractions of the extensor muscles of the right arm set in. Subsequently he improved mentally, but his mind remained weak, and he was transferred to another asylum in 1892.

The notes of this case were furnished by Dr. Christie and Dr. Birkett.

No. 6.—Case of Thermic Fever with Cerebral Disturbance, the result of Insolation. Recovery.

A judicial officer of nervous temperament and temperate habits, aged 23, who had been two years in India, exposed himself to a hot sun on 7th February 1890, and returned to his quarters in the evening, complaining of a feeling of drowsiness, queer sensation in the head, and oppression across the chest. A purgative, followed by a diaphoretic mixture, was prescribed, with evaporating lotion to the head. On the 9th, fever supervened; he had again exposed himself to the sun; at 8 P.M. his temperature was 104°·8 and pulse 115. Antifebrin was at once administered, and the headache and chest symptoms were relieved. On the 10th, 11th, and 12th his temperature remained high, but on the latter day he felt much better. On the 13th he complained a good deal of his head, and bromide of potassium was administered. That night he was moved a distance of 15 miles, and when he reached his destination it was noticed that he was jaundiced. The temperature began to rise again, but fell on the 15th, being 101°·8 at 1.50 P.M.; the pulse at 7 A.M. was 72. On the 16th the temperature fell to 98°·6; he became less jaundiced, and continued to go on very well till the 22nd, when he played a game of billiards in the evening, and got a return of fever. On the 23rd he had headache, and throbbing and burning at the back of the eyeballs; diarrhœa all day. The jaundice and fever were on the increase the next day, and he had slight pain in the left parietal region. On the 26th and 27th he complained a great deal of pain on the right side of the head, and throbbing pain behind the eyes; very black under the eyes; temperature not higher than 101°. Face looked very worn and pale, and had lost a good deal of flesh. Continued to improve gradually; the headache decreasing in intensity; the temperature not rising above 100° until the 12th of March, when he got diarrhœa and a return of fever (temperature 101°), the result of eating an oyster patty. On the 13th the temperature was again normal.

Leave to Europe for nine months was granted.

The treatment consisted in the administration of antipyretics, saline aperients, phenacetin, iodide of potassium, and chloride of ammonium at different periods.

Returned to India at the end of 1891.

No. 7.—Case of Sunstroke, with Recurrences after fresh Exposure. Recovery.

An officer, aged 32, who had served thirteen years in India, had fair health till 1885, when he had an attack of unconsciousness while at work in the Adjutant's office at Kamptee, in the great heat of July. When stationed in Upper Burma on 18th November 1890 he was on musketry duty, and had to remain at the range with no shelter from the sun for nearly six hours every day. On 11th December he had not been long at the range when he felt so unwell he had to stop. He attempted to return to his quarters, but fell, and at the same time perceived a red spot in his right eye. He then became unconscious, but afterwards rallied, and was conveyed home. He felt dizzy, but his sight was unaffected, and he had no pain. Bromide of potassium and aperients were administered. Afterwards he sometimes felt confused; the memory was at times somewhat impaired, but his temperature remained normal; and on the 28th December he was

granted leave, when he went to Japan, and there recovered. On his return, at Rangoon he had an attack of vertigo accompanied by a sense of suffocation, and preceded by a feeling of tremor in his legs; he proceeded to his destination (Bhamo) in March 1891, and his former symptoms, giddiness, insomnia, irritability, etc., returned. He was often warned of an attack of impending unconsciousness by feeling of tremor in legs or hands. He gave up his appointment and returned to his regiment, then at Mangalore, on 30th May 1891.

On the day after his arrival at Mangalore he had a severe attack of vertigo while crossing the open to the adjutant's office, a distance of 400 yards. Had another attack of the same kind on the 15th June. Subsequently he had almost daily slight attacks of vertigo; reading for an hour or so, concentrating his thoughts or vision, or walking for a short distance, brought on the feeling of impending unconsciousness. Complained of feeling of "cold spots" on his head, chiefly on the left side. Bowels irregular; hearing defective; suffered from insomnia; very despondent.

Bromide of potassium, aromatic spirits of ammonia, sulphonal and aperients administered. Rest enjoined.

Was granted one year's leave, which was extended four months in May 1892. Still on leave.

No. 8.—Case of Heat Apoplexy. Probable Cerebral Hæmorrhage. Partial Recovery.

In August 1891, an officer, aged 35, who had undergone considerable exposure, with severe work, during the hot weather in Western India, and who had been otherwise fairly healthy up to that time, with the exception of febrile attacks and hepatic derangement, was attacked suddenly when on duty (survey work) in the house, by what is described by the medical officer as a fit of apoplexy, followed by slight hemiplegia, aphasia, and high fever.

The heat of the weather was intense, but the degree not stated, nor was the patient's temperature recorded, nor its duration specifically stated, further than that it remained high for several days. He was treated by cold douches, calomel purge, diaphoretics, and iodide of potash. He appears to have rather rapidly improved from the first grave symptoms, and was well enough to be sent home at the end of the following October.

He appeared, early in 1892, to be slowly but gradually improving. The hemiplegic condition much diminished, though perfect power not restored. He was nervous, tremulous, excitable, and partially aphasic; spoke imperfectly and with hesitation; facial muscles not under perfect control, but his intellectual condition fairly sound, if weak, though with partial loss of memory, and much nervous exhaustion and debility. Under medical guidance it is to be hoped that improvement may progress, but the prospect of effective service in India seems very remote.

This patient is a man of slight frame, of active and, so far as is known, of fairly careful habits. He had undergone much fatigue and nervous exhaustion in survey work, on boats, exposed to great heat; and says he had been ailing and feeling out of sorts considerably at the time, when on an extremely hot day in August he was suddenly attacked as described. This was followed by high fever, hemiplegia, and aphasia.

The great heat acting on an exhausted nervous system, no doubt, was the

exciting cause of the grave central changes, and it would appear probable that some hæmorrhage may have occurred.

The condition is a grave one, though there is room to hope for recovery, and well illustrates the dangers to which great heat, especially when combined with other depressing causes, may give rise.

The prognosis is doubtful, taking into consideration the possible extent of the structural lesion, but in any case unfavourable to return to a hot climate. It is a good example of the form of insolation known as heat apoplexy.

No. 9.—Case of Sunstroke producing profound and continued Symptoms of Cerebro-Spinal Disturbance.

An officer, aged 32, in the Indian Civil Service, who had previously enjoyed fair health, was attacked with heat apoplexy on the 9th June 1889, rather more than eight years after he had arrived in India. His temperature rose to 109° and he became unconscious, breathing stertorously. By means of the cold douche the temperature was brought down to normal, but he remained insensible for a week, passing his motions involuntarily; was for a short time hemiplegic, and his urine was albuminous. After this, the history was that of gradually increasing intelligence and slow convalescence.

In August 1889 he was still very weak, unable to walk without help, deficient in control over his muscles, grasping objects with difficulty, and unable to write intelligibly. There was no actual paralysis, but his speech was thick, though improving daily. His urine ceased to be albuminous a week after the beginning of the attack.

He was quite intelligent, and ate and slept well.

His nervous system received a severe shock, from which he will be fortunate if it ever entirely recovers, and his best chance of recovery lay in long rest, and change to a cooler and more bracing climate than that of India. For the above reasons he was sent to England.

The treatment consisted in ice to head, cold douche, purgative enemata, hypodermic injection of ether, antifebrin, quinine, blister to nape of neck, purgatives, Easton's syrup, cod-liver oil, etc., with careful nursing and dieting.

After the officer's return home, when invalided in 1889, his medical attendants recorded, in 1891, that he still presented the symptoms noted in his Indian medical statement. Combined with much nervous exhaustion there was evidence of considerable generalised muscular wasting, the interossei muscles having specially suffered. There was diminished tactile sensibility in the lower extremities, and paresis of the muscles of articulation, by which his speech was so much affected as to be sometimes only understood with great difficulty.

The first recorded grasping strength of his right hand, as tested by the dynamometer (Colin), was 57 lbs., and of his left 30 lbs.; the most recent (May 1891) was right hand 116 lbs. and left 111 lbs. Accompanying this was an improvement in the appearance of the muscles and in the walking powers, as the patient was then able to walk for short periods without assistance, though feebly.

The improvement in articulation was less marked. Recurrence of cold and of slight attacks of bronchitis during the winter and spring had somewhat retarded recovery.

A later report by the same physicians (June 1892) states that the patient's condition had somewhat improved, but not in a great degree. Strength had been gained in the flexor muscles of the hand, but not such as to fit it for writing. Power of motion had somewhat increased, but there was no elasticity of gait, and the feet were very slightly raised from the ground.

There was also change for the better in articulation, which was at times distinct though slow and abnormal, indicating effort; but it frequently became incomprehensible if the patient were weary or excited.

The last observation noted—June 1892—was to the following effect: Speech imperfect; gait feeble, short, staggering steps; intellect seemed sound; voice pretty good; gaining flesh; very anæmic; could not write; was slowly improving; Faradic current did not do any good; bromide of potash appeared to have done some good; memory not very good; no aphasia; smoked a little; took a little alcohol; patella reflexes exaggerated; sensation less acute, and muffled; did not know a match was burning his finger till he saw it.

Prognosis of ultimate recovery doubtful. Return to a hot climate in such a case as this was quite impossible.

No. 10.—Case of Insolation. Excessively high Temperature, Effusion, and Death.

A European, aged 39, was admitted to the Calcutta Medical College Hospital on 1st June 1878. The weather that day and for some days previously had been very hot and oppressive. The man was not unconscious, but stated that he had been wandering about the streets during the heat of the day, and at 3 P.M. was suddenly struck with very severe headache and giddiness. He was taken to the hospital at 6 P.M. His temperature was 107°·2 F.; face flushed; pupils a little dilated; skin dry and pungent; tongue also dry; pulse small, and scarcely perceptible at the wrist; respiration embarrassed and heaving. He was at once given a cold shower bath, placed in a wet sheeting, and ice applied to the head. A cathartic enema was given to clear the bowels, and 10 grains of quinine injected hypodermically in the arm. By these means the temperature was rapidly reduced to 102°·6, but only maintained for about an hour. It then began to rise gradually in spite of all treatment (free ice application to head and naked body generally). The face became dusky, the breathing more and more embarrassed, the heart's action irregular, the pulse failing, and unconsciousness setting in. Just before death a convulsive seizure occurred, followed by stertorous breathing and coma. The temperature just before death registered 108° F. He died in a little over two hours after admission into hospital.

Post-mortem examination (twelve and half hours after death).—Body well-nourished and muscular. The skin, especially of the back, of the arms and posterior aspects of the thighs, showed dark purplish discoloration and mottling.

Head sinuses of dura mater loaded with dark fluid blood. Vessels of pia mater engorged. Brain of normal consistency, but very vascular; the puncta vasculosa large, prominent, and numerous. Half a drachm of serous fluid in each lateral ventricle. The brain generally showed nothing abnormal, except that it bled freely wherever incisions were made. Weight 47½ oz.

Heart feebly contracted. Right cavities filled with dark fluid blood, and dilated. Left ventricle somewhat hypertrophied, and contained a little dark fluid blood, no clots. The aortic semilunar valves were thickened,

puckered, and atheromatose. The ascending aorta dilated, and the lining membrane also atheromatose. Weight of heart 14½ oz.

Lungs crepitant but intensely engorged with dark fluid blood, especially in lower lobes and posterior surfaces. On section, large quantities of dark, frothy, fluid blood escaped from the cut surfaces.

Liver congested, the veins loaded with fluid blood.

Spleen a little enlarged, and on section much dark fluid blood dripped from the surface.

Kidneys of about normal size. Marked venous congestion. The pyramids stood out prominently and were very dark.

Stomach and intestines, nothing abnormal.

The notes of this case are contributed by Professor McConnell, Physician to the Medical College Hospital, Calcutta.

No. 11.—Case of Insolation from direct Exposure. Recurrence followed by Dementia. Hepatic Complications. Death.

A private, aged 30, a sober, steady man, had sunstroke at Moulmein in October 1857, and was in hospital till August 1858, when he returned to duty, apparently well. In January 1859 he was again sent to hospital for strangeness of manner, etc., and was thought to be insane. He was sent to the asylum at Madras, and subsequently, in June 1860, to an asylum in England, suffering from chronic mania. The symptoms continued. He died in April 1861. His conditions towards the end were complicated by hepatic disease. At post-mortem examination of the head the calvaria was found to be dense, thick, and heavy, the diploë being obliterated. Dura mater thick, with opacity of the pia mater. Brain substance appeared firm, grey matter, varying from a quarter to half an inch in depth. Ventricles contained a small amount of fluid, and a large number of cysts were on the choroid plexus; the convolutions seemed natural.

The notes of this case were furnished by the late Dr. Christie, C.I.E., of the Royal India Asylum, Ealing.

No. 12.—Case of Sunstroke. Frequent Recurrence of Symptoms of Insolation. Death. Tuberculous Complications.

A gunner, aged 31 years, as far as was known a sober and steady man, was admitted to hospital in India in July 1849, suffering from sunstroke; he recovered and left, but soon returned, having been found wandering about and talking incoherently. He was readmitted several times, and finally sent to an asylum in India in March 1851, and thence to one in England in 1852, in a state of dementia with general incoherence. He died in June 1856.

Post-mortem examination showed the calvaria very adherent to the membranes, the plates of the skull of unusual thickness with the diploë obliterated; the arachnoid contained about 2 oz. of serous fluid; pia mater opaque and thick. The substance of the brain was firm, but appeared healthy. The brain was small. Lungs studded with tubercle.

No. 13.—Case of Sunstroke ending fatally in Eleven Years from Intracranial Changes.

An officer, aged 43, had sunstroke from direct exposure to the sun in 1868 in India. This was followed by headache on the left side, difficulty

of articulation, and other signs of cerebral disturbance. Also singing in the left ear, with defect of vision in the left eye. The memory for recent events defective; conduct very eccentric; exaltation of ideas, great excitement and loss of self-control were followed by profound depression. He was placed under treatment, but the symptoms increasing, he was sent to Europe and to an asylum in August 1870. He was then suffering from general paresis. His ideas were exalted and bombastic, his expression perplexed and unhappy, tongue tremulous and quickly receding, gait unsteady and tripping.

These symptoms continued till August 1872; he then improved to such an extent as to permit of his residing at home for two years, during which he lived a careful, temperate life, and took a fair amount of exercise.

In November 1874 he was readmitted to the asylum suffering from symptoms of general paresis. Speech indistinct, delusion that he was a person of great importance in India; pulse small and quick; pupils strongly contracted; temperature normal. Treated with physostigma. These symptoms gradually increased. In December 1878 he became unable to leave his bed; contraction of the lower extremities set in, and they were rigidly flexed towards the chin. He died in January 1879. Took nourishment freely until within a few hours of his death.

Head examined after death. Body much emaciated; calvaria dense, diploë obliterated; membranes very vascular, and thickened and adherent along the median fissure. This was found to be due to three or four bony plates of the size of sixpence, with small spiculæ passing into the surface of the brain on the left side. The brain weighed only 44 oz.; grey matter deficient, convolutions flattened. Nothing unusual appeared in the brain substance beyond slight softening of the right thalamus opticus.

The notes of this case and the preceding one were supplied by Dr Christie, C.I.E., of the Royal India Asylum.

CHAPTER XX.

GOITRE.

BY FRANCIS NOTTIDGE MACNAMARA, M.D.

Prevalence and Character.—Goitre is very prevalent in its endemic form in many parts of Northern India; in Central and Southern India it is rarely met with. This is a circumstance in the history of the disease which is well illustrated by the returns of the number of cases annually treated amongst the European and native troops. Thus, taking the sum of the three years 1888–90, the number of Europeans treated was, in Bengal (including Northern India) 20, in Bombay none, in Madras 2; the number of natives treated was in the same territorial divisions respectively 81, 1, 2. But these figures do not at all express the prevalency of the disease amongst the native population of Northern India, for the numbers treated there annually, at the various dispensaries, might be reckoned in tens of thousands. In many of the districts, montane and sub-montane, the disease is so common as to be looked upon as one of the ordinary conditions of life, and is not complained of unless it produces urgent symptoms, or, in the case of a woman, hideous deformity.

The tumour does not in its anatomical character differ from that of endemic goitre as met with in other countries. It begins as a simple hypertrophy of all the tissues of the thyroid gland, the vesicles and the colloid substance being chiefly affected; and such the tumour, though of large size, may remain. Or, on the other hand, the vesicles may enlarge into cysts of varying size, or in the progress of the disease the stroma may be chiefly affected and the tumour become fibroid.

The tumour may commence as a slight swelling of a portion of the gland, generally the right lobe, implicating subsequently the whole of the gland; or it may from the beginning implicate equally the whole of the thyroid.

The disease is undoubtedly more frequent amongst women than

men, and young men are more susceptible than children or the middle-aged. The tumour may progressively increase in size, or, as is perhaps more common, the augmentation in size may be very manifest in the early part of the rainy season, and less so at other seasons.

Goitre is distinctly not an hereditary disease,—a family has only to remove from an affected district to insure freedom from it; and, on the other hand, a family, however healthy, moving into such a district may sooner or later become affected in the persons of some of its members. On the question of heredity, however, we shall have more to say when discussing the connection of goitre and cretinism, and the etiology of both diseases; but this question we shall defer till we have considered the distribution of goitre in Northern India, and the conditions of soil and climate under which it prevails.

Distribution in India, and the Conditions under which it occurs.—Probably the principal interest of endemic goitre in India rests in the consideration of its geographical distribution, for it is that which gives us our best hope of disentangling from many conditions the cause of the disease.

Let it be remembered that goitre is found endemic in India in very varying climatic regions; we find it prevalent amongst the steaming mud-flats of Upper Assam, but we find it so also 1400 miles to the west, in patches amongst the arid plains of the Punjab.

Nor is goitre confined in India, as has been sometimes stated, to the montane or sub-montane regions; this is not the case. It is found endemic, far away from the hills, on the east in the delta of the Ganges, and on the west in the district of Moultan.

Perhaps the view which is still most generally held in India of the cause of the disease, is that which had its origin in the writings of the late Dr. M'Clelland, though the teachings of these have been mistaken, for he did not teach that goitre was caused by the habitual use of hard water, but that it depended upon residence on a calcareous soil, water being probably the medium by means of which the specific cause of the disease, possibly lime, is conveyed to the patient's system.

Dr. M'Clelland derived support for his view from the observation of the distribution of goitre in two neighbouring districts of the Himalayan province of Kumaon. He investigated the cases of many villages and groups of villages in these two districts, one of which possessed an eminently calcareous soil resting upon limestone rocks, while the soil of the other was non-calcareous, and the rocks argillaceous or silicious. In the calcareous district, about one-eighth

of the people were afflicted with goitre, and cretins were many in number; while in the non-calcareous region, only one in 500 of the inhabitants were affected, and there were no cretins. Dr. M'Clelland's views were strengthened by what he learnt of the prevalence of goitre upon the calcareous *bhat* soil of certain portions of the Goruckpore and other districts situated between the Gogra-Ganges, and the Himalayas.

But let us commence the discussion, which shall be as brief as possible, of Dr. M'Clelland's theory, with a reference to an observation which he himself made regarding the relative prevalence of endemic diseases generally in the Kumaon districts in question, taking as the instrument for comparison the health of two detachments, each about 400 strong, of men, women, and children at two stations, one Lohoogat in the non-calcareous, the other Petoraghur in the calcareous district. Dr. M'Clelland's table is as follows:—

	Lohoogat (non-calcareous).	Petoraghur (calcareous).
Diarrhœa,	32	5
Dysentery,	7	88
Fevers,	167	232
Enlarged Spleen,	1	2
Goitre,	...	15
Other Diseases,	74	144
Total,	281	486

Now, if this table is made use of in order to show the influence of residence on a calcareous soil in the production of goitre, it must also be taken further to mean that such a soil is productive of fever and dysentery. What the table does unequivocally show is, that goitre prevailed in the more unhealthy of the two stations; and this is a consideration which might be verified by many such comparisons, as, for instance, by the distribution of goitre in the three districts of Lahoul, Spiti, and Kooloo, lying together in the mountain ranges to the north of Kumaon. In Kooloo the average elevation of the inhabited portions is about 5000 feet, the hillsides are covered with forests, and in the valleys extensive irrigation enables the villagers to grow rice, which is their principal crop; wheat, barley, maize, poppy, and tobacco are also grown. The inhabitants are a hardy people of mixed Hindoo-Mongolian race; yet a great deal of sickness prevails amongst them, intermittent fevers and bowel complaints,

[1] *Settlement Report of* 1876. By J. B. Lyall, Esq.

goitre and scrofula. The men of the upper villages consider themselves safe only so long as they can stay there, and cannot be induced to go down into the lower valleys at certain seasons of the year.

Lahoul has a higher elevation than Kooloo, that of the inhabited parts averaging 10,000 feet; the hillsides are bare; the crops are wheat, barley, and millets. The climate, writes Captain Harcourt,[1] is singularly dry and bracing; there is very little sickness; and goitre, if present, is very rare. Spiti is even more elevated than Lahoul; its cultivation and climate are the same, and the people are a singularly healthy race.

The late Dr. Stoliczka, a most observant traveller, has, too, given similar evidence regarding the distribution of goitre in these regions. He contrasts the prevalence of the disease in Bussahir, in the valley of the Sutlej, with the absence of the disease in Spiti, and assumes that the prevalence of the disease in the valley depends upon the presence there of metamorphic micaceous rocks, and that its absence in Spiti is due to the calcareous formation of the valley. But indeed both these views, those of Dr. M'Clelland and of Dr. Stoliczka, as regards the influence of certain soils on the development of goitre, are too narrow; for throughout the regions of endemic goitre in India we shall find the teaching of the Petoraghur table true, that, independently of any peculiarity of soil, goitré prevalence is associated with "greater intensity of endemic diseases generally."

Let us pass to consider the prevalence of goitre amongst the inhabitants of the calcareous *bhat* soils of Goruckpore. The soil in this district is of two principal sorts, the *bangar* of a dry silicious nature, with dry, sandy deposits deeply covering the damp clay bed beneath, and the *bhat* soil, which is very generally calcareous; it is a soil retentive of moisture, and this because the damp clay bed is but thinly covered, and it is "on these calcareous damp lands that goitre prevails, idiots are common, and the people generally are feeble in mind and body." But, adds Dr. M'Clelland, "damp soils in other parts of the plains do not produce that derangement of the glandular and osseous system on which goitre and cretinism depend, because these maladies are unknown in the damp clayey soils of Bengal, Assam, Pegu, and other parts of India; while they are prevalent in certain dry rocky soils in Kumaon, where the waters employed by the inhabitants are derived from beds chemically identical with the calcareous clay bed composing the *bhat* lands of Goruckpore." But the very comparison which is made in the above

[1] *Kooloo, Lahoul, and Spiti.* By Captain Harcourt, late Deputy-Commissioner of the district. London, 1871.

quotation is just the one which, with the help of an extended knowledge of the distribution of the disease in India, completely upsets Dr. M'Clelland's theory, for amongst the regions of India in which goitre is most prevalent is that of Upper Assam, the valley of the Brahmapootra, where the soil is eminently non-calcareous. "The Brahmapootra and its tributaries," writes Dr. M'Clelland, "with a large proportion of iron sand, contain very little calcareous matter; and from this negative evidence it was long since inferred that no great development of limestone rocks was to be expected in the adjacent portions of the Himalaya range." Lower down in its course the Brahmapootra, and its main continuation the Jamoona, bound and water the Mymensingh district of Bengal, in which half the population, says the civil surgeon, suffer from goitre. He does indeed venture an opinion that the disease may be due to the earthy salts present in the Jamoona water; but, as a matter of fact, analysis shows that the water is a soft one,—a fact which is well known to voyagers on the river, especially if they have recently used the comparatively hard water of the Ganges. Analysis made of the Brahmapootra water at Gowalpara in March 1873, before the commencement of the rains, showed that the water contained in solution only ·104 per 1000 of total solids, that its total hardness was 3°·8, and its permanent hardness 1°·8.

We shall return to Mymensingh presently, but just now we have to consider the circumstances of goitre in the valley of the Brahmapootra, the valley of Assam, which opens westwards upon the plains of Bengal. The climate of the valley is throughout hot and damp, while a great extent of the surface is unreclaimed, and covered with jungle and forest, jheels and marshes. The conditions which combine to generate malaria are everywhere present, and, as might be expected, malarious diseases, fever and spleen, and dysentery and malarial cachexia are emphatically the diseases of the valley. Goitre is prevalent almost throughout the valley, and is not more prevalent, or indeed so prevalent, along the base of the hills as on the banks of the central river and its affluents. Thus, taking the case of the central district, that of Nowgong, malarious fever and its sequelæ are the most common diseases of Nowgong, and ill health manifested in various forms, due to the system being saturated with malarious poison. Chronic enlargement of the spleen is very common. Bronchocele is very prevalent; cases of the disease form about one-third of those treated at the dispensaries of the district. The civil medical officers state that the disease is most common in the vicinity of marshes and along the banks of

the rivers. Natives and strangers are alike affected by it, but the disease is by no means always associated with other signs of malarious cachexia, and in fact many of the patients appear to be in good health. There is no report of cretinism in the district. The circumstances of the disease are the same in the neighbouring district of Seebsangor; there also it is most common in the low tracts along the banks of rivers, and in the neighbourhood of marshes; but the disease is often very much localised, and in spots which do not apparently differ from others in the neighbourhood.

Three notices which we have of the disease in Upper Assam are very instructive. The first is that of Poobha Mookh, an outpost for forty sepoys, on the north bank of the Brahmapootra, a clearance in the forest of some 40 acres, open only to the river. The water supply is from the river, a good, soft, drinking water. The post is a healthy one, and would be, reports a medical officer, one of the healthiest in Assam, were it not that a residence there of three or four months insures a well-marked goitre. The other history is that of a remarkable outbreak of goitre which occurred in the same district amongst the sepoys of the Duffla field force in the early part of 1874, when the troops entered the district bordering on the Dikrang river. The encamping ground was a narrow strip of land by the side of the river, but elsewhere surrounded by the jungle; the ground was damp everywhere, swampy in places, the soil the débris of the vegetation of ages. The river was a fine clear stream running swiftly in its boulder bed; the water contained a little over 5 grains per gallon of solid matter. The men of the force suffered from bowel complaints, but still more notably from goitre—nearly every man in the ranks suffered from more or less fulness of the thyroid gland; so that here, as at Poobha Mookh, the men suffered when moved from cantonments into a district notoriously malarious, one in which goitre is almost universal amongst the inhabitants. If instead of entering the district at the most healthy time of the year, they had done so later on, doubtless the sad story of a similar expedition—the Bhootan Campaign of 1864–65—would have been repeated; then the force was decimated by fever and dysentery, but as it was, the Duffla expedition suffered chiefly from malarious diseases of a milder form.

Another outpost in Upper Assam is that of Suddya, on the north bank of the river, well placed on a high sandy bank, well drained, and having an excellent water supply; yet here too a residence of three months is quite sufficient to develop a goitre in any and all of the younger sepoys who are sent thence on detachment duty

from Dibrooghur, the headquarters station of the district. The older men, writes Dr. Whitwell, are seldom if ever affected.

But though Suddya is thus favourably placed, and, like Poobha Mookh, is comparatively healthy, both are in the midst of an unhealthy goitrous region. A late Deputy-Commissioner writes as follows regarding it: "Goitre is so common throughout the district, that it is difficult to say where it is most so. On the banks of the Brahmapootra it is very prevalent, quite irrespective of the nature of the ground on which the houses of the people happen to be erected. In many villages not a single individual above the age of 10 or 12 is free from the disease. Perhaps, if any localities can be found in the district which are worse than others, they are the high banks of the largest rivers. Together with goitre, malarious diseases, fever, spleen, etc., are very prevalent. The whole district is a flat alluvial plain, intersected by numerous streams, some of which are perennial, while others degenerate for a portion of the year into a string of stagnant pools. The whole country, excepting the high banks of the larger rivers, is inundated during the rains, and at other seasons it is a series of jheels, swamps, and bogs alternating with stretches of impenetrable jungle. The soil is alluvial loam, resting on a subsoil which is for the most part sandy, but here and there clayey."

Did space permit, it would be instructive to dwell at length upon the conditions under which the disease is endemic in other districts of the Assam valley. We shall notice only one other locality, and that in Durrung, a district of Central Assam on the north bank of the river. The report comes from a Mr. J. D. Campbell, long resident as a tea-planter in the district. Mr. Campbell states that the disease is very prevalent along certain streams, and he notices its special prevalence at Arrung, a low-lying, steamy, densely-packed village on the margin of a swamp; and he argues from the nature of the place, and from the association there of goitre with fever and spleen, that those diseases must have a common malarious origin.

Not many miles beyond the western frontier of Assam is the military station of Buxa, situated at an elevation of about 1800 feet, in a valley between two ranges of hills. The plateau on which the station stands has resulted from the accumulation of the débris, boulders, and gravel from the surrounding hills. The spur which forms the eastern boundary of the valley is gneissic, while the western spur is mainly a micaceous sandstone. The climate is mild and very damp, and the station is so little elevated above the expanse of Terai at the foot of the hills, that were it not for the strong breeze which each night sets down the hill, lasting till sunrise, it would in all probability be uninhabitable. As it is the troops—

native—who are stationed there suffer not a little from the malaria, fevers, diarrhœa, and dysentery, and a large proportion of the men, after about one year's residence, suffer from goitre. The disease is very prevalent also amongst the Bhooteahs in the neighbourhood. The water supply of the station is from springs, and is of excellent quality, containing about ·06 per 1000 of total solids, of which about one-third is lime. There can therefore be no question that in the case of Buxa the poison, whatever it may be, which causes the complaint is not amongst the mineral constituents of the water which the patients drink.

Passing westwards from Assam, we reach the plains of Bengal, and here we find goitre endemic through the great stretch of country which intervenes between the Ganges, with its affluent the Gogra, and the base of the Himalayas; and we find the disease extending along the eastern bank of the Ganges, or rather its deltaic continuation the Podma river, almost so far as its estuary. Moreover, to the west of the Gogra we may discover the disease in the plains amongst some of the villages of the provinces of Oudh and of Rohilkhund. The climate of the country eastwards from the Gogra is pre-eminently hot and damp, while a large portion of the surface is thickly set with jheels, swamps, and marshy land formed in the deserted beds of rivers, or left on the subsidence of the annual inundation.

Dacca, Tipperah, and Mymensingh are three of the south-eastern districts of Bengal, and in all of these goitre is endemic; but it is perhaps more especially prevalent in the first of these localities. The district of Dacca reaches in a southerly direction to within about 80 miles of the sea, and, placed as it is in the centre of the delta of the Ganges and Brahmapootra, has a surface intersected by branches or loops of those great rivers which are constantly altering the position of their beds, while a large proportion of it is inundated during three or four months of the year, and is left, as the inundations subside, a steaming, offensive swamp. Here is the home of cholera, and here malarious diseases are universally and perennially rife. Next to intermittent and remittent fevers, the most prevalent endemic diseases are elephantiasis and bronchocele. Out of 106 villages, goitre was found in 72 villages belonging to the northern division, and in 27 in the southern division of Dacca. The disease is most common in situations where the inhabitants make use of morass water; and, writes a former civil surgeon of the district, Dr. Adam Taylor, "judging from the nature of the localities in which the disease (goitre) is most frequently met with, it would seem to be rather the effect of malaria than of any cause such as

lime in the water." Such, too, is the opinion of another civil surgeon, Dr. Wise, who has written upon the subject.

To the north of Dacca, between it and the Garrow Hills, lies the district of Mymensingh, another watery district, its surface abounding in marshes and jheels, and with a population enfeebled by malaria, and suffering, like their neighbours in Dacca, from leprosy, elephantiasis, and goitre. The latter is specially prevalent amongst the inhabitants of the churs of the Brahmapootra and of the tract of country which lies along the base of the Garrow Hills.

On the west bank of the Brahmapootra, just after it has turned south on its way to join the Ganges, is the very unhealthy district of Rungpore, with a most instructive history of endemic diseases. One civil surgeon, Dr. Ghose, states that he found 80 per cent. of the people manifestly suffering from malaria; and in connection with the malaria of the district he mentions the prevalency of goitre in many villages. Dr. Bowser, another civil surgeon of the district, writes of the very great mortality from fever, and speaks of "its complications, spleen and bronchocele."

Space will not allow of our following, even briefly, the conditions under which the disease shows itself in the remaining districts of Bengal north of the Ganges, but we may confidently assert that throughout this great stretch of country the most swampy portions of the districts, and the low tracts of land along the course of the rivers, are those in which goitre is discovered, and in which it prevails in its most aggravated form.

Dr. Coates, a former civil surgeon of the district of Chumparun, —one in which the disease is very prevalent,—collected some statistics which he thinks show that both goitre and cretinism are acquired rather than inherited. To healthy parents were born 25 cretins, 35 goitred, 38 healthy children; to goitred parents, 67 cretins, none goitred, 202 healthy children; to cretin parents, 26 cretins, 41 goitred, 51 healthy children. 3452 goitres had 6493 healthy brothers and sisters, all born of unaffected parents; while 1197 goitres had 2714 healthy brothers and sisters born of goitred parents.

Dr. Cameron, another civil surgeon of the district, states that he had made particular inquiries as to the connection of goitre with the semi-idiocy so common amongst the people, and his conclusion was that one condition does not depend on the other. The natives distinguish between the diseases, and believe that goitre is not an hereditary disease; while, on the other hand, they believe that cretinism is hereditary, and, moreover, that its prevalence is much increased by marriage amongst blood relations.

Dr. Coates learned that the water of certain wells was believed to cure goitre, and these he found were deep, well-built, and carefully-kept wells, which had been dug by indigo planters, the water of which, though hard, was much freer from organic impurities than the shallow, ill-kept, village wells, often supplied by the drainage of Terai lands.

Dr. Coates agrees with the view of an intelligent native gentleman, who told him that he remembered the time when the whole locality where he lived was jungle; but now, he said, "everybody cultivates; there is no jungle water to drink, and goitres and fevers and spleens are much fewer than formerly." The district, too, serves to illustrate what has just now been advanced as to the localities in which goitre mostly prevails. Dr. Cullen, a former civil surgeon of Chumparun, reports that the disease is most prevalent in the north, along the Nepaul Terai; in the western tracts, where the country is very low and very extensively inundated, and along the banks of the rivers Secrana and Dunnowtee, which are low, covered with dense vegetation, and connected with many lakes and marshes. Dr. Cullen calls this "the goitrous area of the district." Two-thirds of the population are affected; "and it is to be noted," he writes, "that while malarious diseases prevail throughout the district, they do so with greatest violence in this area."

Westwards from the Gogra there is a marked change in the climate of the plains; it is a hotter and a drier climate, the rivers are less numerous, the rainfall is less, the hot winds blow steadily for a portion of the year, and jheels and marsh, though not unknown, are far less frequently met with than eastward of the Gogra, and in correspondence with this change of climate endemic goitre, outside the Terai lands, becomes a less prevalent disease.

Amongst the hills to the north of Oudh is the kingdom of Nepaul, in one of the valleys of which is Khatmandoo, the capital. The valley is distant in a direct line some 30 miles from the foot of the hills, lying amongst the Himalayas proper, at an elevation of about 4500 feet above sea level. The valley is about 20 miles in length by 16 in breadth, and is densely populated. The alluvial soil, fully watered by rainfall and by plentiful irrigation, yields abundant crops of rice and wheat. The climate, a subtropical one, is delightful, resembling that of the most favoured parts of Southern Europe, and has its hot, rainy, and cold seasons. But though thus far naturally favoured, the valley is not a healthy one; malarious fevers are very rife in the hot and rainy seasons, typhoid fever is but too common, and cholera and smallpox are at times very fatal. Leprosy also is commonly met with; but the

disease which is most strikingly prevalent amongst the people is goitre. In many of the villages in and around the valley as many as 30 and 40 per cent. of the people are affected; men, women, and children are alike so, and foreigners coming to reside in the valley speedily become goitred. Mr. Bramley, formerly surgeon at the Residency, in the full account which he has given of goitre in Nepaul, states his opinion that the disease is not hereditary, and that it suffices for a family to remove to a non-goitrous locality in order to insure the removal of their goitres. Mr. Bramley states that the people who work in the fields are far more liable to the disease than the artisans of the towns. Cretinism, Mr. Bramley states, is unknown in Nepaul, and he notices that the subjects of bronchocele are not remarkable for weakened or impaired intellect. It appears, however, from the result of inquiries made since Mr. Bramley wrote, that cases of cretinism, though few in number, can be found in Nepaul. The natives attribute the prevalency of the disease to something in the water; but the water, so Mr. Bramley reports, is to appearance and taste of the purest kind, and contains a very small proportion of mineral matter.

To the west of Nepaul are the mountainous districts of Kumaon and Gurhwal, of which the former was the scene of Dr. M'Clelland's explorations, and here are several hill stations, civil and military. One of the latter class is Raneekhet, in a direct line about 35 miles from the plains, on a plateau, elevated about 6000 feet above sea level, the soil of which is composed of the disintegrated mica schist of the subjacent rocks. The water is of excellent quality, soft, and containing 5 or 6 grains to the gallon of solid matter. Goitre is common in the neighbouring villages, and shows itself amongst the European troops stationed at Raneekhet. Surgeon-Major Gore records a small epidemic of the disease which occurred amongst the men in the year 1881, when thirteen cases of acute goitre were treated about the same time. Mr. Gore could discover no common factor other than a certain length of residence in the hills, and in a goitrous locality. He observes on the purity of the drinking water.[1]

To the west of Gurhwal, and separated from it by the Ganges, is the valley of Dehra,—the Dehra Doon,—extending thence to the eastern bank of the Jumna river. The most beautiful and most luxuriant of the valleys of the sub-Himalayas, it is yet in many parts notoriously unhealthy; fever and spleen are very prevalent amongst the inhabitants, as is also goitre. Dr. M'Laren, a civil surgeon of the district, draws attention to the circumstance that

[1] *Indian Medical Gazette*, December 1884.

goitred prisoners coming into jail get rid of the goitre after about six months, and this without treatment, and simply under the influence of the healthy regimen of the jail. This result is the more remarkable because the water in use in the jail is very hard, and retains, after careful boiling, nine degrees of permanent hardness.

Passing westwards we reach the very unhealthy Kangra valley, where "miasmatic fever is universal, and the valley is yearly ravaged by dreadful visitations of intermittent fever. Goitre and cretinism are very common, and are to be met with in numbers throughout the valley; scarcely an individual in the valley over 20 years of age is free from goitre, but enlargement of the spleen is comparatively rare." Dr. Dickson notices "that out of 5300 cases of fever, only 26 suffered from spleen; while in the malarious villages along the western Jumna Canal, Dr. Adam Taylor found 30 or 40, often 60 or 70 per cent. of the people suffering from enlarged spleen. Dr. Dickson writes: "The peculiar small limbed, prematurely aged, coarse featured, dwarfish individuals to be met with on every side strike a new-comer painfully."

We have yet to consider the circumstances under which goitre presents itself in the plains of the Punjab, the great plain which extends from the Jumna on the east to Afghanistan on the west, and from the Himalayas on the north to the Great Desert of Rajpootana on the south. The plain is watered by the Indus and its five great affluents, which debouch in a direction at right angles to the mountain chain, and receive in their course scarce any tributaries, for the minor streams which collect the comparatively scanty drainage of the outer Himalayas, with two or three exceptions, run but a short course before they are absorbed by the sandy plains. Moreover, the rainfall of these plains is far less than is that of the Ganges plains; and another factor in the condition of the surface is the more rapid fall in its level, causing the rivers to deposit, not the finer silt which constitutes the mass of the plains of Bengal, but a fine sand which produces a very dry and thirsty soil, and one which is far extended by the influence of the winds, by changes in the channels of the rivers, and by the inundations. And so it results that, outside the sub-montane tract, cultivation is almost limited to the belts of land along the course of the rivers, and to localities which enjoy canal irrigation. Where cultivation is limited, it is so, not as in the eastern plains, owing to an excess of water in the form of jheels and marshes, but rather because of a deficiency of that element.

Now in the Punjab we find a very notable plainward development of goitre, but only—and this is very worthy of attention—

where natural, or still more frequently artificial, conditions have generated a swamp—a typical place of this kind, not far distant from the western border of Rohilkhund, presents itself in a subdivision—that of Munee Majra—of the Umballa district of the Punjab. The district itself, some 80 miles in length and breadth, extends from the hills along the western bank of the Jumna river. The surface of the northern part is undulatory, and is broken by the wide beds of many streams which carry off the drainage of the outer hills; but for the greater part of the year the streams have but a short course in the plains, the water being absorbed by the sands, or led away for irrigation. One only, the Ghuggur, is perennial, and struggles on beyond the southern boundary of the district. Goitre is not a disease of the district generally. Amongst 135,000 patients treated in the dispensaries of the towns of Umballa and Thanesur only one case of goitre is recorded. But the disease is prevalent in certain localities; thus it is common in the north-east corner of the district near the head of the Western Jumna Canal, and has of late years become prevalent lower down the course of the canal, in the subdivision of Jugadhri. But it is in the subdivision of Munee Majra, at the foot of the hills, about 20 miles due north of the city of Umballa, that the most marked and instructive development of the disease occurs. "The subdivision contains sixty-nine villages, and is irrigated by the Ghuggur river, which meets the villages immediately on its exit from the hills. The land looks very much like an ancient alluvial deposit from the river. The characteristic of the locality is the lowness of the land, so that all the villages can be reached by streams carried along ducts called "Kools" from the Ghuggur, and the supply from the river is perpetual. The villages are frightfully underpopulated. Fever is extensively prevalent, and every third person has a distended spleen. Goitre also is very prevalent, and cretinism is very common. The people have no heart and no strength."[1] The superintending engineer of the circle states his opinion that the sickness is due to the abuse of irrigation. "The soil has become waterlogged, owing to a considerable body of water, beyond that needed for food irrigation, being diverted from the river. The water of the river was analysed by Dr. Shepherd, and was pronounced excellent; it contained 15 grs. per gallon of solid matter. The Sanitary Commissioner of the Punjab reported on the subdivision in the year 1871, and his notices of some of the villages which he visited may be epitomised as follows:—

Munee Majra, 6045 inhabitants; lands about famous for rice;

[1] *Settlement Report.*

irrigation by kools from the Ghuggur. Fever and spleen rife in the rainy season; 6 per cent. of the people with goitre. The people say that when, as was the case for some years, irrigation from the Ghuggur was stopped, the decrease of goitre was notorious; when the water came again to the town lands the disease increased. Bara Ferozepore, 3 miles south-east of Munee Majra, 247 inhabitants. Village on the flat, irrigated by kools, from which the people take their water. The miseries and woes of the people are heartrending; no one who can help it stays in the village; the children almost all die young, or grow up deaf and dumb and daft. "In the rainy season everybody's belly swells with spleen, and men and women and children are disfigured with goitre." Chundee, a scattered place of clumps of huts, some on the hills, some on the plains below. The people on the hill take water from a tank, those below from the kools; no goitre, and only one spleen above. Below, both goitre and spleen prevail.

We meet with a precisely similar association of diseases, goitre, spleen, and fever, in a valley amongst the hills a little to the north of Munee Majra. This valley is irrigated in part by kools from the Ghuggur, and at higher levels by the water of mountain springs. But disease is limited to the people inhabiting the villages in the low ground, which is irrigated from the river.

About 120 miles west of the city of Umballa, and about the same distance from the foot of the hills, is the ancient town of Dipalpore, in the centre of the "doab" between the rivers Sutlej and Ravee. The town is situated on the bank of the old bed of the Beas river, and is watered by two canals which run close to the town. The soil is a hard black clay, well suited for rice, large crops of which are grown, with the help of canal irrigation, around the town. Unlike the rest of the district, Dipalpore is notoriously unhealthy. The inhabitants are chiefly agriculturists, and attribute their bad health to the water they drink and to the surrounding rice cultivation; they are almost universally goitred, while cretins and dwarfs are numerous. More women than men are goitred; many children after their fourth year become affected. Strangers are frequently attacked after a year's residence in the town. Animals, especially dogs and goats, commonly suffer. The people do not consider the disease hereditary, but affirm that a weak habit of body and a poor diet dispose to it.

The history of goitre along the course of the Chenaub river is also a very instructive one. The disease is very common in the sub-montane irrigated tract between this river and its affluent the Tavi, but it does not again prevail till some 90 miles lower

down the course of the river, where we reach the town of Midh, situated in the low-lying ground within the limits of the autumn floods. The land about the town is very fertile and highly cultivated. Goitre is very common there, and in the neighbouring villages within 5 miles of the river. Two or three cretins are known in the town, and there may be others whose existence is concealed. The Mohammedans, says the native officer who reports, suffer less than the Hindoos, because their diet is more nourishing. Thirty miles lower down the river we again find a local development of goitre in the town of Chiniot, on the opposite bank to Midh; but, as in the case of Midh, situated within the low tract inundated by the river Chenaub. Assistant-Surgeon Jai Sing, in a very interesting contribution which he made in March 1886 to the *Indian Medical Gazette*, states that the disease is most prevalent in the lowest lying and dampest part of the inundated tract. The people on the higher level suffer if they are obliged to work in the affected tract or drink the water thence. But the disease is unknown amongst the boatmen of the Chenaub. He does not consider goitre or cretinism hereditary, though children are sometimes born cretins—poverty, bad food, excessive rainfall, residence near stagnant pools, and insanitary conditions generally, predispose to both diseases. The swelling, he states, first appears, and then the patients become pale and anæmic and assume the appearance of malaria-stricken people; then their intellect fails, and they become imbeciles. He believes that the cretinism as it shows itself at Chiniot, and at a place 17 miles lower down the river where it is very common, is caused by an intense degree of the poison having been imbibed. He notices that some cretins and imbeciles show no enlargement of the thyroid.

Some 120 miles below Chiniot we reach the city of Mooltan, situated upon the east bank of the Chenaub, in the district to which the city gives its name. The district occupies the angle formed by the confluence of the rivers Chenaub and Sutlej, and is low lying, for the high ground which occupies the centre of the "doab" has disappeared, and the "khadir," that is, the low land along the course of the rivers, here extends across the district from river to river. The district is almost rainless, the fall averaging yearly about 7 inches; but the whole of the land is abundantly watered by the inundations, and by a network of canals. The soil is a stiff tenacious clay, here and there mixed with sand, extending to a depth of 6 or 8 feet, beneath which is a subsoil of almost pure sand. Water is found at a depth of about 25 feet, and the level throughout the year varies but little. The city is surrounded by groves and

gardens, vegetation flourishing luxuriantly along the numerous cuts which lead the water from the neighbouring river. The city is an unhealthy one, the people suffering greatly from malarious diseases, fever, and spleen. Goitre is very prevalent in the immediate neighbourhood; of 35,471 cases treated at the city dispensary during five years, 1630 were for goitre. But as indicative of the patchy distribution of the disease, we have the circumstance that goitre is uncommon at Shoojabad, a town 26 miles south of Mooltan, in land irrigated from the Chenaub, and at Kuhrar, 50 miles south-east of the city, in a locality which is irrigated from the Sutlej. Goitre is met with in some of the villages along the bank of the river opposite to Mooltan, but is nowhere so common as in the neighbourhood of the city.

Far away on the frontier, to the north of Mooltan, is the town of Tank, in the district of Dera Ismael Khan. The town is in a valley which is freely watered by streams from the hills; and here plentiful irrigation has turned a barren plain or camel pasture into a fertile, highly cultivated tract. Many cases of goitre are to be met with in the town, and in a marshy valley in the neighbourhood; elsewhere in the district the disease is scarcely known.

Along the banks of the Jhelum also, we find local development of goitre, and especially at Pind Dadun Khan and neighbouring places along the base of the Salt Range of hills. Where, too, the Indus leaves the Salt Range and spreads its inundation over the low land or "cutchee," we again meet with goitre, especially in the towns of Maree and Kalabagh at its northern extremity.

In the preceding pages we have by no means exhausted the history of endemic goitre in Northern India; it has been our object to supply [1] illustrations typical of the localities in which the disease prevails, and we purposely refrain from comparing them with localities in other parts of the world in which goitre is endemic. It is our wish to present goitre as it appears in India. We shall refer, and that only very briefly, to one locality outside India proper—this is the valley of the Tarim, some 700 or 800 miles to the north of the valley of Nepaul, beyond the Himalayan mountains and the table-land of Thibet. The river in question ultimately becomes lost in a vast stretch of swamps and lagoons, known as Lake Lob. The population is massed along the banks of the river and its tributaries in places rendered fertile by irrigation, and here again goitre and malarious fevers are very prevalent.[2]

[1] For further illustrations and for authorities I must refer to my work, *Himalayan India its Climate and Diseases*, 1880.—F. N. M.

[2] Bellew's *Kashmir and Kashghar*, 1872.

Conclusions.—And now we think we may venture to submit certain conclusions regarding goitre in India.

In the endemic form the disease is confined to certain parts of Northern India, viz. the valleys of the Himalayan mountains, the belt of swampy land or Terai which extends along the base of the range as far westwards as the debouch of the Ganges, the valley of Assam, and the eastern portion of the Cachar valley, the plains of Bengal, Oudh, and the North-West Provinces northwards and eastwards of the Ganges, localities in the Punjab where, by natural or artificial means, a condition of soil resembling in its dampness that of the trans-Gogra-Gangetic districts of Bengal has been produced.

The goitre-producing[1] localities of the plains and hills have these conditions in common—a damp soil and a subtropical climate; and the disease is in a more or less aggravated form as these conditions are more or less marked and persistent. The mineral constitution of the soil may vary; it may be calcareous or non-calcareous, clayey or sandy; but it must be, for at any rate the greater portion of the year, damp; and it must probably be a soil charged with decaying vegetable matter.

Native races of all stocks, Aboriginal, Aryan, Mongolian, are equally liable to the disease; locality, not race, determines its incidence. But to this statement we make an exception, for it is found that the aboriginal people from Chota Nagpore, the "Dhanghurs" as they are commonly called, who are much valued by the tea-planters of Assam and Cachar, because of their resistance to malarious diseases, are seldom if ever affected with goitre, though thousands of them, men, women, and children, emigrate to, and are employed in, the most goitrous regions of Assam.

Over a large extent of the goitrous area of India there is no cretinism, and very many of those affected with goitre are otherwise in vigorous health. Yet one cannot but presume that in the case of a large number of the goitred, the gland has become altered in structure and functionally spoiled. Unfortunately native feeling, especially in the rural districts, is so intensely opposed to post-mortem examinations, that we have no certain evidence under this head; but often the appearance or feel of the tumour, and its resistance to the curative action of the iodide of mercury, evidence very strongly to degeneration or destruction of the normal structure of the gland. Nor can one by learning the percentage of the goitred in any

[1] Advisedly "*goitre-producing*," for the sufferers may be inhabitants of a village situated upon the dry hillside, but then they labour more or less constantly in the irrigated valley below.

district, predict with any certainty the presence or absence of cretinism there.

No evidence can be gathered from the circumstances of goitre in India that the disease causes cretinism; they point to the swelling of the thyroid as being simply a result, and not a constant one, of pathological changes which precede cretinism. Clearly something must be superadded to the conditions which cause goitre in order to develop cretinism.

Yet, that the cause of goitre and cretinism is a common one, or that it works in localities in which the conditions are common, appears to be demonstrated by the absence in India of cretinism in other than goitrous districts. Further, the fact that cretins are not uniformly the subjects of any thyroid peculiarity, appears to be well authenticated.

What we do find in India is, that where in addition to goitre, generation after generation of the people suffer acutely from malaria, becoming an anæmic, malaria-stricken race, there cretinism appears.

Briefly, we find goitre developing under three different sets of circumstances—(1) and of this we have an example in the case of the Duffla expedition. Native troops are moved at the healthiest season of the year into a goitrous, malarious region; they drink the water of a stream flowing swiftly over a boulder bed, the water containing but 5 grains per gallon of solid matter,—clearly the poison comes to the men through the medium of the air, and almost every individual is struck with goitre. In the case of troops which have been moved into such districts at an unhealthy season, the result has been strictly malarious diseases, fever, spleen, dysentery. Goitre in Assam seems to be a mild manifestation of malaria. (2) The disease, generation after generation, shows itself amongst the population of a fairly healthy district, commonly early in adult life, and the tumour increases year by year, sometimes progressively, more often with annual exacerbations at the malarious season; the patient's health may be otherwise good, and mind and body vigorous. The state of the thyroid is analogous to that not uncommonly found prevailing under similar circumstances in the functionally allied organ the spleen; only the latter having more important and a wider range of functions, structural degeneration in its case is more liable to cause ill health than does that of the thyroid. (3) Goitre shows itself amongst a malaria-stricken people, such as those of the Kangra valley, of parts of the trans-Gogra-Gangetic districts, in the Munee Majra district of Meerut, or at Dipalpore or Chiniot; successive generations of the people have resided and intermarried amongst

the same unhealthy conditions, and they become a malaria-stricken, goitrous, anæmic race, feeble in body and mind, semi-cretinous, and to them cretin children are born, or the children shortly after birth become such. And under these circumstances the disease is hereditary, which simple goitre is not. Remove a goitrous family to a healthy region, and goitre ceases from amongst them; but it is more than probable that a cretinous population transplanted to healthy surroundings, and intermarrying with a healthy race, would for many generations exhibit relics, in mind and body, of their former evil state.

But why should the goitrous area of India be thus confined, why does it not extend in Bengal south and west of the Ganges to places in which riparian swamps and rice cultivation are sufficiently common? We cannot answer the question,—we are unable to recognise the conditions under which special forms of malarious and other soil-born diseases can grow and flourish. But wheresoever we discover goitre endemic in India, we find it associated in locality and season with diseases of a malarious nature, arising under similar conditions, and amenable to like sanitary measures. Further, whatever may be the nature of the *materies morbi* which causes goitre, it differs specifically from that which gives rise to other malarious diseases. This appears to be evidenced by its limitation to certain provinces, and often, in them, to localities having a very circumscribed area.

Treatment.—In the early stage of the disease, removal to a healthy district, the use of a stimulating liniment, and, internally, iodine in some form, with or without quinine, as may seem desirable, will remove the goitre. And as we have noticed in the case of the Dehra Doon prisoners, removal from the affected atmosphere and a healthy regimen will, in many cases, effect a cure. But the favourite remedy for use amongst the native population of a goitrous district, is the inunction of an ointment of 12 grains of biniodide of mercury to 1 oz. of lard. The ointment must be rubbed in over the tumour for about ten minutes with a bone spatula, and the patient must then sit, so long as he can endure it, with the tumour exposed to the direct rays of the sun. If the ointment is applied shortly after sunrise, the patient will probably suffer severe pain from the blistering effect of the ointment about noon. About 2 P.M. the ointment is to be applied a second time, very tenderly, with a gentle hand, and then the patient is sent home with orders not to touch or remove the ointment. This treatment will ordinarily suffice; the patient is ordered, if it is not successful, to return after six or twelve months. Major Holmes,

who introduced this method of treatment in the Chumparun district in the year 1857, affirmed that the treatment never failed unless where the tumour was of stony hardness, and that he never heard of any dangerous results following upon the treatment. The treatment has not been so uniformly successful in other hands, but it is still very widely and very successfully employed.

Within the last few years, the removal of the tumour by operation has been practised pretty frequently in India, and with favourable results. Dr. Harold Browne has operated four times,—three times successfully. A full account of his cases, and of his mode of procedure, was published this year (1892) in the November number of the *Indian Medical Gazette*. Drs. Macleod and Raye have also operated successfully in the Medical College Hospital at Calcutta. But the operation has, in India, been most frequently performed by Surgeon-Major John Anderson, Civil Surgeon of Bareilly. He has operated twenty times, and obtained a perfect cure in sixteen cases; one of the other cases left the hospital while still under treatment, and three of the patients died. Dr. Anderson has lost sight of his earlier cases, and can speak positively only as regards the six cases on which he has operated at Bareilly. The first case left without permission while doing well; one case operated on in August 1890, cured; one in March 1891, cured; one in May 1891, died; one in March 1892, cured; one in April 1892, cured. All of these patients are now quite well, showing no symptoms of myxœdema,—a disease which, though looked for, is, so far, not known in India. Eight hundred and ninety-three cases of goitre have been under treatment at Bareilly during the years 1891 and 1892; none of them have been associated with either cretinism or myxœdema, "and this," writes Dr. Anderson, "is also the experience of my assistant-surgeon, who has attended personally to most of the cases."

Dr. Anderson continues:—

> I never operate on a case unless the patient asks for surgical interference, either for the relief of urgent symptoms or deformity; the latter cases are chiefly women. The cases most suitable for operation are those in which the limits of the tumour are well defined. I should not venture to interfere in some of the enormous goitres so frequently seen out here, where the growth is of great size, and extends right across the neck and under the sternum. In all cases I remove the entire gland, and have not noticed any bad effects from doing so. I prefer an oblique incision, because if the wound fails to heal by first intention, it drains so much better. I have never lost a case in which I adopted this measure. After exposing the capsule, I generally lay the knife aside and use my fingers. The vascular

surroundings of the tumour I tie all round with catgut ligatures, tying in two places and cutting between. The risks of hæmorrhage are generally much exaggerated, at all events during the operation. This, at least, is my experience, which is all I pretend to give. The wound should be kept strictly antiseptic, and an attempt made to secure union by first intention. If this fails, as it often does, owing chiefly to troublesome oozing of blood, I always open the wound right out, and leave it so for a few days. The great advantage of this is that all bleeding points can be easily secured. When bleeding has ceased, and the wound begins to granulate, the edges can easily be drawn together with strips of plaster. All attempts at stopping the bleeding by pressure, etc., have generally failed in my hands. The patient often develops a troublesome cough, which keeps it up.

CHAPTER XXI.

THE *FILARIÆ SANGUINIS HOMINIS* AND FILARIA DISEASE.

BY PATRICK MANSON, M.D., M.R.C.P., LL.D.

ALTHOUGH it has long been known that the blood of some of the lower animals, particularly of dogs and birds, frequently harbours the free embryos of certain nematoid worms, it was not until the year 1872 that it was shown that the blood of man is sometimes similarly infested. This important discovery was made by the late Dr. T. R. Lewis;[1] and to this distinguished observer belongs the honour of having thereby opened a wide and hitherto untrodden field for investigation, and of having supplied the key to many obscure though important problems in tropical pathology.

It has also been known for some years that the blood of the dog and certain birds may contain not one kind of embryo nematode only, but embryos of two or even of three distinct species; and that these various embryos may occur in different, or concur in the same individual hosts. The writer has shown that the same holds good with regard to the blood of man. In the *Lancet* of 3rd January 1891 he has described, as occurring in man, two species of embryo filarial hæmatozoa differing so much in habit and morphological characters from the parasite described by Lewis, and from each other, that it is certain they represent different and distinct species.

Nomenclature.—Seeing then that there are at least three species of hæmatozoa to which the term "*Filaria sanguinis hominis*" may be appropriately applied, it is of importance, before attempting their description, to provide each with a distinctive name.

As a basis for a suitable nomenclature I have pitched on certain individual peculiarities of habit appertaining to and quite characteristic of each of the three species.

It has been shown that as regards two of these hæmatozoa they are not always present in the blood, and that they observe a certain

[1] Eighth, Tenth, and Fourteenth Annual Reports of the Sanitary Commissioner with the Government of India.

periodicity in the times of their entrance and exit from the general circulation; whereas, as regards the third species, it observes no such periodicity. The names I have adopted have reference to this habit.

If slides of blood, obtained at intervals during the twenty-four hours from individuals known to harbour the filaria of Lewis, be examined with the microscope, it will be found that, as a rule, and under ordinary conditions of health and habit on the part of the host, it is only in those slides which have been prepared from blood drawn during the night that the parasite can be found. I propose therefore to call this filaria, that to which Lewis gave the name *Filaria sanguinis hominis*, and by which it has hitherto been generally

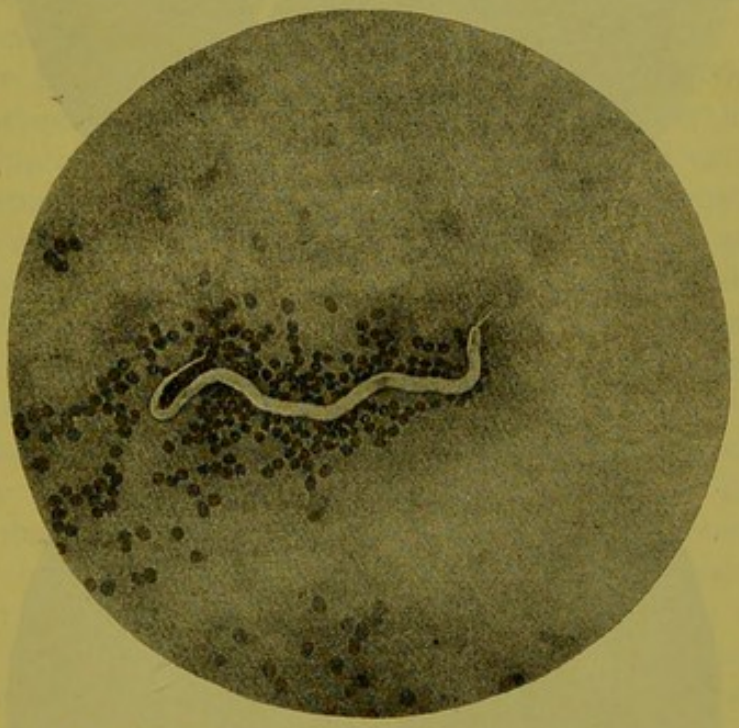


FIG. 51.—*Filaria sanguinis hominis diurna.* × 160.[1]

known, *Filaria sanguinis hominis nocturna* (Fig. 52), or, briefly, *F. nocturna.* If in the same way, and with the same proviso as to health and habit of host, we examine the blood of individuals known to harbour one of the species of blood worm I have recently described, and to which I have just alluded, we shall find that it is present in the blood only during the day. I therefore propose to call this species *Filaria sanguinis hominis diurna* (Fig. 51), or, briefly, *F. diurna.* If the blood of individuals harbouring the third species

[1] The micro-photographs illustrating this paper are by Mr. Andrew Pringle, with the exception of Figs. 55 and 56, which are by Mr. H. B. Bristow, H.M. Consul, Tientsin.

of hæmatozoon is examined, no matter at what time, whether by day or by night, the parasite is found always to be present. I therefore

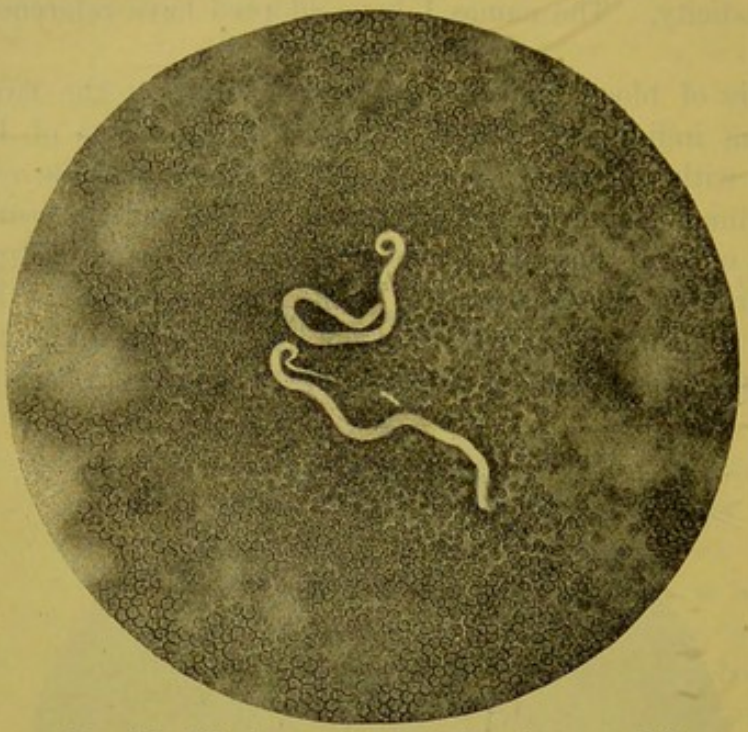


Fig. 52.—*Filaria sanguinis hominis nocturna.* × 160.

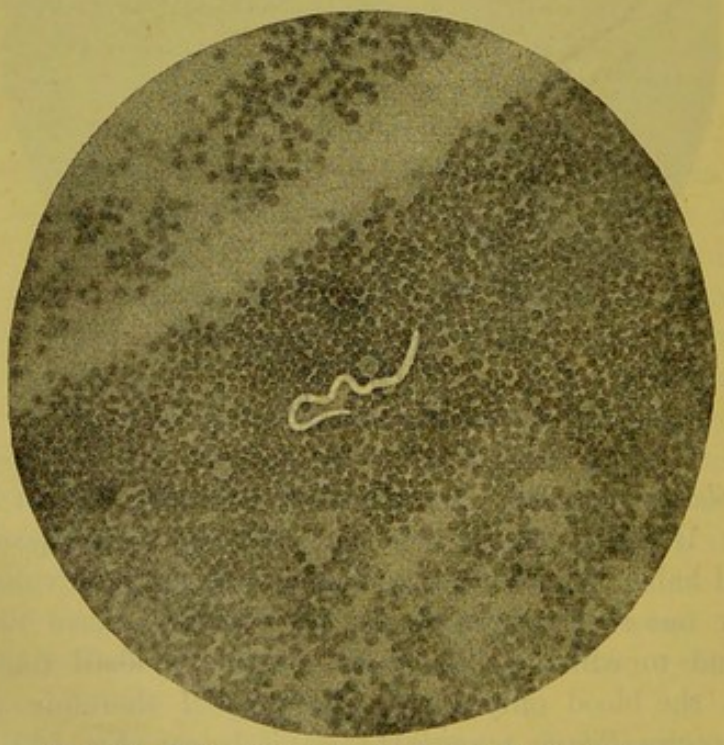


Fig. 53.—*Filaria sanguinis hominis perstans.* × 160.

propose to call it *Filaria sanguinis hominis perstans* (Fig. 53), or, *F. perstans.*

When, in the course of the following remarks, the term "*filaria*" is applied to the minute hæmatozoa circulating in the blood which we are about to discuss, it must be understood that the organism indicated is a filaria only in the sense that it is an *embryo* filaria—the free embryo of a mature parental form which is lodged in some clinically inaccessible part of the body, and which, by a more or less direct channel, pours its brood, the filariæ we see with the microscope, into the circulation. Analogy leads us to infer that the parent worm is a comparatively large animal, probably to be measured by inches, with elaborate alimentary and reproductive organs; it is therefore in marked contrast to the microscopic embryo circulating in the blood, which is an asexual, almost structureless, non-reproducing, and non-developing, simple organism.

Of the three human hæmato-filariæ, the mature form of one only is with certainty known, but we may be sure that the parents of the other two species are also present in every individual whose blood harbours their young. In certain of the lower animals the relationship of the microscopic, immature, circulating embryo to the large, mature, stationary, parental filaria is easily demonstrated. To take one example. In many parts of the world, but especially in China, the dog is liable to be the host of the formidable parasite called *F. immitis*. If we prick the ear of such a dog and examine a little of the blood so obtained with the microscope, we find that it is full of microscopic embryo filariæ very closely resembling the *F. sanguinis hominis*, in fact they might with propriety be called *F. sanguinis canis*. If we kill the dog, we find in the right side of the heart and in the pulmonary artery and its branches two or more—often dozens of mature filariæ, male and female, each of them from 8 to 14 inches in length, and thick as fine whipcord. These are the parents of the embryos we found in the blood with the microscope; we can see exactly similar embryos in the uterus of the parents, and can watch their expulsion through the vagina into the surrounding blood, and we readily recognise their identity.

It is therefore a cardinal fact, and one which the student who would understand the somewhat complicated subject of the *F. sanguinis hominis* must thoroughly grasp, that the organisms which are called by this name are only the embryos of mature parasites living and breeding these embryos in some remote corner of the human body; and that when the word "filaria" is used in this connection in medical literature it usually means filaria embryo.

Description of the *Filariæ sanguinis hominis*.—Before proceeding to the systematic account of what is known of the life

history of these parasites, and of the pathological conditions with which they are associated, I consider that it will conduce to brevity and clearness if I first attempt to describe the parasites as they are met with clinically, that is, as they appear in the blood. They resemble each other so closely that in many particulars one description applies to all. At the same time, there are points of difference between the species available for purposes of diagnosis, and which I think the reader will most readily apprehend if they are described together, and thus directly contrasted.

Their Movements and General Appearance.—As seen in freshly-drawn blood the *F. sanguinis* are long, slender, perfectly transparent, gracefully formed, snake-like animals, exhibiting a prodigious activity. Never at rest for a moment, they coil and uncoil their slender bodies, advance and retire, lash about with their tails, and insinuate their heads among the blood corpuscles. These they toss about like so many balls, keeping those in their neighbourhood in a continual state of agitation. In the case of *F. diurna* (Fig. 51) and *F. nocturna* (Fig. 52), although the movement is such as I describe, and extremely active, it seems to be perfectly purposeless, leading to no result; practically, the animals are stationary. It is otherwise, however, with *F. perstans* (Fig. 53), which is not only as active in coiling and uncoiling itself and wriggling about as the other filariæ, but exhibits at times, in addition, a sort of combined vermicular and snake-like movement leading to distinct locomotion. So much is this the case that, although it is seldom necessary to move the slide to keep *F. diurna* or *F. nocturna* under continuous observation, when *F. perstans* is being studied, one has generally to be constantly shifting the slide to follow the movements of the parasite, and keep it in the field of the microscope.

F. diurna and *F. nocturna* probably possess to a slight extent the power to elongate and shorten their bodies; but this is by no means a noticeable or easily determined feature in their case. *F. perstans*, on the contrary, can be seen, as it travels rapidly about among the corpuscles, to possess this power of elongation and contraction to a remarkable extent. It does not always indulge its locomotive tendencies however; frequently for a time it remains about the same spot, simply wriggling and lashing about in a purposeless manner like the other filariæ. At such times of comparative rest the little animal does not usually attempt to stretch and attenuate itself. Ever and anon, however, it seems to tire of remaining about one spot; it will then suddenly rush through the surrounding corpuscles, insinuating itself over and under and between them; at these times the elongation and corresponding attenuation of the

body become apparent. Being in rapid motion, it is impossible to gauge accurately the extent of this change of dimensions; but if I say, by way of illustration, that from being apparently about the thickness of a goose's quill when not locomoting, it reduces itself when it begins to creep about amongst the corpuscles to the thickness of a crow's quill, and at the same time from being apparently about the length of a finger it becomes as long as a hand, I may succeed in conveying some idea of the relative extent of these alterations.

Frequently lively specimens of *F. perstans* are encountered which, though indulging in active wriggling movements, yet never seem to locomote. If such specimens are carefully scrutinised, it will generally be discovered that the cause of this absence of locomotion is a string or rope of fibrine which the little animal has succeeded in twisting about its body, thus snaring and anchoring itself, so to speak. Round the fixed point, which presently becomes visibly constricted, the filaria moves as round a pivot, the string of fibrine becoming gradually more tense, and looking as if it would bisect the struggling parasite. The point at which this constriction usually occurs is about a third of the length of the animal behind the head —that is to say, at its thickest part. This phenomenon is manifestly an artificial production, and we need not suppose that anything of the sort ever occurs while the parasite is in the body of its human host.

Their Shape.—When filariæ have been some hours on the slide, their movements gradually become much less active, and it is possible to make out several interesting anatomical details. It can then be seen in the case of *F. diurna* and *F. nocturna* that the tail end is acutely pointed, and that what we may designate the head end, after perhaps a very short and slight taper, is abruptly rounded; and that with the exception of this slight tapering towards the head, and the gradual tapering for about an eighth part of the posterior end of the animal towards the pointed tail, the body is perfectly cylindrical, and of uniform thickness. In the case of *F. perstans* the tail is not sharply pointed, but abruptly truncated or rounded, and the taper which thus terminates is much longer in proportion to the rest of the body than is the case in the other filariæ. The taper in *F. perstans* extends through the posterior two-thirds of the animal, and is terminated in the truncated end, where the body becomes reduced to about one-third the thickness of the thickest part of the animal. The cephalic end of *F. perstans* is slightly tapered also, and then abruptly rounded off as in the other filariæ.

The Sheath.—Watching either *F. diurna* or *F. nocturna* under a high power and with a good illumination, from time to time, as it comes into exact focus, the observer can see what appears to be a lash of extreme tenuity dangling from head or tail, or from both, and following them in a passive way in their movements. On closer examination, especially in specimens prepared by certain methods of staining, it is perceived that this appearance is not produced by a lash—in the proper sense of that term, but by the collapsed ends of a very delicate sheath or closed sac, which, though closely applied to and fitting accurately the body of the parasite, is yet not in structural continuity with it (Fig. 54). As this sac is considerably longer than the body of the animal it encloses, and as the animal may shorten or elongate itself and move freely backwards and forwards in it, it necessarily happens that either one or other, or perhaps both ends of the sac are, at times, empty and collapsed, thus giving rise to the appearance of a lash. When the filaria advances, its cephalic end moves into the corresponding cephalic end of the sheath and fills it, and a long ribbon of collapsed sheath dangles from the tail; when the filaria moves in the opposite direction, that is tailwards, the tail moves into the caudal end of the sheath and fills this up, and then a ribbon of collapsed sheath dangles like a lash from the head. Often the ribbon of unoccupied sheath is twisted on itself. This sheath is an exceedingly delicate, structureless membrane—perhaps slightly thicker in *F. nocturna* than in *F. diurna*. If filled out it is seen to correspond in shape to the filaria, that is to say, it is rounded off at the cephalic end like the finger of a glove, and somewhat pointed at the caudal end, though not so acutely as the tail of the animal itself.

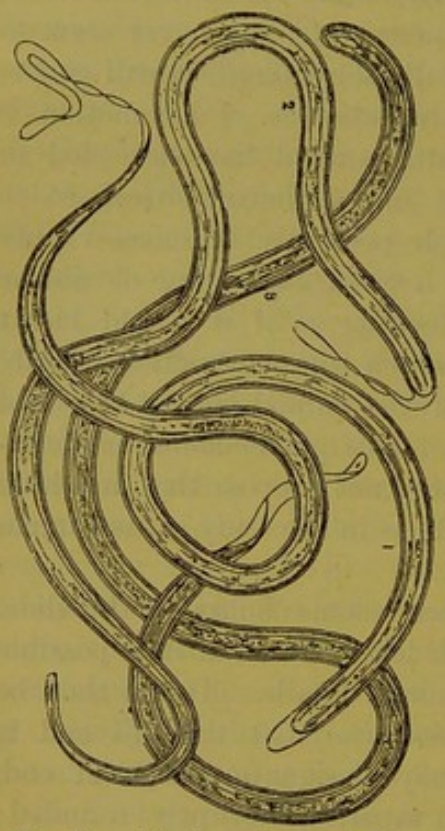


FIG. 54.—*Filaria nocturna*, showing sheath.[1] (Lewis.)

F. perstans has no sheath; it has a very delicate integument which, though in structural continuity with the subjacent substance, I have sometimes succeeded in stripping off. This is only ordinary

[1] The tail end of the sheath is somewhat more pointed than is represented in this illustration.

integument; *F. perstans* has nothing corresponding to the sheath of the other filariæ.

Provided the slides destined to contain either of the sheathed filariæ have been thoroughly cleaned and dried, and are not too cold before being charged, and provided that they have been kept after charging at a moderately warm temperature,—not below 60° or 70° F.,—the presence of the enveloping sheath can always be demonstrated, and that for several days after the preparations were made. Some writers, however, have denied the existence of this structure altogether in the case of *F. nocturna;* others, again, *e.g.* A. J. Zune,[1] declare it to be an occasional feature only. The former set of observers must have employed imperfect methods, or inferior optical arrangements; the latter must have examined their specimens with too low a power, or carelessly, or after they had subjected the slides to certain conditions which, I find after many experiments, cause *F. nocturna* to cast its sheath much in the same way as a snake does its skin.

Filarial Ecdysis.[2]—If freshly-prepared slides of blood, in which *F. nocturna* are present, be kept overnight in a cold place, as in a cold room or near a window in frosty weather, on such slides being examined next day it will be found that a large proportion of the filariæ are, so to speak, naked, having cast their sheaths; and these cast sheaths can readily be discovered lying not far away, collapsed and crumpled up like so many discarded garments (Figs. 55 and 57). In many specimens this process of ecdysis can be seen in actual progress (Figs. 56 and 57), the little embryo, gripped and constricted at some point of its body by the collar-like edge of the hole it has made, struggling to leave the sheath from which it has already partially crept out. It can also be seen that the escape is in every instance effected through a rupture at the extreme cephalic end of the sheath, the head being the first, the tail the last part to emerge. After it escapes from its sheath the filaria looks

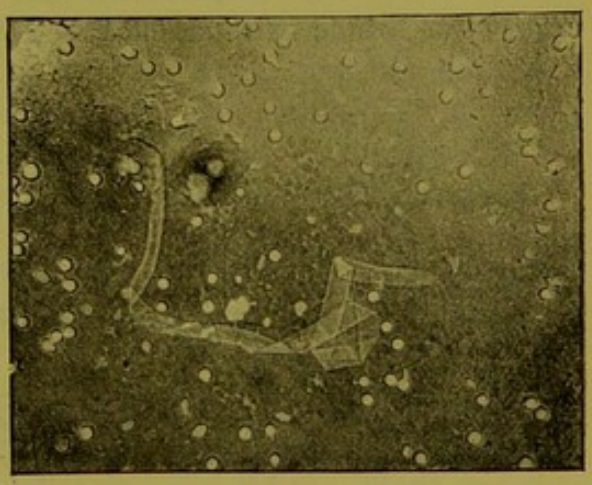


FIG. 55.—The cast sheath of *Filaria nocturna* in chilled blood.

[1] *Mémoire sur la Filariose.* Paris, 1892.
[2] *Brit. Med. Journ.* April 15, 1893.

decidedly attenuated, and I have frequently observed it indulge then in active locomotive movements exactly like those of *F. perstans*.

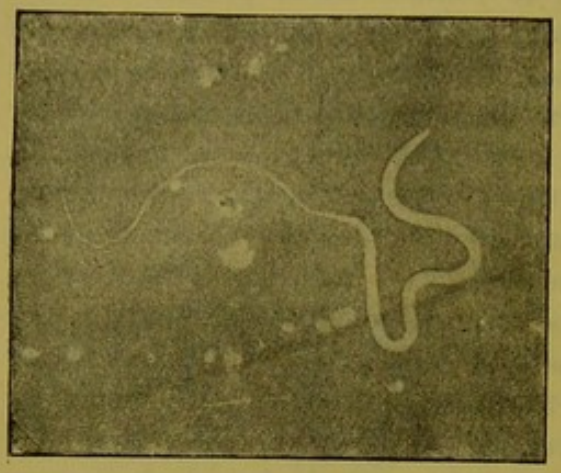


FIG. 56.—*Filaria nocturna* in chilled blood casting its sheath.

I find that a good and convenient way of securing uniform results in this striking and instructive experiment is to proceed as follows:—Six blood slides are put up in the ordinary way, some time after 9 P.M., care being taken that the film of blood between slip and cover-glass is no thicker than a single layer of corpuscles. The slides are then wrapped up, each separately, in blotting paper, and placed in a shallow tin box. The lid of the box is adjusted, and the whole laid on a small block of ice and kept in a room at the ordinary temperature. It is well to place the ice in a watertight box, and to cover the whole with a cloth. This arrangement need not be

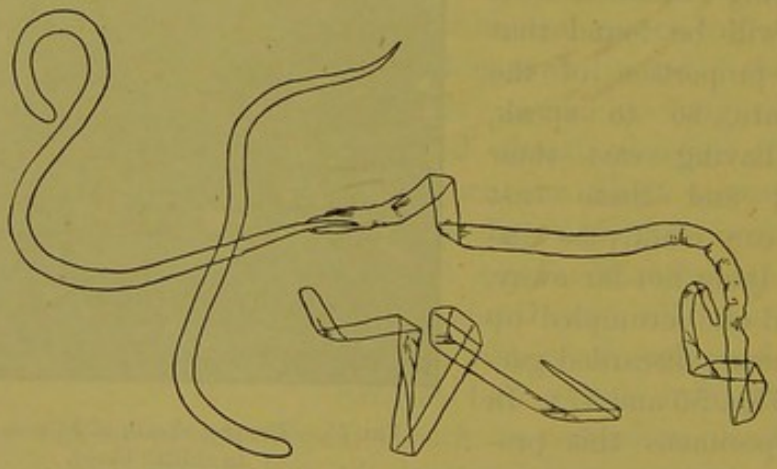


FIG. 57.—*Filaria nocturna* in chilled blood casting its sheath. Preparation about thirty hours old.

disturbed till the following morning. The slides are then—about 8 A.M.—removed. On inspecting them with the microscope, the filariæ are seen at first to be languid in their movements; but as the slides become warmed up to the temperature of the room, the little animals regain their activity and presently begin, and with great vigour, to endeavour to escape from their sheaths. By mid-day some will have succeeded in this, and by evening

the larger proportion are found to be naked, and the preparation will be seen to be strewn at intervals with their empty sheaths. It will also be observed that wherever in the slides the red corpuscles have parted with their hæmoglobin, and their outlines in consequence can no longer be distinguished, there the filariæ rapidly effect their ecdysis. But wherever a filaria is seen to be moving about among still plump and well-defined corpuscles, or in an island of clear serum, there the process is very much delayed—perhaps is never completed or even begun. Manifestly, it is the thickening of the blood, produced by the escape of hæmoglobin from the corpuscles into the plasma, which supplies the mechanical conditions that enable the parasites to burst through and rub off their sheaths. Doubtless, other conditions which bring about a similar escape of hæmoglobin and consequent viscidity of plasma, and which, at the same time, do not kill the filariæ, will have exactly the same effect. Breathing on a cold slip, so that a thin film of watery vapour is condensed on it before applying the blood and cover-glass, is sometimes successful; a little pressure, by breaking up the corpuscles, I have also found at times effective; but the method I describe of chilling on ice is, in my experience, by far the most reliable and the most uniform in its results. The slides must not be frozen; actual freezing kills the filariæ. Short of freezing, and provided the chilling is not kept up for more than twelve hours,—a much shorter time suffices, however,—the lower the temperature, the more likely is the experiment to succeed.[1]

I have not experimented in this way on *F. diurna*, but probably what holds good in this respect for *F. nocturna* holds good for the other sheathed hæmatozoon.

In these chilled slides the liquor sanguinis is not only viscid, but it is stained by hæmoglobin transuded from the corpuscles, and in these respects resembles very closely the blood in the stomach of the mosquito, in which, as I shall presently show, a similar casting of the sheath, probably brought about by similar mechanical conditions, is the first step in the metamorphosis of *F. nocturna*.

Transverse Striation.—Under very high powers a faint, closely set, transverse striation can be detected in the bodies but not in the sheaths of *F. diurna* and *F. nocturna*. A similar but finer striation can be detected with high powers in *F. perstans*. This striation is most probably produced by the longitudinal contraction

[1] Mr. Williams, of Norwich, informs me that he has repeated these experiments on filarial ecdysis lately, and with complete success.

of the body of the helminth throwing a very delicate and but slightly elastic cuticle into folds like the bellows of an accordion.

Anatomical Structure.—When the filariæ have been on the slide for some time, their bodies gradually assume a granular appearance; but when first removed from their host, no such granularity is observable. They are then nearly perfectly homogeneous, and with one or two slight exceptions exhibit no vessel or organ or differentiated structure of any sort, nor can any such arrangement be brought out by lenses of moderate power, or by any of the ordinary processes of staining. It is possible to stain the nuclei of the minute cells of which the embryo is composed (Fig. 58), but no

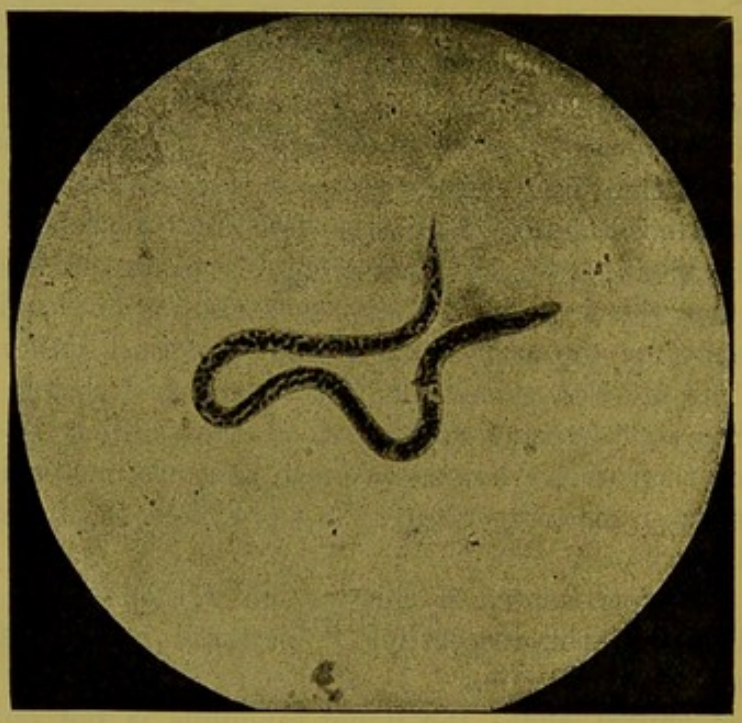


FIG. 58.—*Filaria nocturna* stained so as to show the nuclei.

viscus-like arrangement of these, such as an alimentary canal, is perceptible. In most specimens of *F. diurna* and *F. nocturna*, at a point about the junction of the middle with the posterior third of the body, an obscure, granular sort of aggregation arranged along the middle line, and occupying for some length perhaps about two-thirds of the diameter of the body, can generally be detected. This appearance is usually less distinct in *F. diurna* than it is in *F. nocturna;* nothing similar is visible in *F. perstans.*

The Head and Oral-like Movements.—If attention is directed to the cephalic extremity of the filariæ, a sort of dimpling, gaping, pouting movement can be made out; this is more marked in *F.*

diurna than in *F. nocturna*, and more marked in *F. perstans* than in either. In rhythm the movement reminds one of the breathing movements of the mouth of a fish, but in none of the filariæ can anything like a mouth or alimentary canal or any similar organ be made out.

On carefully watching and focussing the head of *F. perstans*, a minute fang or spine (Fig. 59, *a*) is seen from time to time to be rapidly protruded and retracted like the tongue of a reptile. This organ is so minute and so delicate in structure, and so rapidly moved, that it is difficult to say what its exact shape and anatomy are. It gives the impression of a long, needle-shaped spine, set on a broader bulbous or spatula-shaped base. When retracted, the base can be seen as a minute spot, dark or shining according to the focussing, moving backwards and forwards in what is probably a cavity or sheath in what I may call the head of the animal (Fig. 59, *b*). The length of this fang or tongue may be about equal

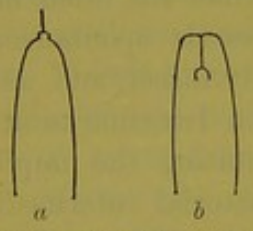


FIG. 59.—Cephalic end of *Filaria perstans* (somewhat diagrammatic and conjectural).

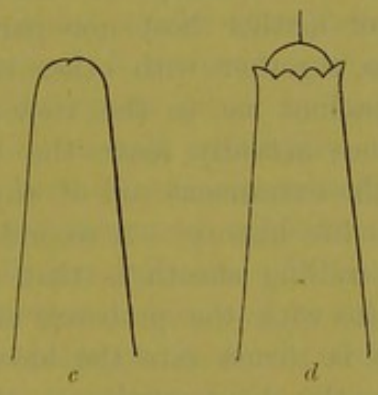


FIG. 59.—Cephalic end of *Filaria nocturna*.

to the breadth of the filaria at its broadest part. According as the spine is retracted or protruded, the anterior end of the worm appears abruptly truncated or markedly conical.

If we examine the head of *F. nocturna* (Fig. 59, *c*, *d*), we can make out a similar though somewhat more complicated arrangement. At one moment it appears to be smoothly rounded off, simple, and structureless (Fig. 59, *c*). At another moment a circlet of six minute, slightly everted lips are seen to surround, like a scalloped collar, what appears to be a thick, broad, rounded tongue, from which occasionally an exceedingly delicate spine or filament is rapidly shot out and retracted (Fig. 59, *d*). If we watch carefully we shall perceive that the circlet of six lips is really the edge of a prepuce-like organ which, from moment to moment, is somewhat slowly drawn back uncovering the tongue, and anon is rapidly

shot over this organ again, with a sudden snapping movement, covering and concealing it completely, the six lips being pursed up and coming accurately together in front. It may sometimes be perceived that when the prepuce is strongly retracted the six lips are distinctly everted.

I have not minutely examined the head of *F. diurna.*

Function of the Sheath and Cephalic Armature.—The function of the cephalic armature of *F. diurna* and *F. nocturna* is evidently to penetrate the tissues of their respective intermediary hosts, after the microzoa have been transferred to the stomach of the latter in the way which I shall presently describe. The function of the sheath is manifestly to act as a sort of mask, or muzzle, whilst the embryo parasite is circulating in the blood of its primary host. But for some such arrangement the parasite would use its formidable weapons on the walls of the vessels, drilling through them, as it subsequently does, in the case of *F. nocturna*, through the tissues of the mosquito. Such a proceeding would be in the interests of neither host nor parasite. The absence of sheath in *F. perstans*, together with other circumstances connected with this parasite, incline me to the view that, unlike the other filariæ, this species does actually leave the blood vessels spontaneously, and without the extraneous aid of any friendly insect, and as a normal step in its life history. A second function I incline to attribute to the long, trailing sheath, is that of facilitating the implication of the parasite with the proboscis of its suctorial intermediary host, when this is thrust into the blood vessels. This is the reason, I believe, for the disproportionate abundance of filariæ in relation to the quantity of blood in the stomach of the mosquito; hundreds being often found in an insignificant quantity.

The V-Shaped Organ.—Another organ can be made out in a large proportion of specimens of *F. nocturna* and *F. diurna*, but it is so minute and so delicate that unless immersion lenses and good illumination are employed it will probably be overlooked. The same remark applies to the structure of the head of the filariæ.

If we look at a point in these filariæ, somewhere about $\frac{1}{450}$ inch behind the head, we may often perceive a triangular, somewhat luminous patch, shaped like the letter V, having its apex at the periphery of the filariæ, and its slightly convex base directed towards the middle line. The apex looks as if it were a fine foramen, and the whole appearance of the organ gives the impression that it is a cavity, the depth of which amounts to about one-fifth of the diameter of the filaria.

Considering the situation of this organ, I am inclined to regard

it as the rudiment of the vagina, which, as will be subsequently pointed out, opens in the mature *F. nocturna* at a corresponding point, and at no great distance from the mouth. In the photo-micrograph (Fig. 58) a break in the continuity of the staining, towards the head end of the filaria represented, can be seen. This, I believe, corresponds to the V organ visible in living specimens. A similar appearance has been described by Nabias and Sabrazès [1] in their stained preparations of *F. nocturna*.

Dimensions.—In consequence of the power of extension and contraction possessed by the *F. sanguinis*, the dimensions of any given specimen must vary considerably from time to time; particularly is this the case with *F. perstans*. It is also to be noted that there is no strict uniformity in the size of the various individuals of the same species—some being larger, others smaller. Consequently, only average or approximate measurements can be given. Roughly speaking, *F. diurna* and *F. nocturna* are of about the same size, just as they are about the same in shape and structure; *F. perstans* may be said to be about a third smaller than these. Lewis gives $\frac{1}{75}$ inch as the average length of *F. nocturna*, and $\frac{1}{3500}$ inch as its average breadth; the measurements given by many other observers, as well as those made by myself, practically correspond with these. Lewis gives as his extreme measurements $\frac{1}{68}$ by $\frac{1}{3000}$ inch, and $\frac{1}{125}$ by $\frac{1}{7000}$ inch; I have measured specimens as large, but never any so small as these figures indicate. Different specimens of *F. perstans* also differ somewhat in size, and as the extensile and contractile power of this hæmatozoon is much more marked than that of the other filariæ, the proportions of any given specimen at any two given times must exhibit greater differences. When *F. perstans* dies, or is nearly dead, it seems to be at about its average length and breadth; its length then may be put down on the average at $\frac{1}{125}$ inch and its breadth at $\frac{1}{3500}$ inch. It will thus be seen that whereas the diameter of *F. diurna* and *F. nocturna* is about that of a blood corpuscle, that of *F. perstans* is considerably smaller.

Viability.—All of the filariæ exhibit a marvellous tenacity of life. In slides in which evaporation and drying of the blood is prevented by oiling the edge of the cover glass, they keep alive for many days; in fact, in ordinary slides, so long as the blood remains fluid, so long do the filariæ continue to move. I have seen them thus languidly moving fifteen days after mounting. As the movements slow down the bodies of the filariæ gradually become more granular. They generally die in an extended or semi-flexed attitude. After death

[1] *Comptes rendus Soc. de biol.* Paris, May 27, 1892.

the body becomes still more granular and less defined, and gradually disintegrates and vanishes.

Numbers.—As regards the number of filariæ in any given slide of blood, this, of course, will depend on the quantity of blood it contains, and the time of day or night, if the case is one of *F. diurna* or *F. nocturna*, at which the blood was drawn, as well as on the degree of filariation of the patient. In an ordinary slide of blood from a case of *F. nocturna*, it is no unusual thing to find from 50 to 100 parasites. The most accurate observations bearing on this point, with which I am acquainted, are those made by Dr. Stephen Mackenzie in a case of filarial chyluria, reported in the *Transactions of the Pathological Society of London*, 1882. After accurately counting the number of filariæ in a known quantity of blood, and assuming that the parasites were distributed throughout the circulation in the same proportion, he estimated that the patient's blood contained from 36 to 40 millions of filariæ. Doubtless in some cases they are even more numerous than this, whilst in others again they are fewer.[1]

Although observations of cases of infection with *F. diurna* are as yet too few to justify definite conclusions on this point, my belief, founded on what I have already seen of this parasite, is that it is not quite so prolific as *F. nocturna*.

I have examined a sufficient number of cases of *F. perstans'* infection to be able to say that this parasite does not occur, as a rule, in nearly the same profusion as either of the other two. Frequently several slides have to be searched before one example is discovered. Some slides may contain one or two, but it is rather a rare thing to find more than five or six in a preparation of moderate dimensions. I have seen slides, however, in which *F. perstans* was very abundant.

Pairing of Filariæ in the Blood.—A singular circumstance about the distribution of the filariæ in the blood is that they tend to occur in pairs (Figs. 52 and 77). This peculiar arrangement happens so frequently that it cannot be the result of accident on all occasions. If in searching through a slide one filaria is encountered, very probably a second will be found not far away. This pairing is especially noticeable in slides on which the blood has been dried in a thick layer, and in which, therefore, there has been, presumably, a minimum of interference with the normal distribution of the filariæ. It is also very noticeable in slides which contain few

[1] In slides of blood prepared as directed in the Appendix, and containing about half a drop of blood in each, I counted on one occasion 413, and on subsequent occasions 120, 359, 327, and 300 filariæ.

filariæ, as is so frequently the case in slides of *F. perstans.* If in searching for the latter parasite, after perhaps several slides have been examined and found blank, a filaria is at last encountered, the chances are that in close proximity to the first, and on the same slide, a second will be discovered. I possess slides containing *F. nocturna* in great abundance, in which this pairing arrangement is so marked that any explanation, such as coincidence, is inadmissible. Very possibly this, what might be called premature pairing of immature animals, may have some bearing on the future sexual relationships of the individuals. Whatever may be its object, or whatever its explanation, there can be very little doubt about the fact.[1]

Filarial Periodicity.—In all these curious anatomical and physiological arrangements we can see how steadily and ingeniously nature fights in the interest of the parasite, so as to give it the best opportunity possible against the long odds it has to contend with in its efforts to attain maturity and secure the continuation of its species. But in none of them are evidences of what may be called design so manifest as in that known as "filarial periodicity," so termed by my friend, the late Dr. Spencer Cobbold.

This point, seeing that it has a bearing on at least two of the filariæ, may, in order to avoid repetition, be best discussed here.

My attention was first directed to this strange phenomenon in 1879. At that time I was endeavouring to ascertain the degree of prevalence of *F. nocturna* among the population of Amoy, China, and surrounding country. To help me I employed native assistants. After a time I observed that work during the day seldom resulted in the discovery of filariated subjects, whereas work during the evening or night was much more frequently rewarded by "finds." Suspecting that this was not altogether accidental, I made a series of systematic examinations, extending over many weeks, on the blood worms of two individuals, the blood being examined every three hours; and also a number of isolated examinations at irregular times, in other individuals similarly infested. As a result of this I found that a very definite and, in ordinary states of health and habit, a very inflexible law regulates the entrance into and exit of the filariæ from the circulation. During the day, save for a very rare straggler, they are entirely absent. Towards sunset—about 5 or 6 o'clock—they begin to enter the general circulation.

[1] I have not attempted to do so, but I think it might be possible to determine if this arrangement is a sexual one, and if the pair consist, in every or most instances, of a male and a female. If the V organ is found in one and not in the other, I would look upon the former as female and the latter as male. To work out this interesting biological point in a reliable manner, thick films of blood and special staining methods would have to be employed.

Gradually, as the night wears on, their numbers increase. About midnight they are most numerous. As morning approaches they get fewer and fewer, and by 8 or 9 A.M. they have disappeared. The following evening they come in again about the same hour,

FIG. 60.—Chart illustrating filarial periodicity in a case of *Filaria nocturna* infection. Observations made every three hours, the same quantity of blood being examined each time.

increase in numbers till midnight as before, and then again gradually disappear as on the previous day. This goes on every day, and from day to day and week to week, and, doubtless, from year to year. Unless under certain abnormal conditions, to be presently described, this periodicity is never interrupted. These observations on the periodicity of *F. nocturna* have been amply confirmed by Myers in Formosa, Mackenzie in London, Sonsino in Egypt, and many other observers in different parts of the world.

When I became convinced that besides *F. nocturna* the blood of man might harbour at least two other species of filarial hæmatozoa, I set about ascertaining if they too observed a similar periodicity. A series of systematically conducted observations convinced me that there was nothing of the kind in the case of *F. perstans;* but that *F. diurna*, on the contrary, observes a well-defined periodicity, but a periodicity which is the exact opposite, as far as the time of appearance and disappearance of embryos in the blood is concerned, to that characteristic of *F. nocturna*. I found that *F. diurna* comes into the general circulation about 9 or 10 in the morning, increases in numbers till 1 or 2 P.M., then decreases in numbers, gradually disappearing for the night about 9 or 10 P.M.

Inversion of Filarial Periodicity.—Dr. Stephen Mackenzie, by a series of very interesting and carefully conducted experiments on the periodicity in *F. nocturna*, has shown that filarial periodicity is somehow bound up with the quotidian habits of sleeping and waking. By causing a patient, in whose blood *F. nocturna* abounded at night under normal conditions, to invert his habit as regards sleeping and

waking,—that is, to sleep during the day and remain awake during the night,—the periodicity of the filariæ in his blood was correspondingly inverted—that is, they came into the blood during the day and disappeared from it during the night. These experiments I have repeated and confirmed, and also somewhat varied. Some of my experiments show that if sleep is indulged in for short times and at short intervals, and this habit kept up for several days, filarial periodicity becomes completely broken up and is no longer maintained—that, in fact, filaria embryos, under such circumstances, are constantly present in the circulation. The febrile state seems to exercise a similar influence, but change in the hours of eating produces no corresponding change in periodicity. (See chart, Fig. 67, p. 780.)

The mind naturally craves for some explanation of this singular phenomenon. What is its object, and what is the mechanism by which it is brought about?

Object of Filarial Periodicity.—The object is not far to seek, at all events in the case of *F. nocturna*, whose periodicity is certainly an adaptation to the nocturnal habits of the particular species of mosquito which, as will appear in the sequel, acts as its intermediary host. Probably a similar explanation applies to the periodicity of *F. diurna*, and the absence of periodicity in *F. perstans;* but as the intermediary hosts of these parasites are not yet known, this may be considered as being only very probable.

Theories as to the cause of Filarial Periodicity.—As regards the mechanism by which periodicity is brought about, there have been many conjectures; but as yet they are only conjectures. That it is not the simple fact of sleeping and being awake which causes the ingress and egress of filariæ, is evident from the circumstance that in the case of *F. nocturna* the embryos begin to enter the circulation hours before the usual time for sleeping, and begin to leave it hours before the usual time for waking; and the same remark holds good in an inverse sense for *F. diurna.*

1st. It has been suggested that filarial periodicity depends on an intermittent parturition in the parent filaria, corresponding in time and rhythm to the influx of embryos into the blood. As a corollary to this, we must assume that the life of the embryo filaria is at most an affair of a few hours only, and that daily the swarm dies and disintegrates in the liquor sanguinis, or is removed by other means from the blood. It has been observed, however, that in chylous urine, and other forms of chylous discharge associated with *F. nocturna*, the parasites are always present in the chylous discharges by day as well as by night, although in the

same cases the filariæ are present in the blood during the night only. From this fact I think we are justified in inferring that the birth of embryos is more or less continuously carried on, and, therefore, that periodicity is quite independent of this process. Doubtless the reproductive activity of many of the species of filaria is very great; but it is difficult to believe that any number of parent worms, which we could reasonably conceive to be present in any given human being, could give birth every night, and night after night for years on end, to a daily swarm of 40 millions of young. Nor can we see what object in the interests of the parasite could be served by the implied enormous daily mortality. Further, on the supposition of intermittent reproduction and death, how can we account for the occasional stragglers met with in the blood at those times when the great swarm is normally absent; and how comes it that the embryo, which can live for days in blood outside the body, should die so soon in blood inside the body, where, being in its natural habitat, its life, we might suppose, would be most prolonged?

2nd. Myers[1] has made some very elaborate and careful observations—partly confirmed by Dr. Stephen Mackenzie—on the respective activity and viability of *F. nocturna* embryos according as they are observed in slides of evening blood or slides of morning blood. He asserts that the embryos in evening blood are more active and live on the slides longer than the embryos in the morning blood; and he considers this want of activity and relatively feeble vitality of the embryos in morning blood an indication that they are about to die. Deducing the inference from experiments on the effect of carbonic acid and oxygen on filariæ mounted on slides, he concludes that the assumed daily death of the filariæ is attributable to the relative deficiency of oxygen in morning blood; and the ingress of the filariæ, their greater vigour and vitality, to the attractiveness and nutritive properties of the evening blood, which he maintains is relatively richer in oxygen. But if this is the explanation of the periodicity of *F. nocturna*, how explain the exactly opposite periodicity of *F. diurna*, or the absence of periodicity in *F. perstans?* If Myers' hypothesis is correct, that which attracts one species of filaria repels the other but closely similar species. Nor does this hypothesis explain the presence in the blood of the occasional diurnal straggler, and, moreover, it implies an apparently unnecessary mortality of 40 millions per diem.

3rd. I have suggested that when the filariæ are absent from the general circulation, they are detained in some organ or tissue of the host, waiting there the recurrence of physiological conditions

[1] *Chinese Imp. Mar. Customs Gaz. Med. Rep.* 32nd issue, 1886.

which will again permit of their circulation in the blood. What these conditions are, and what the organ or tissue is in which the filariæ are detained, I cannot say. Support is given to this hypothesis, however, by the fact that in the analogous parasite of the dog (*F. immitis*, whose embryos seem to observe a modified periodicity), when the embryos are fewest in the general circulation, they are to be found in enormous numbers in the blood of the lungs, thousands in every drop. If this should prove to be typical of what happens in the case of the human blood-worms, the resting or restraining organ or tissue will probably be found to be different for the respective parasites. I have examined in examples of *F. nocturna* infection day blood from the lungs (expectorated) and from the spleen (aspirated), and Myers has examined day blood from the spleen and liver (aspirated), with negative results; so that it is probable that if the filariæ do rest in any of the viscera, those just mentioned must be excluded.

I think that sufficient proof is forthcoming[1] that parturition by the female filaria is continuous; at all events, that there is no diurnal periodicity about the process; and also that the newly born filariæ are being continually carried by the lymph stream into the blood. Hence the stragglers occasionally met with in the blood at those times when the great swarm of embryos is normally absent. These are newly born filariæ, which, after making one or more rounds or part of a round of the circulation, are filtered out as soon as the blood carries them to the organ or tissue in which the majority of the swarm is resting or detained.

Life Span of Filaria Embryos.—How long the individual filaria embryo lives has yet to be determined; but if their viability outside the body is any guide to their viability inside the body, their span of life must be reckoned, not by hours or days, but probably by weeks. That they do die is certain, for otherwise they would accumulate to such an extent in the blood as to make life to the host impossible; but where they die, when they die, and what kills them, as well as what fixes and liberates them, are all as yet mere matters of speculation.

Diagnosis of Embryo Filariæ.

For purposes of comparison, and to facilitate diagnosis, I have arranged the characteristic features of the three filariæ as above described in the following table:—

[1] *The Filaria Sanguinis Hominis and certain new forms of Parasitic Disease in India, China, and Warm Countries.* London, 1883.

	Filaria diurna (Figs. 51, 78).	*Filaria nocturna* (Figs. 52, 54, 58, 77).	*Filaria perstans* (Figs. 53, 59, 78).
1.	Measures $\frac{1}{75}''\times\frac{1}{3500}''$ or thereabouts.	Measures $\frac{1}{75}''\times\frac{1}{3500}''$ or thereabouts.	Measures $\frac{1}{125}''\times\frac{1}{2500}''$ or thereabouts.
2.	Is provided with a sheath.	Is provided with a sheath.	Has no sheath.
3.	Caudal end tapers gradually for one-eighth or one-fifth of the length of the animal, and ends in a sharp point.	Caudal end tapers gradually for one-eighth or one-fifth of the length of the animal, and ends in a sharp point.	Caudal end tapers more gradually for two-thirds of the entire length of the animal, and is abruptly truncated where it becomes reduced to one-third of the diameter of the thickest part of body.
4.	Cephalic end rounded off, and has distinct pouting movements. Minute anatomy not known.	Cephalic end rounded off, and has obscure pouting movements produced by the movements of a six-lipped prepuce.	Cephalic end is either conical or truncated, passing from one shape to the other by a peculiar jerking, extending and retracting movement.
5.	Minute anatomy not known.	From time to time a thick tongue-like organ, provided with a delicate retractile spine, is protruded at cephalic end.	From time to time a minute tongue-like organ, provided with a retractile spine, is protruded and withdrawn at cephalic end.
6.	Appears in the blood during the day, disappearing from it at night.	Appears in the blood at night, disappearing from it during the day.	Present in the blood both by day and by night.
7.	Has a wriggling but no locomotive movement.	Has a wriggling but no locomotive movement.	Has a locomotive as well as a wriggling movement.
8.	Slightly marked granular aggregation about the junction of the middle with the posterior third of the body in some specimens.	Well marked granular aggregation about the junction of the middle with the posterior third of the body in some specimens.	Body homogeneous throughout, and no such aggregation.
9.	Has a V shaped organ.	Has a V shaped organ.	No V shaped organ.

FILARIA SANGUINIS HOMINIS DIURNA. (Figs. 61, 78.)

The free embryonic form as seen in the blood, and which has just been described, is the only stage in the life history of this parasite about which we have any certain knowledge.

The physiological and morphological characters of the sharp-tailed and the blunt-tailed filariæ are so marked and so distinctive that it is impossible to regard them as anything but different species; but, I can readily understand, objection may and probably will be taken by some to the inadequacy of the data from which I infer the existence of a third species of blood-worm in man. My confidence, however, in the regularity of the operations of nature, even as affecting these lowly organisms, is such, that the one fact

of difference of periodicity in the sharp-tailed, sheathed filariæ is to me quite sufficient to prove difference of species. The opposite periodicities of *F. diurna* and *F. nocturna* constitute, from a physiological point of view, a radical and specific difference, seeing that they imply a difference in intermediary host.

Although, when seen alive in the blood, the embryo *F. diurna* resembles so closely *F. nocturna* as to be practically indistinguishable therefrom, a singular difference is observable between the

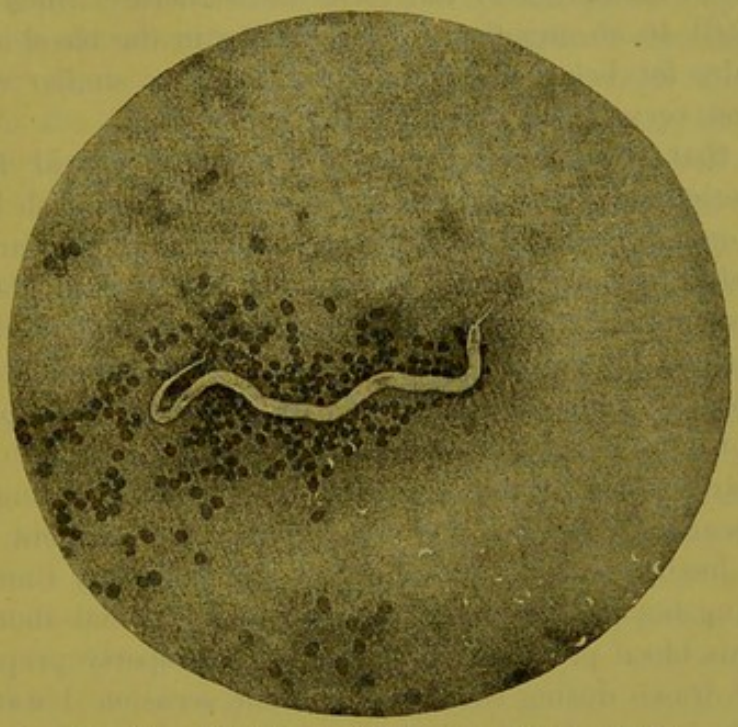


FIG. 61.—*Filaria sanguinis hominis diurna.* × 160.

species when seen post-mortem on dried and stained slides of thickish blood films. If Figs. 77 and 78, pp. 848, 849, are compared, it will be observed that whereas *F. nocturna* is arranged in graceful curves, such as a clever draughtsman might produce with a flourish of his pen, *F. diurna*—the larger and lower of the parasites in the photomicrograph (Fig. 78)—looks shrunken and thickened, and has assumed a stiff, rigid, and very ungraceful attitude. This distorted, ungraceful appearance of *F. diurna* in thick blood-films is perfectly characteristic. Unless at the very edge of the film of blood, where it is exceedingly thin, and therefore has dried almost instantaneously, this appearance is very common; so much so, that I consider it a diagnostic mark of value.

A close anatomical resemblance between two specimens of filaria embryos is no sufficient reason for concluding that they belong to the same species. It is a well-known fact that nearly all filaria

embryos closely resemble each other, even although they may belong to widely different species. In many cases the species, as regards their embryos, are only distinguishable by the physiological tests of habit and ultimate developmental change. Moreover, a close resemblance anatomically is what we would look for in two species so closely resembling each other in habit and conditions of life. If the form and structure of *F. nocturna* is that which is best adapted for circulating in the blood *at night*, and being abstracted from it by an insect, surely the form and structure which would be best adapted to an organism for circulating in the blood *during the day*, and also for being abstracted from it in a similar way by an insect, must very closely resemble this.

Seeing that so much depends on the physiological feature of peculiar periodicity, I have been very careful to establish by a long, and more or less consecutive series of observations the fact that, in a normal state of health and habit, in certain individuals a filarial hæmatozoon appears in the blood during the day and disappears from it during the night. I made consecutive examinations of a negro's blood, in whom the parasite I call *F. diurna* was present, at intervals of four hours during one week, with the result of finding that the parasites invariably appeared in the blood during the day and disappeared from it during the night. This patient, who was in perfect health, both I and others[1] examined many times during the following ten months; and we never failed to find abundance of filariæ in his blood provided the slides were properly prepared, and from blood drawn during the day. On one occasion I watched the influx of filariæ into the circulation, examining slides of blood drawn hourly. The following table shows the result of this examination :—

1 slide of blood drawn at	5	A.M.	contained	0	*F. diurna.*	
,,	,,	6	,,	,,	0	,,
,,	,,	7	,,	,,	0	,,
,,	,,	8	,,	,,	1	,,
,,	,,	9	,,	,,	3	,,
,,	,,	10	,,	,,	21	,,
,,	,,	11	,,	,,	16	,,
,,	,,	12	,,	,,	21	,,

I also watched the egress of the filariæ in the evening; then

1 slide of blood drawn at	6	P.M.	contained	7	*F. diurna.*	
,,	,,	8	,,	,,	8	,,
,,	,,	9	,,	,,	4	,,
,,	,,	10	,,	,,	1	,,

[1] He was in the London Hospital for some time for operation for the radical cure of hernia, and was seen there and examined by Sonsino, of Pisa, Stephen Mackenzie, and others.

It may be objected by some that, granted the accuracy of these observations, the explanation of the peculiarity in periodicity in this case may lie in some abnormality of the particular individual parasite, or in some peculiarity in this particular human host, which led to an inversion of the usual periodicity of what, after all, was only an ordinary *F. nocturna;* one case, it might be said, is not enough to draw conclusions from. To this objection I would answer, that in the hundreds of cases of *F. nocturna* infection I have examined, I never saw the characteristic periodicity inverted unless under circumstances of disease or inverted sleeping habits; nor, so far as I can ascertain, is there any such abnormality recorded in the literature of the subject. Moreover, the case on which these observations were made is not the only one of *F. diurna* I have met with. The man Mandombi—whose case Stephen Mackenzie recorded[1]—had in his blood a large sharp-tailed, sheathed filaria which appeared during the day. Possibly the disease from which this patient suffered might be responsible for irregularities in periodicity, and his blood parasite was really *F. nocturna;* but I have lately received from Old Calabar blood-slides, prepared from a healthy individual, of which those prepared from blood drawn during the day contain filaria embryos possessing the characters of *F. diurna*, whereas those which have been drawn during the night from the same man contain none. More recently still I have found *F. diurna* in the blood of a negress from Dahomey. Further and more extended observations on this point are, of course, highly desirable; but that which has already been observed is, to my mind, conclusive as to the specific difference between *F. diurna* and *F. nocturna.*

As to the rest of the life history of this parasite, its intermediary host, and its mature form, little or nothing is positively known. I am informed that there are certain flies,—*Chrysops dimidiatus*, van der Wulp,—hominivorous blood-suckers with diurnal habits, which frequent the creeks and plantations of Old Calabar. They are called "mangrove flies" by Europeans, "uyo" and "ukpom" —there appears to be two species—by the negroes; and I am inclined to regard these flies as possible intermediary hosts for *F. diurna.* They are said to be very voracious. They attack any exposed part of the body. After gorging themselves with blood, they drop off on to the ground. In the event of their feeding on an individual with *F. diurna* in his blood, doubtless they will imbibe the parasite, and possibly act as its intermediary host, very much in the same way as the mosquito does for *F. nocturna*, or cyclops for *F. medinensis.*

[1] *Clinical Soc. Trans.* vol. xxiv. 1890.

As regards the mature form of *F. diurna*, the only conjecture I have any grounds for making is that it is the long, but imperfectly known *F. loa* (see p. 962). The patient who supplied me with blood for my observations on *F. diurna* informed me that when a child he had a *F. loa* in his eye; that after a time it disappeared spontaneously; and that this is not an uncommon parasite in his country. *F. loa* is a sexually mature parasite. Professor Leuckart kindly sent me a drawing of the embryo from the uterus of this parasite; and, so far as I could make out from the drawing, they seem to correspond with *F. diurna* as regards shape, although a sheath is not represented.

Pathological Relations.—I am not aware of any disease which might be attributed to this parasite, though, doubtless, in the future, as our acquaintance with it extends, it may turn out to be not absolutely innocuous.

Treatment.—I cannot suggest any treatment further than a careful prophylaxis in the direction of a pure water supply.

FILARIA SANGUINIS HOMINIS NOCTURNA (*FILARIA BANCROFTI.*) (Figs. 62, 54, 58, 77.)

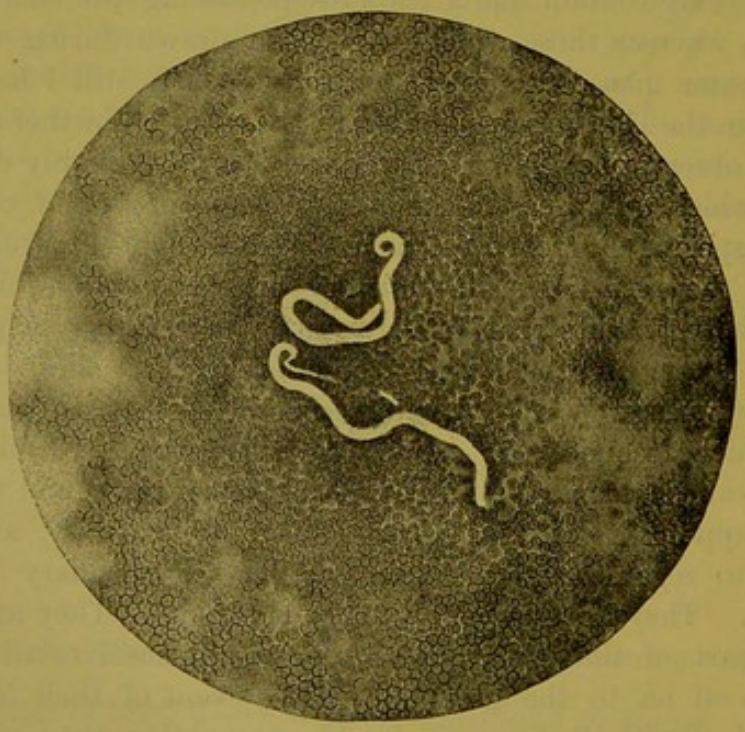


Fig. 62.

History.—This parasite was first discovered by Demarquay[1] in 1863. He found the embryonic form, such as has just been

[1] *Gaz. méd. de Paris*, 1883, t. xviii. p. 665.

described, in the milky fluid of a chylous dropsy of the tunica vaginalis in a patient from Havana. Although Demarquay described and demonstrated the parasite, his discovery excited very little attention; in fact, as pointed out by Magalhães,[1] it was completely overlooked, even by his own countrymen, until a Brazilian physician, Dr. Bernhaus de Lima,[2] recalled the forgotten observation. In 1866, Wucherer,[3] in Bahia, found the same parasite in chylous urine, and, after collecting a series of twenty-eight similar cases, published in 1868 an account of his independent discovery. In 1870, Lewis,[4] in India, in ignorance of what had been done by Demarquay and Wucherer, found the same filaria—also in chylous urine. It was not, however, until Lewis discovered the fact that the human blood was the normal habitat of the embryo filaria that its full significance as a factor in tropical pathology began to be apprehended. In 1874, Sonsino,[5] also in ignorance of these previous discoveries, found the filaria in the blood of a Jew lad in Egypt; and since that time he and many other workers in different countries have gradually extended our knowledge of this nematode until, at the present day, our information about the principal facts of its life history, and many points relating to its bearing on pathological subjects, has become, in most particulars, fairly complete.

The Parental Form; *Filaria Bancrofti.*—The immature, embryonic nature of the filariæ seen in the blood and certain morbid discharges was early recognised, and diligent search was accordingly made for the parental forms inferred to be present somewhere in the body of the human host. These parental forms were first discovered by Bancroft,[6] in Brisbane, Australia, in 1876. Since that time *F. Bancrofti*, so named by Cobbold,[7] has been found, in India, Brazil, and China, some half-dozen times, in lymphatic abscesses of arms and legs, in lymphatic dilatations in the spermatic cord and scrotum, and, in one doubtful instance, in the left ventricle of the heart.

Description of the Female.—Usually the male and female are found together. The female Cobbold describes as follows (Fig. 63): "Body capillary, smooth, uniform in thickness. Head with a simple, circular mouth, destitute of papillæ. Neck narrow, about

[1] *Rev. dos Cursos theoricos e prat. da Fac. da Med. de Rio de Janeiro*, No. 3, Anno iii. 1886.

[2] *These inaug.* Rio de Janeiro, 1881.

[3] *Gazetta Medica da Bahia*, December 5, 1868, and September 30, 1869. Leuckart's *Parasiten*, Band ii. p. 640.

[4] *Loc. cit.*

[5] *Veterinarian*, April 1874. [6] *Lancet*, July 14, 1877. [7] *Parasites*, 1879.

one-third the width of the body. Tail of female simple, blunt-pointed; reproductive outlet close to head; anus immediately above the tip of the tail. Length of largest female, 3½ inches; breadth, $\frac{1}{90}$ inch." As this description was made from glycerine-preserved specimens, the original dimensions of the fresh helminths may have been somewhat different. A simple alimentary canal, marked by a slight bulging and constriction at the junction of œsophagus and intestine, runs from mouth to anus; the remainder of the interior of the worm is occupied by the double uterine or ovarian tubes, terminating at the posterior end in the ovarian tubules and uniting anteriorly in a short vagina. From commencement to termination these uterine structures are stuffed with myriads of embryo filariæ at all stages of development.

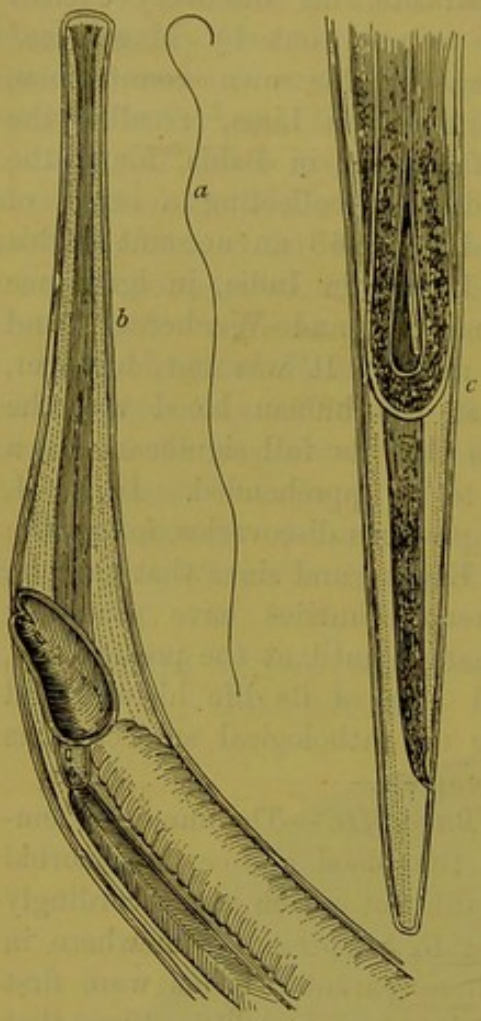


FIG. 63.—*Filaria Bancrofti*. Mature female form of *Filaria nocturna*. (After Cobbold.)
a, Natural size.
b, Cephalic end.
c, Caudal end.

Development of Embryo.—As the vagina is approached the gradual formation of the embryo can be traced, and some interesting points in its anatomy made out. From what is seen there, backed by analogy, it is believed that the loose sheath which envelops the free embryo *F. nocturna*, as seen in the blood, is really what, at an earlier stage of development, was the chorional wall of the ovum. This point has not been worked out in *F. nocturna* itself, but in the closely analogous blood-worm of *Corvus torquatus* I have followed the process. In the latter parasite, for some time before the embryo reaches the vulva, it is engaged in a vigorous endeavour to separate as far as possible the poles of the globular ovum (Fig. 64). By dint of vigorous working and stretching it gradually gets the poles of the ovum further apart, so that by the time the embryo emerges from the vulva the oval sack has become converted into the elongated, closely-fitting sheath

(Fig. 64). Before the commencement of this stretching process the ova of *F. nocturna* measure about $\frac{1}{300}$ inch by $\frac{1}{750}$ inch."

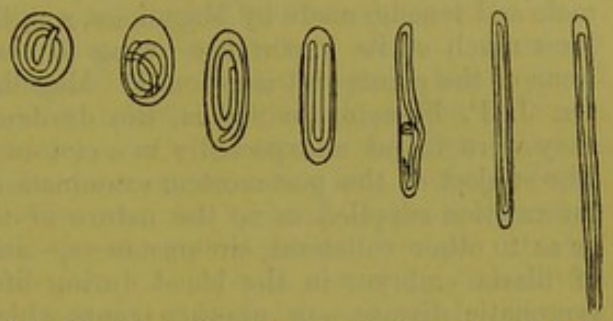


FIG. 64.—Stretching of the chorional envelope to form the sheath of the free embryo *Filaria corvus torquatus*.

Description of the Male.—Only fragments of what with certainty was known to be the male worm have been found, and the description of these is very incomplete. The width of a fragment measured by Lewis was $\frac{1}{180}$ inch. Bourne[1] gives a brief description of the caudal end of a more complete male, *F. Bancrofti*, found by Sibthorpe in Madras. He says that the anus appeared to be sub-terminal, and that the two spicules were slightly in advance of the anus. In the specimen examined one spicule was broken off, but the other remained *in situ*. This undisplaced spicule was seen to be broad at its proximal extremity, but gradually tapered until it became capillary at its free end. About halfway down the spicule there was a lateral prominence, at which point the organ was folded on itself, so that this prominence was the actual free end, whilst the extreme end of the capillary portion was doubled back and applied to the broader base close to the body of the animal. Very probably this peculiar arrangement of the spicule arose from damage done to the specimen in mounting; at all events, it is so unusual in the filariæ that the description cannot be accepted without further confirmation. Neither Bourne nor Sibthorpe give any measurements, nor do they describe the number and arrangement of the caudal papillæ.

The reader will perceive that the description of the male parasite, on which classification principally depends, is still very incomplete. As recent discovery has shown that the embryos of several species of filariæ may haunt the human blood, it becomes a matter of great practical importance, as well as of scientific interest, that the distinctive features of the various species, both in the embryonic and in the mature form, should be carefully stated. Medical men, should they encounter either embryo or mature hæmatozoa, ought therefore to note every feature and circumstance which might contribute to give precision to our knowledge about these points. It will not suffice simply to state that a filaria embryo, or a mature filaria, was found in the blood or in the tissues. The size of the parasites; their periodicity; their exact location; the diseases, if any, with which they were associated, must be stated. In the case of a mature male, in addition to these, a careful note should be made of the structure of the caudal end;

[1] *Brit. Med. Journ.* June 1888.

the number, size, and shape of the spicules and caudal papillæ; the position of the anus; and other zoological features.

An important and exhaustive description of two mature filarial hæmatozoa, male and female, made by Magalhães, a skilled and accurate helminthologist, loses much of its importance owing to absence of reliable information on some of the points just mentioned. Magalhães received these parasites from Dr. J. P. Figueiro de Saboia, Rio de Janeiro, with the information that they were found unexpectedly in a clot in the left ventricle of the heart. The subject of the post-mortem examination was a child; but there was no information supplied as to the nature of the disease which caused death, or as to other collateral circumstances,—such as the presence or absence of filaria embryos in the blood during life, or the presence or absence of lymphatic disease,—or on other points which might have served to throw light on the identity of the parasites. The position assigned to them, the *left* ventricle, is a very unusual one for mature filariæ to occupy; one so fraught with danger to the host, and therefore to the parasite itself, that I have some suspicion that there has been an error in observation, and that the clot containing the parasites really came from some neighbouring structure, and not from the one assigned to it. Assuming they were *F. Bancrofti*, if the particular specimens in question came from the left ventricle, and were normally located in this instance, it is a very curious thing that, in the thousands of post-mortem examinations made annually in tropical and filarial countries, a similar occurrence has not been noted either before or since. These worms are too large to be often overlooked. My belief is that if the filariæ described by Magalhães were *F. Bancrofti*, as I believe they were, they came from the lymphatics in the thorax—probably from the thoracic duct, or from the veins, or the right side of the heart.

Magalhães expresses himself with great caution as to their identity with *F. Bancrofti*. He points out many features they had in common with *F. Bancrofti;* but he does not positively assert their identity.

As no detailed account has appeared of the zoological characters of Magalhães' parasites in English literature, and as the magazine in which they were originally published is not very accessible to English readers, I append a translation of part of this important paper here.[1] It is well to state that the dimensions of the parasites described were probably considerably altered by the medium in which they were preserved.

Description by Dr. Magalhães of two Filariæ found in the Left Ventricle of the Heart at Rio de Janeiro.

Female Filaria.—Filiform, very fine, white opalescent, not quite opaque. Body cylindrical, almost uniform in thickness, very gradually attenuating towards tail, marked by very fine transverse striæ in the cuticle, otherwise smooth save for corrugation of the cuticle from contraction of subjacent muscular layer. Cephalic extremity club-shaped, united to the body by a thinner portion—the neck; it is provided with a simple buccal orifice, terminal, circular, unarmed, without papillæ; the tail is simple, and ends in a blunt point. Œsophagus cylindrical, narrow, widens at terminal portion into a bulb, a contraction or kind of nipping separates it from intestine, the initial portion of which shows wide dilatation; terminal portion of intestinal

[1] *Revista dos Cursos theoricos e praticos da Faculdade de Medicina de Rio de Janeiro*, No. 3, Anno iii. 1886.

tube narrower than rest, forming rectum; anus on ventral surface near caudal end. Genital orifice close to head; the two ovarian tubes very long, tortuous, occupying almost entire extent of body of worm, full of ova and embryos. Thickness of body, 0·18 mm., gradually diminishing to 0·61 mm. Total length of body, 155 mm. Cuticle horny, very elastic. Muscular system formed by bundles of elongated cells in the shape of fibres placed longitudinally; absence of muscular bundles in lateral portion of worm easily seen, and constituting lateral spaces or lines running from head to tail; in the middle of each lateral space can be seen a line, probably representing a longitudinal canal of water vascular system; in the spaces there is a finely granular matter, with nuclei symmetrically disposed. Dorsal and ventral lines cannot be made out. At cephalic extremity, in addition to longitudinal muscular bundles, are also oblique bundles which converge from periphery to buccal orifice.

Measurements of Female Worm.

	Millimetres.
Total length (worm in two fragments),	155·0000
Thickness of cephalic bulge,	0·3315
,, cuticle,	0·0153
,, neck, 0·612 mm. from mouth,	0·2850
,, body, 0·5830 mm. from narrowest part of neck,	0·5300
,, body on level of vagina,	0·5830
,, body two centimetres from mouth,	0·7150
,, cuticle at this point,	0·0220
,, body in the anterior third beyond this point,	0·7000
,, cuticle at this point,	0·0330
,, middle third of body,	0·6625
,, cuticle of middle third,	0·0371
,, posterior third,	0·6095
,, cuticle,	0·0530
Diameter of buccal orifice,	0·0046
Distance of genital orifice from mouth,	2·5600
Thickness of tail, 0·053 mm. from extreme end,	0·1696
,, cuticle here,	0·0159
,, tail at level of anus,	0·2385
,, cuticle here,	0·0106
,, tail, 0·361 mm. from extreme point,	0·3339
,, tail, 0·5565 mm. from extreme point the level of doubling of ovarian tubes,	0·3388
,, cuticle here,	0·0157
Distance of anus from tip of tail,	0·1325
Length of oblique anal passage in musculo-cutaneous structure,	0·0159
Diameter of anal orifice,	0·0053
Length of rectum obliquely directed to dorsal margin,	0·4505
Thickness of rectum,	0·0371
,, ovarian tubes at their termination,	0·0848
,, ,, in middle third-empty,	0·3445
Diameter of intestinal tube at same level,	0·0971
Breadth of lateral spaces at same level,	0·1272
Diameter of central canal in lateral spaces,	0·0053

Male Filaria (Fig. 65).—Cylindrical, capillary, white opalescent, body apparently uniform in thickness, but in reality attenuating slightly towards

tail; cuticle presents fine, transverse striæ, otherwise smooth; cephalic end rounded and simple, presenting no intumescence nor any narrowing as of a neck, but becoming continuous with the body without presenting any line of division; the mouth is terminal, simple, circular, and unarmed; the pharynx has a cylindrical cavity: in the portion adjoining the mouth the muscular layer is thicker, giving rise to a bulbous aspect; œsophagus cylindrical and narrow, terminating in a dilatation, and separated from intestine by a constriction. Terminal portion of intestine forms a narrow rectum opening out into a cloaca common to the intestinal and genital system, and lying on the ventral surface 11 mm. from the tip of the tail. From this orifice projects a portion of a spicule enveloped in a sheath. There are four pairs of præanal papillæ, and four post anal; of the latter the two last are diminutive; the papillæ have a villous appearance. The tail is not so blunt as that of the female worm, and describes one and a half to two spiral turns. The lateral lines or spaces are very perceptible, and are 0·008 to 0·007 mm. in width; a median line probably indicates the central canal; the spaces extend from head to tail; they are formed of a granular substance and nuclei, refracting light strongly, and disposed alternately and symmetrically in two or three rows on either side of the median canal. The ventral and dorsal lines are imperceptible. The muscular system is made up of bundles of longitudinal fibres running the length of the body everywhere except at the lateral spaces. At the cephalic end there is a system of arched and converging fibres. In the tail can be seen two small, deeply situated, muscular bundles starting from the root of the spicule; one bundle is directed forwards the other backwards; they are lost in the muscles at the margin of the body, and must represent the retractores and protractores penis. The latter, enveloped in its covering, projects through a point on the ventral surface 0·11 mm. on the proximal side of the point of the tail, and can be exactly represented by the arc of a circle 0·23 mm. in diameter; it measures 0·17 mm. in length. The seminal tube appears to be simple and sinuous. The entire worm is 83 mm. long, varying in thickness from 0·40 to 0·28 mm.

Fig. 65.—Caudal extremity, showing spicules and papillæ of Magalhães' filaria. (After Magalhães.)

Measurements of Male Worm.

			Millimetres.
Length,			83·0000
Thickness of anterior third,			0·4070
,, cuticle here,			0·0220
,, middle third of worm,			0·3080
,, cuticle here,			0·0210
,, posterior third of worm,	first measurement		0·2862
,, the cuticle here,	first measurement		0·0212
,, posterior third,	second ,,		0·2420
,, the cuticle here,	second ,,		0·0159
,, just before the last turn of tail,			0·2544

	Millimetres.
Thickness of cuticle here,	0·0159
„ curved part,	0·2310
„ the cuticle here,	0·0106
„ tail at last papilla,	0·1570
„ the cuticle here,	0·0140
„ tail at cloacal opening,	0·1210
„ cuticle here,	0·0110
„ tail at extreme end,	0·0424
„ the cuticle here,	0·0110
Præanal papillæ, 1st pair, length,	0·0318
„ „ thickness,	0·0159
„ 2nd pair, length,	0·0318
„ „ thickness,	0·0123
„ 3rd pair, length,	0·0318
„ „ thickness,	0·0123
„ 4th pair, length,	0·0318
„ „ thickness,	0·0123
Postanal papillæ, 1st pair, length,	0·0110
„ „ thickness,	0·0092
„ 2nd pair, length,	0·0192
„ „ thickness,	0·0110
„ 3rd and 4th pair, length	0·0092
„ „ thickness,	0·0055
Width of lateral space,	0·0075
Diameter of buccal orifice,	0·0051
„ pharynx,	0·0583
„ œsophagus,	0·0440
„ terminal dilatation of œsophagus,	0·0848
„ „ constriction of œsophagus,	0·0530
Length of œsophagus,	0·9900
Diameter of initial dilatation of œsophagus,	0·1060
Distance of spicule base from end of tail,	0·1100
Length of spicule projecting beyond cloaca,	0·0220

The reader will perceive one very marked discrepancy between Magalhães' description of the male worm found by Saboia, and Bourne's description of the male worm found by Sibthorpe; Bourne says that the specimen he describes had two spicules, whereas Magalhães mentions only one. In a letter recently received from Magalhães, however, he informs me that, on re-examining his preparations of Saboia's parasite, he has come to the conclusion that there are really two spicules; the one spicule, having been superposed on the other, concealed it to a certain extent, giving rise to the appearance of a single spicule with a double outline.

Their Movements and Viability.—The parent filariæ possess considerable activity, the male especially exhibiting a great tendency to coil. One specimen, a female, which I saw on the cut surface of an amputated lymph scrotum, looked like an animated thread of catgut, or a thick, white horse-hair, wriggling about on the bloody, lymphous surface. Bancroft kept some specimens alive for a whole day.

COMPARISON OF DESCRIPTIONS OF FILARIA BANCROFTI.

	COBBOLD.	LEWIS.[3]	MANSON,[3]	MAGALHÃES.
Body,	Capillary, smooth.	Smooth, without striæ.	Smooth, without striæ.	Filiform, smooth, without striæ.
Colour,	White.	White.	White.	White.
Shape,	Of uniform thickness, but figure attenuated towards tail.	Thickness not quite uniform.	Thickness not quite uniform.	Not quite uniform.
Head,	Club-shaped.	Club-shaped.	Club-shaped.	Club-shaped.
Neck,	Narrowish, having $\frac{1}{2}$ of thickness of body.	Having $\frac{1}{1\frac{1}{2}}$ inch (0·54 mm.).	Neck?	Having 0·285 mm., nearly $\frac{1}{4}$ of thickness of body.
Tail,	Not doubled, obtuse (blunt).	.....	.....	Not doubled, obtuse.
Mouth,	Simple, circular, terminal, destitute of papillæ.	Terminal, simple, without labial divisions.	Simple, terminal, circular.	Simple, terminal, circular, unarmed, without papillæ.
Œsophagus,	Cylindrical, terminating in a dilatation.[1]	Cylindrical, continuous with intestine without line of demarcation.	.....	Cylindrical, ending in a dilatation.
Intestine,	Begins by a dilatation.[1]	Without dilatation?	.....	Begins by a dilatation.
Anus,	Immediately above the end of tail.	.....	.....	Near the end of tail.
Genital orifice,	Near the mouth, $\frac{1}{10}$ inch distant.	.....	Near the mouth, $\frac{1}{5\frac{1}{2}}$ inch distant.	Near the mouth, 2·56 mm. distant.
Ovarian tubes,	Double.	Double, $\frac{1}{2\frac{1}{2}}$ inch (0·123 mm.).	Double, $\frac{1}{7\frac{1}{5}}$ inch (0·127 mm.).	Double, from 0·084 to 0·344 mm.
Length of worm,	3½ to 4 inches.	(Fragment, 1½ in.)	(Fragment, 2 in.)	155 mm.
Thickness,	$\frac{1}{35}$ inch (0·28 mm.).	$\frac{1}{1\frac{1}{5}}$ inch (0·254 mm.).	$\frac{1}{1\frac{1}{2}}$ inch (0·211 mm.).	0·715, 0·662, 0·609 mm.
Habitat,	Hydrocele of cord;[2] lymphatic abscess.	Coagulum in elephantoid tumour.	Lymphatic vessel. Lymph-scrotum.	Left ventricle of heart.

[1] According to figure.

[2] My impression is that it was found in a dilated lymphatic of the cord which simulated, but which was not true encysted hydrocele.

[3] Both Lewis's and my own description were made from fragments of the parasite only; hence their incompleteness.

The Habitat of the Parent Filaria is proved to be, in the majority of instances, if not in every case, the lymphatic system. The proofs of this are numerous—1st, the free embryos are found in chyle and lymph; 2nd, they are found there in some cases in which they cannot be found in the blood; 3rd, the ova have been found in the lymph, and must have come from a parent worm lying in the lymphatics, as the ova are mere passive bodies, unable of themselves to work their way through the walls of vessels; 4th, the parent worms themselves have been found in lymphatic dilatations and in lymphatic vessels; 5th, the diseases with which the filaria is associated are diseases of the lymphatic system.

Intermediary Host.—As no evidence of growth or development can be seen in the embryo in its hæmatozoal stage; and as it seems powerless to advance in these directions while in the human body, or even to quit the body; it was assumed that the services of an intermediary host are required, first, to pick it out of the cir-

culation, and, afterwards, to foster it for a time and until it shall acquire a certain degree of maturity and independence. Proceeding on this supposition, I examined, in 1877, a number of suctorial insects which had fed on a filariated man. I found that the parasite was imbibed by all of them, and digested by most. I also found that in a certain species of mosquito it was not usually digested, but that lying in the tissues of the insect it gradually underwent developmental changes, eventuating in enormously increased bulk, great muscular power and activity, and a well-defined alimentary system; that the mosquito, in fact, is the intermediary host of *F. nocturna*.

It is possible that several species of mosquito are efficient intermediary hosts; but, certainly, all species are not qualified for this *rôle*. Myers states that in Formosa the mosquitoes there are not effective in this respect; and it will probably be found that in districts in which mosquitoes abound, but in which the inhabitants are not liable to harbour *F. nocturna*, the mosquitoes are of this description.

The particular species of mosquito with which I experimented, and which I found an efficient intermediary host, is a small, brown, unstriped insect about $\frac{3}{16}$ inch in length.[1] It is, like the filaria, nocturnal in its habits; and it thrives best when the temperature of the air is between 75° and 84° F. The male insect, owing to the peculiar structure of its proboscis, is not a blood-sucker; the female alone possess the power to pierce the skin and imbibe the blood. On filling herself with blood, she retires to some shaded spot near water, on which, after from three to five days, she deposits a little boat-shaped agglomeration of eggs. She then dies, either on the water, or falls into it after death.

By feeding this species of mosquito on the blood of a filaria infested man, and afterwards dissecting a series of the filaria charged insects at increasing intervals from the time of their feeding, the metamorphosis of the parasite can easily be made out. My observations thereon have been confirmed in great part by Lewis,[2] Sonsino,[3] Silva Araujo,[4] and Bancroft,[5] by similar observations on filariated mosquitoes; and also indirectly, but in a very remarkable and convincing manner, by Grassi[6] in his study of the

[1] More fully described by Myers, *loc. cit.*

[2] *Proc. Asiatic Soc. Bengal*, March 1877, p. 89; and *Fourteenth Annual Report of the San. Com. with the Gov. of India*, 1878.

[3] *Med. Times and Gaz.* May 27, 1882, p. 554.

[4] Referred to by Magalhães, *loc. cit.*

[5] *Scientific Lectures.* Brisbane, 1879.

[6] *Centralbl. für Bakter. u. Parasit.* Band vii. No. 1, 1890.

metamorphosis of *F. recondita* of the dog in the flea. *F. recondita* is a hæmatozoon in many respects closely resembling *F. nocturna*; the steps of the metamorphoses of these two parasites in their respective insect intermediary hosts are strikingly similar.

To describe fully the steps of this metamorphosis would occupy too much space; those interested in the subject, if they have not the opportunity to follow them out for themselves in the mosquito, will find them fully detailed and illustrated in the *Transactions of the Linnean Society of London*.[1] Briefly, the principal steps are as follows:—The mosquito after piercing the skin with its proboscis, pumps the blood containing filariæ into its stomach. Arrived there (Fig. 66, *a*), the parasite, after casting its sheath (Fig. 66, *b*), penetrates, by means of the formidable cephalic armature I have already described (p. 749), the walls of the stomach, and works its way in the course of a very few hours into the thoracic tissues of the insect. In the thorax it gradually passes into a sort of passive, chrysalis condition, in which all movement, except an occasional flicking motion of the caudal end, is suspended (Fig. 66, *c*, *d*, *e*, *f*, *g*, *h*). During this passive state the body grows thicker; and a four-lipped mouth and an anus become visible,—an alimentary line running along the centre of the body and connecting the two (Fig. 66, *i*, *j*, *k*, *l*, *m*, *n*). When this has been completed the little animal begins to grow rapidly, at first in breadth but afterwards in length, acquiring, by degrees, as it stretches out, great strength and activity (Fig. 66, *o*, *p*, *q*, *r*, *s*), and a peculiar three-lobed caudal appendage (Fig. 66, *t*). About the end of the sixth day of its stay in the mosquito the filaria has become a formidable looking object (Fig. 66, *u*, *v*), as seen through the microscope. It is just visible to the naked eye, measuring $\frac{1}{16}$ inch in length by $\frac{1}{850}$ inch in breadth. If a little water is added to the slide on which it is lying it becomes prodigiously active, seeming to be suitably equipped, both as regards strength and structure, for a temporarily independent life in water, and a subsequent journey through the tissues of a human host. The conical head is well adapted for boring its way onwards. The lobed arrangement at the caudal end (Fig. 66, *t*) suggests by its shape that it is intended as a foot to push against; a foot which, on being drawn after the animal advancing through the tissues of the definitive host, would readily collapse and offer no impediment to progress; but which, on being thrust backwards, would open up like a tripod and afford an admirable base to push forwards from.

When the filaria has arrived at this stage in its development, it

[1] 2nd series, "Zoology" vol. ii. part 10, p. 367.

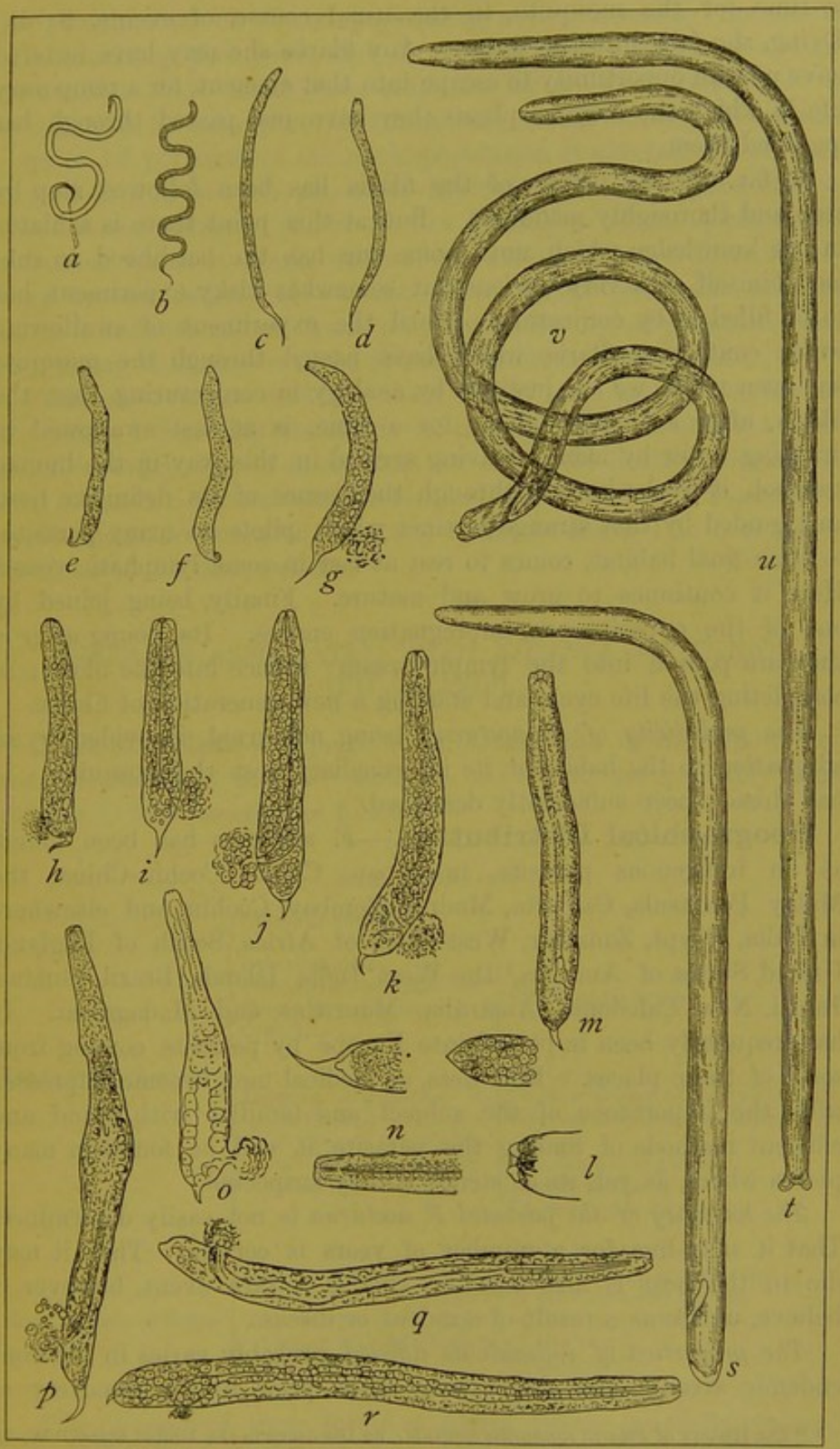


FIG. 66.—Metamorphosis of *Filaria nocturna* in the mosquito.

is time for the mosquito, in the usual course of events, to die. Dying, she falls into the water. Any filariæ she may have fostered have now an opportunity to escape into that element, for a temporary life in which the metamorphosis they have just passed through has qualified them.

So far the life history of the filaria has been followed step by step and thoroughly made out. But at this point there is a hiatus in our knowledge which, until some one has the hardihood to subject himself to a very obvious but somewhat risky experiment, has to be filled in by conjecture. Until the experiment of swallowing water containing filariæ which have passed through the mosquito has been made, we are justified by analogy in conjecturing that the filaria, after swimming about for a time, is at last swallowed in drinking water by man. Having arrived in this way in the human stomach, it works its way through the tissues of its definitive host, and, guided by that strange instinct which pilots so many parasites to their final habitat, comes to rest at last in some lymphatic vessel. Here it continues to grow and mature. Finally, being joined by one of the opposite sex, impregnation ensues. Its young after a time are poured into the lymph stream; thence into the blood; so completing the life cycle and starting a new generation of filariæ.

The periodicity of F. nocturna, being nocturnal, is evidently an adaptation to the habits of its intermediary host the mosquito. It has already been sufficiently described.

Geographical Distribution.—*F. nocturna* has been found, as an indigenous parasite, in Japan, China, Cochin-China, the Malay Peninsula, Calcutta, Madras, Bombay, Cochin, and elsewhere in India, Egypt, Zanzibar, West Coast of Africa, South of England, United States of America,[1] the West India Islands, Brazil, Guiana, Tahiti, New Caledonia, Australia, Mauritius, and Madagascar. It has frequently been imported into Europe by patients coming from some of these places. Doubtless, as medical men become impressed with the importance of the subject, and familiar with rapid and efficient methods of finding the parasite, it will be found in many places where, as yet, its existence is only suspected.

The longevity of the parental F. nocturna is not easily determined. That it may live for a number of years is certain. That it may die in the host is also certain; this is a rare event, however, I believe, unless as a result of accident or disease.

The proportion of individuals affected probably varies in different endemic areas. The observations made prior to the discovery of

[1] "The History of *Filaria sanguinis hominis:* its Discovery in the United States," W. M. Mastin, M.D., *Annals of Surgery*, November 1888. Also Osler's *Practice of Medicine*, 1893.

the phenomenon of filarial periodicity were necessarily inaccurate as determining this point, and no very extensive series of similar observations have since been made, so far as I am aware. It is certain that in some parts of China and Brazil—Amoy and Bahia—quite 10 per cent. of the adult population is affected. I venture to predict, that in such places as Shertullay in Travancore, Samoa, Huahine and others of the South Sea Islands, in which elephantiasis and other forms of filaria disease are extensively prevalent, nearly every second individual—except those whose lymphatic systems are in a measure cut off from the circulation by elephantiasis, and very young children—will be found to harbour this parasite.

From observations made on blood, kindly sent me by Dr. Elcum, of Cochin, I find that the filaria is present there in about 1 individual in every $3\frac{1}{2}$. I found it in 20 out of 74 healthy individuals from whom Dr. Elcum sent me blood slides. If those affected with elephantiasis are included, the ratio would be about 1 in every 4,—21 times in 88 observations.

DISEASES ASSOCIATED WITH *FILARIA NOCTURNA* (FILARIASIS).

Classification.—I propose to classify the diseases most frequently associated with *F. nocturna*, and which are probably attributable to this parasite, as follows:—

FILARIA DISEASE.

- Elephantoid disease.
  - Forms of abscess.
  - Lymphangitis. Dermatitis and cellulitis. Forms of erysipelas. Orchitis. — Elephantoid fever.
  - Diseases originating in obstruction, followed by varix of lymphatics and compensatory anastomosis.
    - Chyluria.
    - Chylous dropsy of the peritoneum.
    - Chylous dropsy of the tunica vaginalis.
    - Varicose groin glands.
    - Lymph scrotum.
    - Other lymphatic varices.
- Elephantiasis arabum.
  - Diseases originating in obstruction, followed by occlusion of lymphatics without efficient anastomosis, . .
    - of the lower extremities.
    - of the scrotum and prepuce.
    - of the vulva and clitoris.
    - of the upper extremities.
    - of the breast.
    - of other parts.

Two or more of these forms of filaria disease generally concur in the same individual. There are cases, however, in which only one

of them is present. For this reason, and because I think that by so doing the clinical picture will be more complete and more easily drawn and comprehended, I shall describe each of these diseases separately. The reader must understand, however, that in nature there is no hard and fast line between the types; and he must bear in mind that, as I have said, they frequently concur, and, also, that one type frequently gradually runs into another.

Relative frequency. — Although there are some very valuable statistics dealing with *Elephantiasis arabum*, there are no statistics, so far as I am aware, dealing with the relative frequency of the different filaria diseases to each other, or as a whole. Judging from my own experience, I would say that of all the filaria diseases elephantiasis of the legs is the form by far the most frequently met with, and is very common in the endemic areas. Next in frequency I would rank varicose groin glands, then lymph scrotum, and perhaps orchitis; these are fairly common. After them I would place elephantiasis of the scrotum. Chyluria is not a common disease, even in the tropics; although one is apt to gather an opposite impression from books dealing with the subject. Chylous dropsy of the tunica vaginalis is equally rare; in large native hospital practice two or three cases of each of these diseases may on an average be encountered in a year. In the course of a large experience I have seen elephantiasis of the female genitals only once; of the arm, also only once. Although in the South Sea Islands, in parts of India, in Madagascar, and in Brazil such cases are not uncommon, in China they are very rare. I have never met there with a case of elephantiasis of the mamma, head, ear, or of this disease in any other situation than in the leg, scrotum, arm, and female genitals; neither have I ever seen a case of chylous dropsy of the peritoneum. Abscess of filarial origin is, I believe, fairly common; whilst lymphangitis and elephantoid fever, although sometimes occurring alone, are present in nearly every case of filarial disease at one time or another in its progress.

Filarial Abscess.

Abscesses frequently form in scrota and legs affected with elephantiasis. Such abscesses are, as a rule, probably nothing more than ordinary phlegmons, and what one would expect to meet with as the result of injury or inflammation of lowly organised and semi-diseased tissues. They are not directly attributable to the filaria. But in districts in which this parasite is endemic, abscess of originally apparently healthy tissues, and occurring without any

very obvious cause, is not uncommon, and, I believe, in many instances is due to the irritation set up by a dead *F. Bancrofti.* The so-called lymphatic abscess of the arm in which Bancroft first found the parent worm, and the similar abscess in which dos Santos [1] found the same parasite, were evidently of this nature. I was once led to make search for a parent filaria in the contents of an abscess of the thigh, which I had opened in a Chinaman, by observing that the patient had well-marked filarial varicose groin glands; and my diagnosis of "filarial abscess" was confirmed by the discovery of the disintegrating body of a female filaria in some grumous, dark red clots, which were evacuated with the pus. In Mackenzie's chyluria case, presently to be cited, the formation of abscess about the shoulder was so intimately related in time to evidences of the death of the parent filaria, that it is more than probable that the dead body of the worm was the cause of the abscess. Of the irritating properties of the bodies of dead parasites, and of their extravasated young, we have ample evidence in what happens in the case of the guinea-worm.

If abscess occur without obvious reason in a patient with signs of filaria infection, or in whose blood the parasite is known to be present, there is good ground for supposing that the abscess may be the result of the death of a parent filaria, and search should be made in the contents of the abscess for the worm. If this is done systematically, I have no doubt that in a proportion of cases the diagnosis "filarial abscess" will be borne out by the discovery of the remains of the parasite.

Filarial abscess in the limbs is of little gravity if freely incised and treated antiseptically. But filarial abscess arising from death of the worm in the deep lymphatics of the thorax and abdomen is an occurrence of the gravest character. It proved fatal to Mackenzie's case; and, doubtless, similar mishaps are not infrequent in filarial districts.

Lymphangitis, Dermatitis, Cellulitis, and Elephantoid Fever.

In almost all the filaria diseases, at one time or another, the patient suffers from what Sir Joseph Fayrer has happily termed "elephantoid fever." Some cases suffer frequently from this fever, others, again, only rarely; it is not very frequent in chyluria, it is very frequent in the early stages of elephantiasis. Cases differ much in this respect. The fever depending on lymphangitis, and this, in its turn, depending on a variety of circumstances,—the

[1] Magalhães, *loc. cit.*

principal among which is the degree of liability to different forms of traumatism,—it is evident that the frequency of recurrence of the fever will depend very much on the nature of the patient's occupation, the degree of exposure he has to submit to, the character of his clothing, his general health, and so forth.

Both Waring[1] and Vincent Richards[2] give some statistics bearing on the frequency of the recurrence of the attacks of elephantoid fever in elephantiasis.

Febrile paroxysms occur.	Waring (Travancore).	Richards (North Orissa).
Once a month,	38	125
Twice a month,	36	243
Three times a month,	17	24
Four times a month,	6	21
Five times a month,	5	...
Once in two months,	10	49
Once in three months,	24	44
Once in four months,	10	21
Once in five months,	3	...
Once in six months,	9	41
Once in seven months,	2	...
Once in twelve months,	8	33
Once in twenty-four months,	1	...
Irregular,	43	12
Had only one attack,	4	...
No fever for one year,	2	...
No fever for two years,	2	...
No fever for three years,	1	...
No fever for fifteen years,	2	...
No fever for ten years,	1	...
Had no fever,	...	22
Not mentioned,	...	1
Total,	224	636

There has been some discussion as to the order of the relationship between the fever and the lymphangitis—whether the fever precedes and is the cause of the lymphangitis, or the lymphangitis precedes and is the cause of the fever. With our present knowledge of the pathology of these diseases there can be little doubt that the lymphatic affection precedes and is the cause of the fever. This view is further confirmed by direct observation of cases; and by the fact, frequently observed, that in the case of scrotal elephantiasis cessation of the febrile attacks ensues on the thorough removal of the tumour; in other words, the seat of the lymphangitis being removed, fever can no longer recur.

[1] *Indian Annals of Medical Science*, No. ix. 1858.
[2] *Ibid.* No. xxx. 1873.

Although elephantoid fever rarely endangers life it is often of very great severity; and the attendant affection of the lymphatics, together with the febrile headache, prostration, thirst, and vomiting, are very distressing. The lymphangitis appears to commence sometimes in the abdominal or pelvic lymphatics, and to spread downwards; at other times it appears to originate in some trifling wound of the integument, and spreads upwards. Prolonged standing, violent exercise, friction of the scrotum against the thighs, scabies and the attendant scratching, bites of insects, sun erythema, standing in wet rice fields or salt water, wounds from thorns, and so forth, are some of the most frequnt causes of its explosion.

When the attack is fairly established the lymphatic trunks are hard and cord-like, very tender to the touch or on movement, their course being marked out by a red streak of superficial congestion. The groin glands become swollen and tender, and there is much aching in the loins and back. The integuments of the implicated area inflame as a rule, becoming swollen, heavy, and tense from accumulating effusion. When the scrotum, testes, or cord are implicated, much suffering is produced by a powerful and oft repeated spasm of the cremaster muscle, dragging the inflamed testis up into the inguinal canal. Severe rigors, simulating the rigors of ague, for which they are very often mistaken, usher in the fever, and may continue to shake the patient for several hours. The succeeding pyrexia is apt to run high,—the thermometer rising to 105° or 106° F.,—and be attended with delirium. It is usually accompanied by severe headache, intense thirst, very often vomiting, and much general distress. After a day or two of suffering, profuse diaphoresis sets in. This is sometimes accompanied by weeping of a lymphous or chylous fluid from the implicated leg or scrotum, whereby the tension of the inflamed parts is much relieved, and the attack, for the time being, comes to an end. Rigor, pyrexia, and diaphoresis may recur after a few hours of comparative comfort, and this for three or four times; but, as a rule, there is only a single paroxysm, unless abscess has formed, in which case paroxysm follows paroxysm until the abscess is opened or discharges spontaneously.

Elephantoid fever simulates very closely ordinary intermittent fever. As the two forms of disease are often co-endemic, it is not surprising that they are frequently mistaken one for the other. Apart from the local signs accompanying lymphangitis, elephantoid fever is further distinguished by the great length of its constituent stages; by the fact that it observes no marked periodicity; that it is not accompanied by enlargement of the spleen, nor followed by the profound anæmia so constantly resulting from malarial affections.

Further, there is no reason to suppose that elephantoid fever is directly controlled by quinine and other antiperiodics.

It has frequently been remarked, especially by the earlier

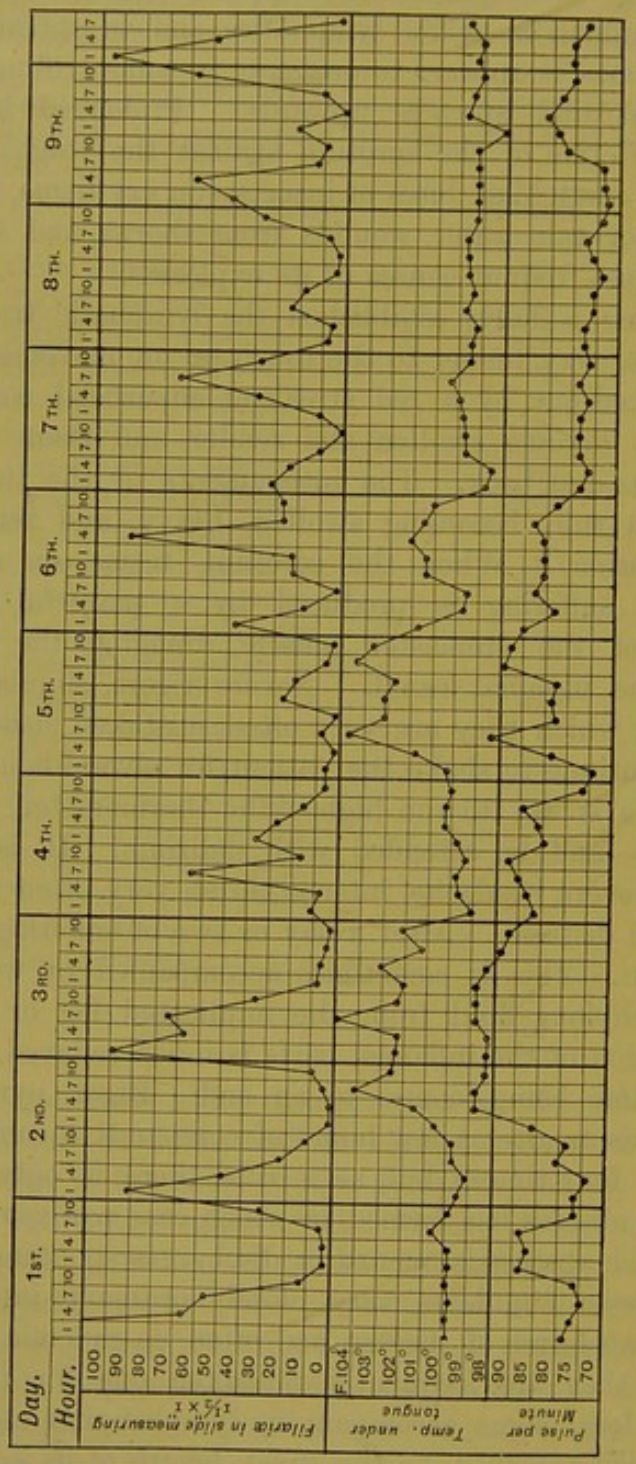


FIG. 67.—Chart showing the temperature and pulse curves in a case of elephantoid fever and filarial orchitis. The effect of fever in disturbing filarial periodicity is also recorded. Observations were made every three hours, and the filariæ in a fixed quantity of blood enumerated.

writers on the diseases of India, that the accessions of elephantoid fever are in some way related to the recurrence of the lunar changes; and this belief is very prevalent at the present day among the

natives. Dr. Allan Webb[1] frequently refers to it as "Moon Fever"; and other writers, some of them recent, more than hint at some such relationship as this expression implies. Fayrer,[2] who himself has no such belief, remarks of the strong conviction entertained by the natives on the subject, that "in many cases you might as well try to convince the person that he had no disease at all, as that it was not so determined." Francis Day[3] made some careful observations with the view of arriving at a decision on the subject; from these he concluded that there was no connection whatever between the moon and elephantoid fever. In one severe case which he had under continuous observation for six months, he noted that the first paroxysm of each successive attack occurred at the following irregular and non-cyclical intervals,—twenty-five, nineteen, forty, sixteen, twenty-eight, nine, and twenty-two days. The Chinese, so far as I am aware, do not associate elephantoid fever in any way with lunar influences; the superstition, for such it seems to be, is confined to India.

Forms of Erysipelas.

Most attacks of lymphangitis and elephantoid fever are associated with a degree of erythema of the implicated part; very generally the subcutaneous tissues are involved as well as the skin. As a rule, no suppuration results; but at times, especially when there are malarial complications, abscesses may form, or the parts become gangrenous or slough. In such cases forms of blood poisoning are apt to supervene and prove fatal.

Brazilian physicians describe several types of erysipelas which are, in a measure, dependent on a lymph stasis possibly of filarial origin. Rio de Janeiro appears to enjoy an unenviable notoriety in this respect. Bad drainage, and bad sanitation generally, may have a good deal to do with these cases by fostering germs of a septic character, which find a ready-made soil in the tissues of filariated individuals, so frequently affected with chronic lymph stasis. The physicians of Rio recognise two forms of this type of erysipelas; a *circumscribed form* which, from the descriptions given, seems to be ordinary elephantoid cellulitis; and a *diffuse or erratic form.* Dr. Rego arranges the cases of the latter type into three groups—(*a*) *spreading erysipelas* of the ordinary type: (*b*) *erratic erysipelas*, in which one attack follows on another in the same or in a different

[1] *Indian Annals of Medical Science*, No. iv. April 1855.
[2] *Tropical Diseases.*
[3] *Madras Journal of Medical Science*, vol. i. 1860.

region of the body; and which, if associated with grave typhoid symptoms, is very fatal; (*c*) *spreading cellulitis*, which, after a time, attacks the joints, giving rise to the appearance of acute rheumatism. This is also a very dangerous type. Mazaé Azéma describes similar forms of malignant erysipelas as occurring in Réunion. These malignant forms are not common, apparently, in India, the South Sea Islands, China, or other centres of elephantoid disease.

Orchitis.

The frequency of orchitis among the inhabitants of certain districts in warm countries—orchitis unconnected with gonorrhœa, stricture, and the usual causes of secondary inflammation of the testis—has often been remarked. It has very generally been attributed to malaria; doubtless, in many instances, for no better reason than that it occurs in malarious countries, and because the elephantoid fever with which it is associated resembles, in respect of the pronounced rigor, pyrexia, and diaphoresis, an ordinary ague. French writers have described a form of orchitis which appears really to own a malarious origin; but there is certainly a second group of cases of endemic orchitis which are quite independent of such a cause. Cases occur in countries—New Caledonia, for example—which are practically free from malaria; they are also common in many hot countries where malaria may or may not be prevalent, and are evidently unconnected with malarial poisoning. Such cases are reported from New Caledonia,[1] Brazil, Guiana, Guadaloupe, Martinique, Cochin-China, India (Fayrer, Chevers, Maitland), and I have frequently met with them in China, and, doubtless, they are found in other hot countries. In an interesting paper in the *Indian Medical Gazette*,[2] J. N. M., Berhampore, states that in certain districts of Bengal a large proportion of the inhabitants are subject to this form of recurring orchitis, and that it is attributed by them to lunar influences such as, according to native ideas, dominate elephantoid manifestations generally.

The symptoms are those of a rapidly developed orchitis, the attendant fever having all the characters of elephantoid fever—sudden onset, violent and prolonged rigor, high fever, profuse diaphoresis, a duration of over twenty-four hours, a possibility of one or two relapses after slight remission, absence of both splenic enlargement and anæmia. The testicle swells rapidly, inflammation usually involving epididymis and cord; clear or chylous effusion may be thrown out into the tunica vaginalis, and there may be some

[1] Valence, *Arch. de méd. nav.* Jan. 1890.

[2] Vol. i. p. 213.

thickening and inflammatory puffiness of the scrotum. Pain and tenderness are extreme, and there may be a good deal of vomiting, headache, and malaise. With the disappearance of the fever the pain rapidly subsides, and by degrees the swelling of the testis and cord, and the effusion are partly or wholly absorbed. If the fluid in the tunica vaginalis be clear, that, too, is generally absorbed; but if it be chylous, absorption may not take place; indeed, such attacks are frequently the first remarked event in the development of a permanent chylous dropsy of the tunica vaginalis. Similar attacks recur at longer or shorter intervals, and not infrequently are associated with or take the place of other inflammatory manifestations of filaria disease. The patient, whose chart I have given at page 780 in illustration of the temperature curve of elephantoid fever, was at the time of the observations there recorded the subject of this form of orchitis. This was his first attack, but he had been subject almost monthly and for many years to attacks of a similar fever attended with inflammation of the inguinal glands, of which, apparently, the orchitis on this occasion had taken the place.

More extensive and more accurate observations are very much wanted on this form of orchitis. I believe it to be attributable to *F. nocturna*. It is certainly often concurrent with the presence of this parasite in the blood, and it is intimately associated with well recognised forms of filaria disease. Bancroft's find of mature filariæ, in what he describes as an "hydrocele of the spermatic cord," is significant in this connection; I would suggest that this "hydrocele" was most probably a dilated lymphatic. Occlusion of lymphatic vessels in this situation would certainly tend to lymph stasis in the testicle, and strongly predispose to orchitis. Valence rejects the theory of the filarial origin of this form of endemic orchitis, as he does the theory of the filarial origin of endemic lymphatic varix; he attributes varices and orchitis to Corre's "lymphathexie," but he adds, "Il est vrai que nous n'avons examiné ni le sang, ni la lymphe de nos malades."

Moty has recently recorded[1] two interesting cases of what he considered to be, and which, doubtless, were, filarial lymphatic varix of the spermatic cord. The very considerable swelling, which ran from the inguinal canal to the bottom of the scrotum, resembled a hernia—indeed in both cases it was considered to be such. Under this impression M. Moty proceeded to perform the operation for radical cure on one of the cases, and it was not until cord and testicle had been fully exposed that he discovered its true nature. He found several soft cysts on the surface of the testicle, which was

[1] *Revue de Chirurgie*, Jan. 1892.

much enlarged, and great thickening of the cord extending as far up the inguinal canal as the finger could follow it.

Chronic enlargement of the testes and thickening of the spermatic cords are very common conditions among Easterns, and are very probably, in many instances, of filarial origin, and of the same nature as M. Moty's cases just referred to.

Chyluria.

The etiology and pathology of chyluria, long a puzzle to physicians, is now thoroughly understood. There can be no doubt that the symptoms of this disease are attributable to the escape of chyle and lymph into the urine from the rupture of a filaria-induced lymphatic varix, situated somewhere in the urinary tract. Mazaé Azéma seems to have suspected that, at least in some cases, lymphatic varicosity in the urinary tract might be at the root of the pathology of chyluria; but the merit of having first definitely formulated this mechanical theory of chyluria belongs entirely to Vandyke Carter,[1] whose views have been abundantly confirmed by many subsequent observers. Wucherer was the first to point out the frequency of the presence of *F. nocturna* in chylous urine; his observations were subsequently confirmed by Crevaux, Lewis, Sonsino, myself, and others. Lewis soon afterwards showed that the filaria was also nearly constantly present in the blood as well as in the urine of these cases; and Mackenzie (p. 812), by a very valuable post-mortem examination in a case of filarial chyluria, demonstrated the varix in intimate relationship with the urinary tract in complete accordance with Carter's hypothesis. Havelhing, Curnow, and Ponfic have also made similar post-mortem examinations in cases of filarial chyluria, with results closely resembling Mackenzie's. These, and a host of observations on the living subject by other workers, have contributed to build up as complete a piece of pathology as any of which medicine can boast. The subject will be more fully discussed when the pathology of the filaria diseases is treated of collectively.

Of the many theories which, from time to time, have been propounded to explain the phenomena of chyluria, it is hardly necessary to do more than allude to them; any interest they may have for us at the present day is purely an historical one. The piarrhæmia, or fatty blood theory of Claude Bernard, Robin, and Bouchardat; the acquisition of gland properties by lymphatic channels theory of Roberts, may be mentioned. It is just possible that one or both of

[1] *Trans. Med.-Chir. Soc. Lond.* vol. xlv. 1862; *Trans. Med. and Phys. Soc. Bombay*, 1861-62.

these theories may apply to the rare cases of non-tropical chyluria which crop up at long intervals in the hospitals of Europe, but they have no place in the pathology of tropical chyluria.

Symptoms.—Chyluria attacks both sexes and any age. There is an impression that it is more common in women than in men; if this be well founded, the greater liability of women is probably attributable to the effects of gestation on the mechanism of the abdominal lymphatic circulation.

Unless the patient has been the subject of some other form of filarial disease, until the appearance of chyle in the urine, health may have been perfect. Sometimes the chyluria is preceded by a feeling of discomfort over the kidneys, about the groins, down the front of the thighs, and a dragging sensation, or pain even, may be experienced in the testes and deep in the perineum. In uncomplicated chyluria elephantoid fever is rarely a prominent symptom. As a rule, the first intimation the patient has of anything being amiss with the urinary organs is the appearance of chyle in the urine; or, possibly, the first symptom is an attack of retention of urine, presently relieved by the passage *per urethram* of reddish clots and a quantity of milky fluid.

The chylous appearance of the urine may disappear after a day or two; more frequently, however, it persists for weeks or months or even years. Occasionally it goes away for a time, often for a very long time, but it is almost certain to return sooner or later, again to disappear and again to relapse; so that a person who has once had symptoms of chyluria is liable to a recurrence of them, often without apparent reason. Without completely disappearing, the degree of milkiness or sanguinolence of the urine varies from time to time; on some occasions scarcely a trace of faintly opalescent-like clot can be discovered, whilst at other times it may be opaque, dead-white, *café-au-lait* colour, salmon colour, or almost like blood.

The general health of chyluric patients is usually fairly good; but if the chylous discharge persist for any length of time, debility, anæmia, and mental depression are sure to ensue. The disease is not directly fatal, and, unless when retention of urine results from the impaction of clots in the urethra, beyond the feeling of dragging in the loins and weakness, the patients experience no inconvenience.

The Urine.—Chylous urine when newly passed, or when heated, emits a peculiar heavy, milky, urinous odour. It is said to be very prone to rapid decomposition. The reaction, specific gravity, and amount vary in different individuals, and from time to time in the same individual. As a rule, the quantity passed is in excess of the

normal; but this is not a constant feature, and is much influenced by the amount of fluid consumed, heat of the weather, exercise, and so forth. As before mentioned, the colour of the urine, its degree of transparency, milkiness, or sanguinolence, are also subject to great variations; no two specimens passed at different times, by the same individual even, being exactly alike.

Clots are sometimes passed *per urethram*; as a rule, however, they do not form until the urine has escaped and stood for a short time. After standing a few moments, if the vessel into which it has been passed be not disturbed, the entire bulk of the urine coagulates, becoming converted into a reddish, tremulous, blanc-mange-like jelly. The coagulum at first rapidly contracts, becoming redder, denser, and more fibrous as it becomes smaller. On standing, the urine separates into three fairly well-marked zones: an upper and comparatively thin one of a white, creamy-looking material; a lower and very scanty one of small, dark clots, and dark, reddish-brown sediment; and a large central layer of white, salmon-coloured, or pinkish fluid. In the latter the contracting clot floats. Under the microscope the upper layer is found to consist principally of minute granules of fatty matter, with a few larger fat globules; the middle zone contains the same minute fat granules, but in smaller proportion, and a few lymph and reddish discoid corpuscles like those of blood; the lowest stratum contains also minute fatty granules, a larger proportion of lymph and red corpuscles, epithelium, crystals of urinary salts, sometimes a few casts, and, almost always, embryo filariæ in a more or less moribund condition. The coagulum, in addition to fat granules and both forms of corpuscles, contains also many filariæ entangled in the meshes of the fibrine.

If the urine be shaken up with ether, the fatty matter it contains is dissolved and it becomes clear. Boiling, or nitric acid throws down a copious deposit of albumen. Traces of albumen persist often after apparent recovery, and all chylous or lymphous appearances have cleared away.

Although most chemists who have analysed chylous urine agree about the nature of the ingredients, there is considerable discrepancy as to the proportions in which these are present. This discrepancy is readily explained by the varying proportions of chyle, lymph, and renal secretion in different specimens of chylous urine, even from the same individual; and also by the varying proportions of the ingredients of chyle according to the nature of the food consumed, and the time of digestion represented by the chyle in the urine of any given specimen. I do not see that it would serve any useful purpose to give these analyses in detail; suffice it to say that most,

if not all, of the published analyses, seem to be compatible with the view that chyluria is produced by a leakage from the lymphatic system into the urine.

Ordinarily the urine is least chylous in the early morning, most chylous towards the end of the day, the degree of this apparently harmonising with the nature of the food consumed, and the amount of exercise indulged in. The following table, prepared from a series of consecutive observations on a case under my care, gives some idea of these fluctuations. The table also serves to show that the presence of the filaria in the urine is not regulated by the law of periodicity governing its presence in the blood; in chylous urine it is present as frequently by day as by night.

TABLE SHOWING THE FLUCTUATIONS IN THE CHARACTER OF CHYLOUS URINE, AND THE PERSISTENCY OF FILARIA EMBRYOS THEREIN COINCIDENT WITH NORMAL INTERMITTENT FILARIAL PERIODICITY IN THE BLOOD. PRECIS OF OBSERVATIONS MADE EVERY THREE HOURS DURING A PERIOD OF SIX DAYS.

	Hour of Observation—A.M.				Hour of Observation—P.M.			
	3	6	9	12	3	6	9	12
Total quantity of urine, in ounces,	25	14½	11	55	75	90	78½	56
Total number of filariæ in six observations, one drop of sediment each,	4	7	36	43	2	26	28	3
Total number of filariæ in the blood; six observations, one slide each,	10	0	0	0	0	1	62	50
Average quantity of urine in three hours,	4⅙	2⅔	1⅚	9⅙	12⅔	15	13	9⅔
Average filariæ in drop of urine,	⅔	1⅙	6	7⅙	4	4⅔	4⅔	⅔
Average filariæ in slide of blood,	1⅔	0	0	0	0	⅙	10⅔	8⅔
Number of times urine watery and clear,	4	3	0	1	0	2	0	1
Average number of times urine chylous,	2	3	6	5	6	4	6	5

Filariæ in the Urine.—Failure to find filariæ in chylous urine, as in blood, is very often owing to the faultiness of the method of examination employed and to want of patience, and not to absence of the parasite. That the filaria is absent in a small proportion of cases is certain. The reason for this I shall point out when speaking of the pathology of the filaria diseases; meanwhile, I would remark that from this circumstance it by no means follows that the filaria is not the cause of the disease in question. In the considerable number of cases of chyluria which I have investigated from

time to time, I can only recall two or three instances in which the filaria was persistently absent. Wucherer, Crevaux, Lewis, Bancroft, and many others are unanimous in testifying to its almost invariable presence in tropical chyluria. The best way to set about finding it in the urine is to make the patient pass water into a large conical glass, and to break up the clot with a glass rod as soon as it forms. The glass is then allowed to stand for some hours, until a dark red sediment collects at the bottom of the urine. A drop of this sediment is taken up by a pipette and placed on a glass slip, between two strips of wet paper placed across the slip about half an inch apart; the cover-glass, its edges resting on the strips of paper, is then applied, and search made in the comparatively thick layer of urine with a low power of the microscpe—inch or half-inch objective. Another plan is to select a minute piece of clot, compress it, after teasing up, under a cover-glass, and search for the filariæ in the usual way. Whilst a considerable number of filariæ are expelled by the contracting clots into the fluid urine and fall to the bottom of the glass as a sediment, the larger number are retained in the meshes of the coagulum as in a net, and concentrated in proportion as this contracts. It is almost useless to attempt to find filariæ in a drop of newly passed urine, or in the upper or middle layers of urine which has stood for any length of time; they are too far apart to be likely to be found in the first case, and they have been nearly entirely removed by the contracting clot and by subsidence in the second.

Diagnosis.—There are few difficulties about the diagnosis of chyluria. It is readily distinguishable both by objective and subjective symptoms from such diseases as cystitis, pyelitis, phosphaturia, abscess opening into the urinary tract, hæmoglobinuria, and ordinary hæmaturia. It must frequently happen, however, that there arises a difficulty in diagnosis in countries such as Egypt, Mauritius, Madagascar, and some other places in which hæmaturia and chyluria are co-endemic. In these countries both diseases may concur in the same individual. Sonsino and Corre have recorded such cases. The microscope will at once clear up any doubt about the nature of such a case, by discovering the characteristic spined bilharzia ova, indicating "endemic hæmaturia"; and the equally characteristic filaria embryos, indicating chyluria.

Chylous Dropsy of the Peritoneum.

A good many cases of chylous ascites and a few of chylous dropsy of the pleura have been recorded. It may be gathered from the records that the majority of these cases depended on rupture of

the receptaculum chyli, or of the chyle vessels, or of the thoracic duct, from causes altogether unconnected with *F. nocturna*; such cases, therefore, do not fall to be considered here. There is a case recorded by Winckel[1] which may fairly be attributed to the filaria. The patient, aged 39, the widow of a missionary, had resided in Surinam for ten years. A year after returning to Europe she observed that her abdomen was tense, and that a tumour protruded from the vulva. The latter turned out to be a prolapsed uterus forced down by fluid in the abdomen. She was tapped in September 1874, and two litres of a milky fluid were withdrawn. The fluid was found to contain large numbers of small, very active, filiform entozoa—0·01 mm. by 0·2 mm.—with rounded heads and sharp tails. Soon after the tapping the left leg swelled and became tense and painful; this swelling, however, gradually subsided. The urine kept normal, and no tumour could be felt in the abdomen. In 1875 ascites returned, and in August 1876 she was again tapped, nineteen quarts of milky fluid escaping. Two days later she died suddenly in great pain, apparently from rupture of some vessel in the abdomen.

Doubtless this was a case of filarial lymphatic varix which had leaked into the peritoneum. Similar occurrences must be rare, although why this should be so is difficult to understand, considering the comparative frequency of chylous leakage into the tunica vaginalis, and the frequency of varix of the abdominal lymphatics.

Chylocele; Chylous Dropsy of the Tunica Vaginalis; Lymphocele.

This disease has long been recognised, and is referred to by all recent systematic writers on diseases of the testis. It was to this affection that Vidal applied the ill-chosen term "galactocele." It is rare in Europe, but not uncommon in the tropics, and is almost invariably associated with *F. nocturna* in the blood.

In many of these cases the swelling of the testicle is the only external evidence of filarial infection, but not infrequently it is associated with varicose groin glands, lymph scrotum, or other filarial disease.

Usually its presence is not suspected until the surgeon taps what he supposes to be an ordinary hydrocele, when, instead of the familiar straw-coloured fluid, a liquid like milk flows through the cannula. If in countries in which *F. nocturna* is endemic an opaque hydrocele-like swelling of the testis be present, and be associated

[1] *Deutsches Arch. f. klin. med.* 1876, Bd. xvii. p. 303.

with varicose groin glands, it may almost with certainty be diagnosed chylocele.

Magalhães has pointed out an important diagnostic mark distinguishing these vaginal dropsies from ordinary hydrocele; a chylocele is always less tense in the morning, and is seldom at any time so tense as ordinary serous hydrocele.[1]

Chylocele has frequently an antecedent history of filarial orchitis, or of some form of lymphangitis, or of elephantoid fever—very often mistaken for malarial fever. There are other cases, however, which give no such history, the swelling having slowly developed like an ordinary hydrocele.

According to my experience this form of dropsy of the tunica vaginalis seldom attains a great size. Sometimes, observing a slight fluctuating enlargement of a testicle in a case of varicose groin glands, I have, from curiosity about the diagnosis, tapped it with a hypodermic syringe, and found it to contain but a drachm or two of the characteristic milky-like fluid. Others, again, attain a capacity of 8 to 10 oz., and are inconvenient from their size.

As a rule, the fluid of a chylocele is white or pinkish. It coagulates rapidly after withdrawal in the great majority of instances. Strange to say, now and again a case is met with in which the fluid does not coagulate, even after standing for hours. I presume that in those cases the fibrinogen ferment, which is normally present in lymph and chyle as in blood, is for some reason absent, and that there has been no accidental admixture of blood at the time of tapping to take its place.

The fluid, which is highly albuminous, under the microscope is seen to contain much finely divided granular matter of a fatty nature, lymph and, generally, red corpuscles like those of blood, and, almost invariably, very large numbers of filariæ. In some examples these parasites are to be found in enormous numbers; they are much more numerous than in lymph from varicose groin glands or lymph scrotum. In the sediment of the fluid—especially in the sediment of the non-coagulating variety—the filariæ can be very readily found in the little shreds and clots adhering to the sides of the dish containing the fluid. Some of them are plump and active, but a few show signs of degeneration, and a few may be moribund or dead. If into the fluid of the non-coagulating variety, just alluded to, a few cotton fibres be dropped, and time be allowed for them to subside to the bottom of the vessel and to remain there for a short space before being placed under the microscope, a very striking phenomenon may be observed. The cotton fibres on

[1] *Revue de Chirurgie*, No. 4, June 1892.

inspection are seen to be beset by hundreds of wriggling filariæ entangled by their lash-like sheaths, clumps and groups of them reminding one of the pictures of the head of the Gorgon.

I would suggest the following explanation of the prodigious number of filariæ found in the fluid removed by tapping in these cases:—The chyle or lymph in chylocele is evidently derived from a ruptured lymphatic. With the chyle filariæ enter. Much of the fluid is reabsorbed by the tunica vaginalis; but as the filariæ are not capable of finding and re-entering the stomata of the serous sac and so regaining the lymphatics, they are perforce compelled to remain behind, and so gradually accumulate, caught much in the same way as salmon are in a stake net. Hence their enormous numbers; they are trapped or filtered out as it were.

I cannot find any record of a post-mortem dissection of a chylocele. Mastin,[1] in treating a case by free incision, found at the operation the tunica vaginalis dense, thickened, smooth, and pearly white. At the upper part, where it begins to be reflected over the testis, he discovered a small, round, granular-looking mass about the size of a pea. This projection he snipped off, thereby opening three or four vessels which did not bleed, which he believed to be, and which doubtless were, lymphatics.

There is no record of any parent filaria ever having been found in one of these chyloceles; but it is just possible that they might wander into it through the ruptured lymphatic and, not being able to find their way back again, remain prisoners in the tunica vaginalis.

I have sometimes thought that the very great frequency of ordinary hydrocele in tropical countries might be attributable to interference by the filaria with the free return of the lymph from the tunica vaginalis. This is merely a speculation; I have no facts to go on, further than the great frequency of hydrocele in filarial countries, and the idea that hydrocele may depend as much on defective absorption as on excessive secretion.

Varicose Groin Glands. (Fig. 68.)

A frequent feature in filarial infection—sometimes existing alone, sometimes associated with other forms of filaria disease—is the peculiar affection of the inguinal and femoral glands, to which I attach the name "varicose groin glands."

According to Mazaé Azéma,[2]—who writes of this disease as he saw

[1] *Amer. Med. Weekly*, Louisville, Ky., vol. ii. p. 617; *Annals of Surgery*, Nov. 1888.
[2] *Traité de la Lymphangite endémique des Pays chauds*, Réunion, 1879.

it in Réunion, where it is very common,—it commences insidiously, generally between the thirteenth and twentieth years, rarely later, slowly increases during four or five years, and then, save for attacks of lymphangitis, remains stationary, to subside in some cases spontaneously about forty-five. Men are more subject to it than women (in twenty cases five only were women), although pregnancy aggravates for the time being the swellings when once established.

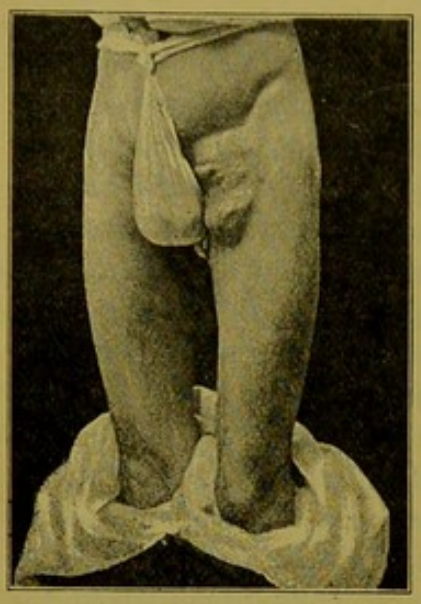


FIG. 68.—Varicose groin glands. (Photo. by Mr. Ed. Henderson.)

In a well-marked example both groins are occupied by large, obscurely lobulated swellings which, to the touch, feel baggy, doughy, semi-fluctuating, with here and there a firmer kernel-like lump. The superjacent skin is not thickened or altered in any way, and can be easily glided over the underlying tumours, which are also slightly movable. There is no actual pain unless during one of the attacks of lymphangitis which usually, though not in every case, from time to time recur. After standing for a long time, or on severe exercise or strain—particularly during very hot weather, the parts feel tense and inconveniently large and distended. By pressure with the palm of the hand, especially when the patient is made to lie down, the swelling can be very much reduced; on removing the hand it rapidly returns. There is no marked impulse on coughing, no tympanitic sound on percussion, and no gurgling or sudden slipping up of the tumour on taxis as in hernia.

In most cases the swellings can generally be roughly divided into two parts—femoral and inguinal. The former lies in the region of the cribriform fascia, and is usually oval in form, the long axis being vertical; the inguinal, usually the larger swelling of the two, follows the course of Poupart's ligament, and in well-developed cases may extend from near the spine of the ilium to the inguinal canal. Generally both sets of swelling are present; but it sometimes happens that the femoral glands alone, or the inguinal glands alone are affected. Usually both groins are affected, but it sometimes happens that the glands of one side only are involved. The consistency of the swelling varies in different cases from large, soft, semi-fluctuating masses, such as I describe, to smaller, questionably varicose, semi-indurated glands, resembling those generally met with in elephantiasis.

If a hypodermic needle be thrust into the swellings, chylous or lymphous fluid can be aspirated; and if the barrel of the syringe be laid aside, this fluid will continue to drop from the needle until a drachm or two, or many ounces even, have distilled away. Just as in chyluria or lymph scrotum, the fluid may be clear, milky, or sanguinolent. It coagulates rapidly, contains lymph corpuscle, and sometimes red discs like those of blood, and, almost invariably, filariæ, rarely filaria ova. On withdrawing the needle fluid may continue to escape through the puncture in the skin; a little pressure with the finger will stop this in a few minutes; but for some hours the swelling in the groin continues to enlarge, probably from escape of lymph into the areolar tissue from the punctured lymphatic. This rather alarming looking swelling, the consequence of the puncture, does not take long to subside; I have never seen any bad result.

Attacks of lymphangitis are common in these cases, and lymph scrotum, chyluria, or elephantiasis may in course of time supervene. Corre and Mazaé Azéma say that the varix tends to disappear spontaneously after a number of years. Should elephantiasis supervene, the varicosity gives place to induration and gradual contraction of the dilated vessels.

There can be no doubt about the nature of these swellings, although as yet post-mortem examination has thrown comparatively little light on the subject. In many instances they are but a small portion of a more extensive varix, involving pelvic and abdominal lymphatics, and arising from filarial obstruction in the thoracic duct. In such cases the fluid in the varix is usually chylous. In other instances the fluid is lymphous, and in these the obstruction is often probably in the glands themselves, and may not involve the higher lymphatics; in these latter cases the filaria, though present in the gland lymph, may not always be found in the blood.

Very few dissections have been made in this disease. Amussat[1] dissected a case in which enormous dilatation of the lymphatics of groin, pelvis, and abdomen, and of the thoracic duct was found. The enlarged groin glands had been mistaken for hernias. Similar tumours in a patient from Mauritius were mistaken in the same way for hernias by Trélat and Nélaton.[2] The patient died soon after an operation for a fistula near the anus, evidently from septic lymphangitis. At the autopsy the tumour in the right groin was found to be principally under the cribriform fascia, and was made up of closely united lobes. It was definitely defined above, below,

[1] Breschet, *Le système Lymphatique*, Paris, 1836, p. 290.
[2] *Gaz. des hôp.* 5th July 1864.

and at the sides, but behind communicated with the deep lymphatics. It was full of rose-coloured fluid. Varicose lymphatics occupied the inguinal canal and upper part of the cord. On both sides a mass of dilated lymphatics ran upwards, around the iliac vessels, the two masses blending together near the diaphragm. In a similar case Nélaton made an exploratory incision into the tumour, by which a quantity of chylous fluid escaped; soon afterwards the patient was seized with a rigor, and died of pyæmia. In this case M. Sappey injected the tumour on the opposite side, and found it to consist of a network of dilated lymphatics. Busey [1] refers to a dissection by Petters of a somewhat similar case. Mazaé Azéma made two dissections, and also found that the abdominal lymphatics were extensively involved.

It is evident from the frequency with which these tumours have been mistaken for hernia, even by surgeons of experience, and the serious consequences which may ensue from such an error, that great care must be exercised in the diagnosis of any tumour occurring in the groins in patients from tropical climates. Magalhães [2] has indicated a valuable diagnostic mark as between this affection of the glands and epiplocele. If the patient be made to lie down on his back with the buttocks raised on a cushion, the swelling in varicose groin glands slowly diminishes or even disappears; if, now, pressure be made with the hand over the seat of the tumour, and the patient then made to stand up, unlike what would happen in any form of reducible hernia, the swelling from refilling of the dilated lymphatics will slowly return.

Lymph Scrotum. (Fig. 69.)

This disease has frequently been described of late years, and under a confusing variety of names. It is sometimes designated nævoid elephantiasis of the scrotum, sometimes varix lymphaticus, or by other inconvenient and somewhat pedantic names. It is by no means an uncommon affection in the endemic area of *F. nocturna*, and may or may not be associated with other filaria diseases.

The cases present great variety both as to history and clinical features, as well as to gravity.

The usual history received is that the disease has been in existence for several years; that the patient's attention was first called to his scrotum by an attack of inflammation and elephantoid fever, usually called ague; that on a discharge taking place from the scrotum, fever and swelling subsided; that ever afterwards, at uncertain

[1] *Lymph Channels*, New York, 1874, p. 78. [2] *Loc. cit.*

intervals, the scrotum has discharged a lymphous or chylous fluid; and that the patient is subject to attacks of inflammation and elephantoid fever. Frequently there is also a history of abscess having terminated the first attack of scrotal inflammation, and perhaps that it has recurred once or twice subsequently. In rare cases no history of fever and inflammation can be elicited.

On inspection the scrotum is seen to be slightly red or dusky, and somewhat enlarged, perhaps twice or three times the normal size. The testes may or may not be enlarged, and there may or may not be single or double hydrocele. The scrotum is relaxed and pendulous, or firmly braced up to the perineum. To the touch it feels thickened, soft, sometimes silky, and at the same time roughened by many projections and irregularities. The raphé is prominent. Studded irregularly over the surface, singly or in groups, or arranged in ill-defined lines running towards the groins, herpes-like vesicles can be made out. Some of these vesicles may be no larger than the head of a pin, others may be ampullæ the size of a finger tip; between these extremes vesicles of all sizes are encountered. The colour of the varices varies with the nature of their contents and the thickness of their walls. They may be clear and straw-coloured, or milky white, or sanguinolent looking. Sometimes only one or two vesicles can be detected, but as a rule they are much more abundant. The varices are usually larger near the raphé, becoming smaller towards the groin, perineum, and penis.

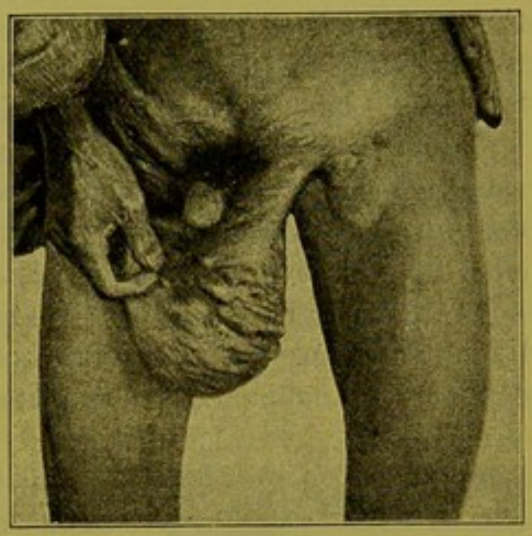


FIG. 69—Lymph scrotum.

If one of the varices be pricked, or rupture spontaneously, a clear, or a milky, or a sanguinolent-looking fluid escapes, sometimes in drops, sometimes spurting out with force as if escaping from a large vein. Collected in a glass this fluid coagulates rapidly, throwing down after a time a scanty reddish sediment, and becoming coated on the top with a thin pellicle of a cream-like substance. As the coagulum contracts it becomes denser and redder. Examined with the microscope the fluid, when milky or sanguinolent, is found to contain lymph corpuscles and red discs like those of blood at various stages of development; also, much finely divided fatty granular

matter, and, almost invariably, living filariæ. When clear the lymph may contain only lymph corpuscles, some granular matter, and filariæ. There may be difficulty in finding the filariæ if care be not taken to collect a considerable quantity of fluid, to stand this for several hours, and search the sediment or a small portion of the contracted clot. Occasionally filariæ may be found in the scrotal discharge, but not in the blood of the patient. Sometimes a thorough and properly conducted examination may fail to detect filariæ either in the lymph or in the blood; such cases are rare, however. I have once found filaria ova in the lymph.

When a varix opens spontaneously, or is pricked, discharge usually goes on for an hour or two. Occasionally it continues to flow for days, reducing the patient to a state of extreme weakness, and, unless active measures be taken, perhaps endangering his life.

I know of no recorded post-mortem examination of a case of lymph scrotum. Doubtless in those cases in which the discharge is chylous or sanguinolent, the scrotal varix is only part of a larger varix, which includes the abdominal lymphatics. In a certain number of cases the varix is entirely extra-abdominal, depending on obstruction in the inguinal glands. In this class of case the contents of the varix are lymphous and clear, containing filariæ, although no filariæ may be discoverable in the blood. Frequently in operating on lymph scrotum associated—as it usually is—with varicose groin glands (Fig. 70), by pressing with the palm of the hand on the groin tumours I have caused quantities of lymph to well up from the mouths of severed vessels in the upper and outer part of the wound; so copious has the flow of lymph been in some cases that I considered it prudent to include the gaping mouths of the severed lymphatics in a ligature. That the vesicles visible on the surface are really lymphatics, is proved by their contents and the nature of the endothelium lining them.

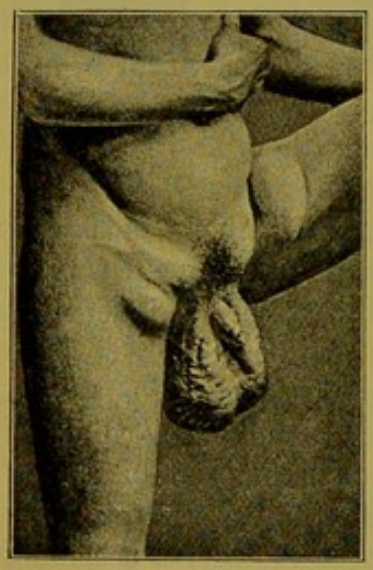


Fig. 70.—Lymph scrotum and varicose groin glands.
(Photo received from Dr. Rennie, Foochow.)

If the discharge of a lymph scrotum be clear, and contain filaria embryos, and if filaria embryos are not to be found in the blood, there is a good prospect of being able to find the parent worm in the scrotal tissues should they be amputated. If the

discharge be chylous, and filariæ be found in the blood, the prospect of finding the parent worm in the scrotum is not good, as in that case it very probably lies in the thoracic duct, or in some part of the intra-abdominal lymphatic system. The reason for this I shall presently explain.

Lymph scrotum may terminate in elephantiasis of the scrotum. If it be observed that a scrotum which formerly discharged frequently ceases thus to empty itself periodically, it is about to pass into a state of confirmed elephantiasis. Not uncommonly amputation of a lymph scrotum is followed by chyluria; at other times, by elephantiasis of a leg.

Other Lymphatic Varices.

Lewis[1] mentions a case of chyluria in which a copious white discharge welled up from the conjunctivæ of both eyes, which were trachomatous and leucomatous. Filariæ were present in the blood, in the urine, and also in the conjunctival discharge, which doubtless came from congested and ruptured lymphatics. I have also recorded[2] a case in which a lymphous discharge exuded from the calf of one leg and was associated with elephantiasis, varicose groin glands, and filariæ. Of the many instances of lymphatic varix of the integuments of different parts recorded in medical literature, probably not a few were of filarial origin, although the parasite may not have been sought for, or, if sought for, was overlooked in consequence of the faulty methods of search employed. There is some reason for believing that a form of diarrhœa may be of this nature, but no reliable observations have been made on this point.

Elephantiasis Arabum. (Figs. 71 and 72.)

Although medical opinion admits the filarial origin of the various diseases just described, there is not the same unanimity regarding the etiological relationship of *F. nocturna* to true *Elephantiasis arabum*. In my own mind I am convinced that such a relationship does exist in an important section, if not in all forms of tropical elephantiasis. When I come to discuss the pathology of the filarial diseases, I hope to refer to evidence sufficient to convince the reader that this view, if not absolutely proved, has been so nearly proved that the classifying of elephantiasis among the filarial diseases is justifiable as well as convenient.

[1] *Indian Annals of Medical Science*, January 1874.
[2] *The Filaria Sanguinis Hominis*, London, 1883, p. 116.

Geographical Distribution.—Elephantiasis—when I use this word I mean to indicate tropical, endemic *E. arabum*, not *E. græcorum* or leprosy, nor the rare and sporadic forms of elephantiasis arising from lymph stasis of non-filarial origin met with at times in all countries—has a very wide area of distribution, being met with very generally all over the tropical and subtropical world, from Japan and the Southern States of the Union in the north, to Australia and Argentina in the south. Hirsch and Felkin state the limits for the Eastern Hemisphere at 35° N. to 25° S., and for the Western Hemisphere at 25° N. to 30° S. As far as known, its distribution is coextensive with the distribution of *F. nocturna*. Like that parasite, it affects some places more than others, preferring apparently low-lying, damp localities, where the water supply is bad, and where the insanitary habits of the people afford abundant opportunity for the filaria to gain access to the human body.

In most warm countries a few cases are met with here and there all over the land, whilst in particular and often very limited districts a very large proportion of the population may be affected. Such a district is Shertullay in Travancore, where, according to Waring (1855), out of a population of 48,591, 2133, or 1 in 21·3, had elephantiasis. Shertullay is an ideal mosquito district. The yearly rainfall averages 100 inches; the land is low-lying, water-logged, swampy, and full of creeks. There are hardly any wells, the people obtaining their water from shallow pools and tanks. Northern Orissa, where elephantiasis is also extremely common, seems to have similar physical features; and a like description applies to large districts in Bengal and elsewhere where elephantiasis is extensively endemic. To suppose, however, that the physical features of a country have anything more than an indirect influence in determining the prevalence or the reverse of the disease, would be a mistake. I know of many places in China where the country might be described as mountainous, yet in which elephantiasis is met with in every village. And there are many mountainous islands in the Eastern Archipelago, in the South Pacific, and in the Indian Ocean, in which large numbers of the inhabitants suffer from elephantiasis of legs or scrotum. I may instance Sumatra, Samoa, New Caledonia, the Fiji Islands, Tahiti, Mauritius, Madagascar. But of all places in the world the islands of the Society and Georgian groups seem to be the most afflicted. According to Saville,[1] in the mountainous island of Huahine "at least seven-tenths of the male population who have reached the age of puberty are suffering more or less from *Bucnemia*

[1] *Skin and other Diseases of India and Hot Climates.* Fox and Farquhar. London, 1876.

tropica" (elephantiasis). Gros,[1] referring to the same island, gives a somewhat lower proportion, on apparently a more limited acquaintance with the people; he says that one-twentieth of the entire population is not an overestimate.

A statement made by several writers on elephantiasis, when treating of the influences that determine its geographical distribution, is to the effect that as far inland as the sea-breezes reach, so far inland will elephantiasis be found, suggesting the idea that the sea-breeze has something to do with the production of the disease. The grounds for such a supposition are quite inadequate. This is one of those specious generalisations which, though deduced from insufficient data, have had peculiar attractions for the half-informed medical mind, and which do not bear the light of criticism and patient investigation. Elephantiasis is found in the centre of Africa, on the western side of Lake Nyassa, in the centre of the Soudan,[2] and hundreds of miles up the Congo; and it is entirely absent as an endemic disease in many islands well within the endemic zone, Formosa[3] for example, and where perhaps neighbouring islands afford examples in abundance.[4]

My belief is that extended investigation will show that the distribution of elephantiasis is determined by a variety of factors, the principal of these being the distribution of one or more species of mosquito capable of acting as the intermediary host of *F. nocturna*; and that this in its turn depends on such circumstances as an adequate rainfall, a summer temperature of at least 80° F., a suitable soil, and stagnant water. Only second to these in importance is the character of the drinking water supply; the habits of the people with regard to its use and management; and, as determining the explosion of the lymphangitis, which is the immediate first step in the development of the disease, the occupations and personal habits of the people as affecting their liability to injuries and irritation of the legs and scrotum.

Age.—According to Waring and Richards, whose statistics are the most extensive available, elephantiasis is unknown in infancy, is rare in childhood, becomes more common in adolescence, and increases in frequency, in proportion to the numbers living, with each decennial period. This increasing frequency with advancing years probably depends on the fact that old age has not only its own susceptibility, but also inherits disease acquired at an earlier period.

[1] *Arch. de méd. nav.* Mai 1892.

[2] Davidson, *Geographical Pathology*, 1892; Felkin, *The Geographical Distribution of Tropical Diseases*, 1889.

[3] Myers, *Med. Rep. Chinese Imp. Mar. Customs*, No. 21, 1881.

[4] Gros, *Arch. de méd. nav.* Mai 1892.

DURATION OF DISEASE WHEN OBSERVED.

Disease has existed.	Waring (Travancore).	Richards (N. Orissa).	Per cent.
Under 1 year,	44	8	3·29
1 to 5 years,	196	183	24·03
6 „ 10 „	197	169	23·09
11 „ 15 „	136	94	14·57
16 „ 20 „	126	114	15·18
21 „ 25 „	79	32	7·02
26 „ 30 „	71	26	6·13
31 „ 35 „	30	3	2·08
36 „ 40 „	23	5	1·77
41 „ 45 „	11	2	1·07
46 „ 50 „	2		
51 „ 55 „	2		
Doubtful,	28		1·77
	945	636	100·0

AGE OF PATIENTS.

	Waring (Travancore).	Richards (N. Orissa).
5 to 10 years,	2	2
11 „ 15 „	12	
16 „ 20 „	54	44
21 „ 25 „	71	91
26 „ 30 „	117	115
31 „ 35 „	98	159
36 „ 40 „	156	
41 „ 45 „	110	210
46 „ 50 „	112	
51 „ 55 „	70	
56 „ 60 „	66	
61 „ 65 „	22	15
66 „ 70 „	10	
Over 70 „	10	
Doubtful,	35	
	945	636

Sex.—Little reliance can be placed on the statistics we possess in attempting to determine the respective liabilities of the sexes. In Eastern countries women are very diffident about consulting European physicians; consequently, in all hospitals and dispensaries in these countries female patients are greatly in the minority, and statistics of disease, as affecting them, must considerably understate the facts. Doubtless, however, owing to the relative immunity women generally enjoy from exposure to the exciting causes of lymphangitis, they are less frequently affected with elephantiasis than men. Waring states the proportion of the male population

affected with elephantiasis in Shertullay as 1 in 16·5, of the female population as 1 in 38·5. Observers in other countries are very generally agreed as to the preponderating proportion of male cases.

Occupation.—Judging from my own experience, I would say that the field workers and the labouring classes generally are proportionately much more frequently attacked with elephantiasis in the leg than the richer and more sedentary classes. This difference in liability is less marked in scrotal elephantiasis; to this as well as to another form of filaria disease, chyluria, I think the rich are just as liable as are the poor. The reason for this appears to be, that although filarial lymphatic congestion may be as common, or nearly so, among the rich as among the poor, the latter, having to be on their feet all day, having to work in field or jungle without shoes or perhaps garment of any kind, are more exposed to causes of crural lymphangitis and its consequences than are the rich and sedentary; whereas in all classes the scrotal and abdominal lymphatics run about the same risk of filarial obstruction and subsequent injury.

Race.—It is very frequently stated that the dark races are more liable to elephantiasis than the white, the only reason for this assertion being that whites are rarely affected. The true reason for the comparative immunity of the white races, which as regards some countries is undoubtedly a fact, is that whites as compared to the coloured natives live more hygienic lives, are more careful about the water they drink, and seldom have to work in the fields or to go about barefooted or half-clothed, or do any manner of manual labour; consequently they are more rarely affected with *F. nocturna*, or, if affected, are not so much exposed to causes of lymphangitis. Relatively to the numbers of the two races affected with elephantiasis of the legs, I would say that there are more whites affected with chyluria and scrotal tumour and varicose groin glands than there are blacks; the reason for this need not be restated.

When whites assume the habits of the coloured races among whom they live, they get the same diseases. This fact is plainly brought out in the case of elephantiasis in many parts of the world, and nowhere more markedly than in the island of Huahine, already referred to. Saville says,[1] and his statements are borne out by the more recent observations of Gros, that when in this island foreigners adopt, as they generally do, native habits, they sooner or later acquire the prevailing disease; and that in 1873 of the fourteen Europeans resident there eleven were "heavily afflicted with elephantiasis arabum." Indian and Brazilian experience points to the same conclusion. Barbadoes is another case in point.[2]

[1] *Loc. cit.* [2] Hillary, 1759; Hendy, 1784.

PARTS AFFECTED WITH ELEPHANTIASIS.

	Waring.	Day.	Richards.	Silva Araujo.	Total.		Per cent.
Right lower extremity,	291	27	203	97	618		
,, ,, and right upper,	10	0	7	3	20		
,, ,, and left upper,	4	0	4	1	9	659	31·67
,, ,, and both upper,	2	0	0	0	2		
,, ,, and scrotum,	0	2	0	8	10		
Left lower extremity,	272	24	171	102	569		
,, ,, and right upper,	5	2	5	2	14		
,, ,, and left upper,	9	0	2	2	13	601	28·88
,, ,, and scrotum,	1	1	0	3	5		
Both lower extremities,	296	31	201	136	664		
,, ,, and both upper,	15	4	10	3	32		
,, ,, and left upper,	15	0	5	3	23		
,, ,, and right upper,	16	1	8	2	27		
,, ,, and scrotum,	0	1	0	5	6		
,, ,, and left hand and mamma,	1	0	0	0	1	755	36·29
,, ,, and right upper and scrotum,	0	0	0	1	1		
,, ,, and both upper and scrotum,	0	0	0	1	1		
Right upper extremity,	1	0	10	3	14		
Left upper ,,	2	3	6	3	14		
Both upper extremities,	0	0	4	4	8	37	1·77
Left upper extremity and mamma,	1	0	0	0	1		
Other Parts.							
Scrotum alone,	3	3	0	19	25		
Mamma alone,	0	1	0	0	1		
Face,	0	0	0	2	2	29	1·39
Lobe of ear,	1	0	0	0	1		
	945	100	636	400	2081	2081	100

Regions of the Body affected.—The statistics of Waring, Day, Richards, and Silva Araujo, dealing with 2081 cases of elephantiasis, show that in 96·84 per cent. one or both lower extremities alone or along with other parts were affected; in 5·86 per cent. one or both upper extremities alone or with other parts were affected; in about 2·3 per cent. the scrotum alone or with other parts was affected: the mamma was involved only once in every 690 cases, and the lobe of the ear only once in the entire series of 2081 cases. They mention no case of elephantiasis of the labia or clitoris, although these parts are at times attacked; nor do they refer to elephantiasis of the integuments of the head or buttock, cases of which are recorded by other writers.

The cases of elephantiasis I have had to deal with in China were

more or less selected, and are therefore useless for statistical purposes. Many came to the hospital I was in charge of for the removal of scrotal tumour, and consequently the proportion of such cases was in excess of what really obtained in the district. I cannot say, therefore, if these Indian and Brazilian statistics apply to China. They certainly would not apply to Huahine; for of the 11 foreigners, already referred to as being on that island in 1873, 4 had elephantiasis in one leg only, 1 in both legs, 3 in both legs and also in both arms, 2 in both legs and in scrotum, and 1 in one arm and scrotum. Gros reports of 19 cases observed by him in Tahiti, that in all of them the lower limbs were affected; and of 15 cases noted, apparently in Huahine, 9 had both legs affected, 2 one leg, 2 both arms and scrotum, 1 one arm and both legs, and 1 both arms and both legs. Other reports from the South Pacific Islands seem to indicate a greater proportion of cases with both arms and also with scrotum affected than is the case in India. It would appear from this that the liability to implication of the upper extremities and scrotum is in proportion to the intensity of the local endemic influence; that is to say, that where a small proportion of the population is affected with elephantiasis the proportion of arm and scrotum cases to leg cases will be small too, but that where the population is extensively affected the proportion of arm and scrotum to leg cases will be very much increased. In other words, the greater the number of cases of filarial infection in a district, the greater are the chances of extreme degrees of filarial infection occurring.

Incidence and Progress.—In the vast majority of cases, elephantiasis dates from an attack of lymphangitis and elephantoid fever. Although with the subsidence of the inflammation and fever the greater part of the effusion may have been absorbed, some degree of swelling remained. The inflammation relapsing at irregular intervals of weeks, months, or years, each attack added a little to the swelling. Thus by successive additions, the deformity, so aptly compared to an elephant's leg, was gradually built up. After a time, although the inflammatory attacks become milder, or recur at longer intervals, or perhaps cease for good, the swelling may still gradually increase; especially is this the case when the scrotum is the seat of disease, for this part may continue to enlarge for years after the attacks of lymphangitis which signalised the commencement of the trouble have ceased.

Often although only *one* leg is attacked the glands of *both* groins are affected; consequently, we see cases of one-sided elephantiasis with two-sided adenitis. Sometimes at the outset, and

before the integuments have become thoroughly and permanently involved, there is the appearance of a kind of metastasis. With the first attack of lymphangitis one leg swells; the next attack may involve the other leg, and then the swelling may leave that which was first attacked; this may occur more than once. Often, after one leg has been affected for some time the other leg or the scrotum may be attacked; or the scrotum may be first attacked, and later on, one or both legs. Every variety of combination of this description, as well as of liability to fever, rapidity of progress, and bulk of affected organs, is met with.

In the earlier attacks of lymphangitis it is not unusual for the leg or scrotum to obtain relief from the inflammatory tension by a sort of weeping exudation of lymph from the surface, and the history of many cases of elephantiasis of the scrotum is that of antecedent lymph scrotum gradually becoming consolidated and merging into true elephantiasis.

As a rule, beyond the swelling and consequent weight and stiffness and occasional attacks of lymphangitis and fever, elephantiasis of the leg gives rise to little inconvenience, and is attended with but little suffering or risk to life. In the case of elephantiasis of the scrotum, the weight of the tumour, reaching perhaps to the knees or even to the ankles, may be so great as to make walking almost impossible, and in such cases extensive gangrene or abscess may, at times, even endanger life.

Symptoms, Morbid Anatomy, and Histology.—In a general way a part affected with elephantiasis may be said to be hypertrophied. The hypertrophy, however, does not extend to all the tissues implicated in the tumour, but is confined principally to the skin, subcutaneous areolar tissue, the aponeuroses, intermuscular connective tissue, and the sheaths of vessels and nerves. In consequence of pressure and constriction, the muscles may be atrophied or the subject of fatty degeneration. The veins and arteries are enlarged, and the bones may be thickened and roughened by osteophytic growths. In rare cases the bones are atrophied. The lymphatic trunks are usually much enlarged and distended, and the glands swollen and dense from increase of connective tissue.

On cutting into a leg or scrotum affected with elephantiasis the morbid tissues are seen to consist of two parts; an outer capsule, from half an inch to several inches in thickness, of hard, dense resistent, whitish tissue—the hypertrophied deeper layers of the skin and more superficial layers of the subcutaneous areolar tissue; and an inner layer of loose, blubber-like, dropsical, yellowish connective tissue—the hypertrophied subcutaneous fascia. In the case of the

leg, the greater part of the enlargement consists of the outer and denser layer; in the case of the scrotum, the bulk of the tumour is made up of the dropsical and looser inner layer, the outer layer at the same time often being an inch or two in thickness. The hypertrophied connective tissues, although they appear soft and loose, are not easily torn through; they are strengthened by irregular fibrous bands running in all directions.

On section, the outer layer is seen to consist principally of fibrous tissue, the strands of which are arranged horizontally, vertically, and obliquely, and are closely interlaced one with another. The epidermis is greatly thickened in some places, hardly affected in others; the papillæ are sometimes atrophied, sometimes enlarged; the sudoriparous glands, where not atrophied, have their ducts elongated and their glandular elements more or less degenerated; the same is the case with the hair follicles. The walls of the capillaries are thinned, the vessels themselves dilated and increased in number; the lymphatic radicles are usually similarly enlarged and distended.

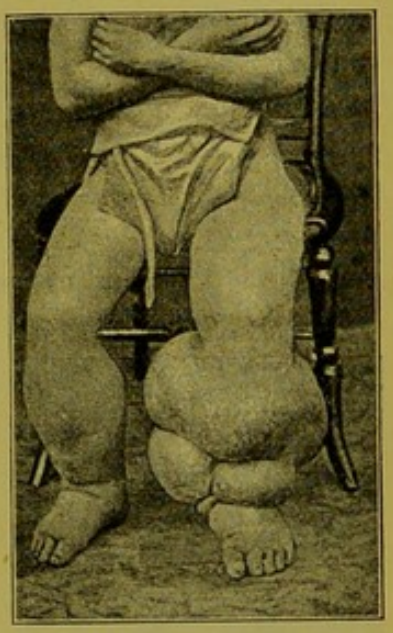


Fig. 71.—Elephantiasis of the legs.

Elephantiasis of the Lower Extremities. — When the lower extremities are affected, the disease in most cases is confined to below the knee, involving principally calf, ankle, and foot. In some instances the upper limit may be somewhat sharply defined, in others again the disease seems to merge gradually into the healthy tissue. There is nearly always a deep and well-marked sulcus surrounding the ankle, and permitting some degree of movement in that joint. Elsewhere the integuments are hard, dense, thick, more or less pigmented, and rough. The papillæ are in places pilose, in other places tuberous and warty. Only at the sides and bottom of the ankle sulcus, and in some instances between the toes, does the skin appear to be at all healthy. In these situations elephantoid growth seems to be repressed by the pressure of opposing surfaces. The hair grows irregularly and coarsely, and the nails are thickened, rough, and misshapen. The parts do not perspire readily, and the sensibility is somewhat diminished.

When the disease invades the thigh, the skin is not so coarse as in the leg; it cannot, however, be pinched up or glided over the

subjacent structures, and it may be thrown into large folds about the knee. Localised patches of elephantiasis or elephantoid œdema, accompanied or unaccompanied by elephantiasis elsewhere, are sometimes met with in the thigh.

The leg may attain enormous dimensions, as may be seen from the following statistical table by Waring:—

MEASUREMENTS TAKEN ROUND THE ANKLE OF 340 CASES OF ELEPHANTIASIS OF THE LEG. (WARING.)

In	2	patients the ankle measured	8 inches in circumference.
,,	28	,,	9 ,,
,,	68	,,	10 ,,
,,	103	,,	11 ,,
,,	61	,,	12 ,,
,,	40	,,	13 ,,
,,	15	,,	14 ,,
,,	9	,,	15 ,,
,,	5	,,	16 ,,
,,	1	,,	17 ,,
,,	5	,,	18 ,,
,,	1	,,	19 ,,
,,	1	,,	21 ,,
,,	1	,,	24 ,,
340	Average measurement, 11.		11 $\frac{7}{12}$ inches.

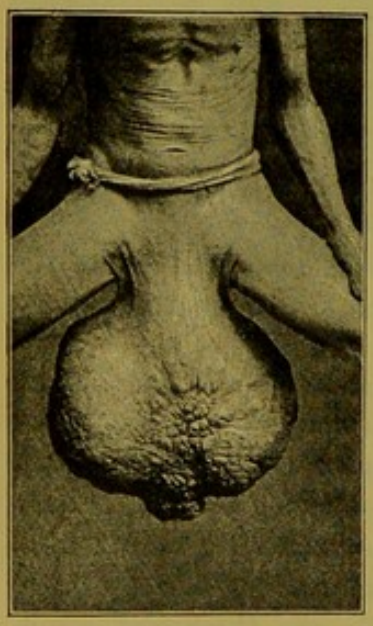


FIG. 72.—Elephantiasis of the scrotum.

Scrotal Elephantiasis.—When elephantiasis attacks the scrotum this is gradually converted into a huge pear-shaped tumour with its narrowest part at the pubic and perineal attachment (Figs. 72 and 73). The testes, being more or less firmly bound to the bottom of the scrotum by the remains of the gubernaculum testis, are dragged down and the cords greatly lengthened; as a rule, they are to be found towards the back part and considerably below the middle of the tumour. It is important to remember this fact, and also the existence of the fibrous connection of the testes to the bottom of the scrotum. The penis, being firmly held in place by the suspensory ligament, is not much dragged down, and is therefore to be found in the upper part of the neck of the tumour not far from the anterior surface, the glans lying at the bottom of

a long tunnel opening by a wide orifice some way down the face of the tumour. This tunnel is lined by the telescoped prepuce and the inverted sheath of the penis. As a rule, the larger the tumour the smaller, proportionately, is its neck and attachment. This, if cut across, would be found to be triangular in shape (Fig. 73, B), the base of the triangle looking forwards, the apex—often bifid from dragging down of the folds of the nates—backwards to the anus, and the sides looking to the thighs. The integument covering the upper part of the sides of the neck is usually soft, pliant, and healthy in appearance, and looks as if it were borrowed by dragging from the thighs. Henderson,[1] however, points out that this appearance of health is deceptive, and is very likely produced by pressure of the thighs on the tumour. Hence, Indian surgeons of the greatest experience warn us against being deceived by this healthy look of the skin into making flaps out of it when the case comes for operation; they say disease is almost sure to return if flaps of any kind are made. I think this dictum is a little too sweeping; I have often made small flaps from such skin without return of disease; though, I confess, I have also seen cases in which flaps, apparently healthy at the time of operation, took on disease at a later date. French operators ignore the danger, and generally make flaps to cover the testicles. Sometimes the disease extends well on to the surface of the abdomen, in which case the neck is entirely constituted of diseased structures.

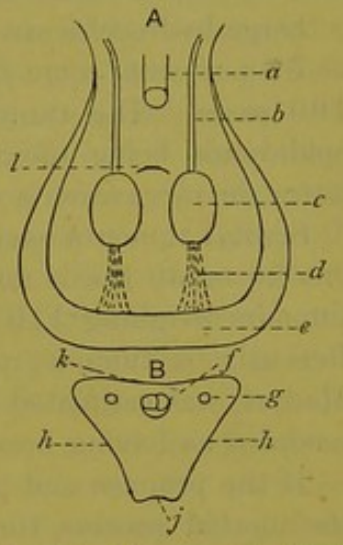


FIG. 73.—Diagrammatic sketch of elephantiasis of the scrotum.

A.

a, Penis attached by suspensory ligament to os pubes.
b, Spermatic cord elongated.
c, Testis carrying large hydrocele in front.
d, Fibrous remains of gubernaculum testis connecting testis with bottom of scrotum.
l, Usual position of preputial orifice.

B.

Cross section through the neck of a narrow-necked elephantiasis of the scrotum.
g, Spermatic cord.
f, Penis.
h, Crural surface of neck.
j, Bifid posterior angle of neck anterior to anus.
k, Anterior surface.

The surface of a scrotum affected with elephantiasis is rough and coarse, and not very sensitive. If it be contemplated for some time, especially if it be irritated by being scratched with the nail or with a pen or knife, it is seen slowly to undergo changes of shape. These changes are the result of the contractions of a hypertrophied dartos. The movement spreads after a few seconds from the focus of irritation, and reminds one

[1] *Edin. Med. Journal*, January and March 1880.

of the slow peristaltic movements of the intestine, or of a dilated stomach sometimes perceived through the walls of the abdomen. This irritability of the dartos persists for many hours after a tumour has been amputated; by compressing the blubbery, spongy tissues of the centre of the tumour, the muscular contractions squeeze out much lymphous fluid, and thereby diminish the bulk of the mass very considerably.

Large hydroceles are present in most cases; according to McLeod,[1] in 56 per cent., more frequently according to my experience in over 100 cases. The tunica vaginalis is always much thickened, the epididymis being often somewhat separated from the testis; the latter, in rare cases, is completely atrophied by pressure.

Scrotal tumours vary much in size—10 lbs. is a common weight, but 20 or 30 lbs. is not unusual; and there are cases on record of tumours weighing 110 lbs. (Clot Bey, Fayrer), 111 lbs. (Partridge). Recently, a tumour removed by Surgeon-Major W. R. Browne, at Madras, was estimated to weigh 140 lbs. when *in situ*. The largest recorded as having been removed weighed 224 lbs.[2]

If the prepuce and penile integuments are specially implicated in the morbid process, they may increase proportionately, or even disproportionately, with the enlargement of the scrotum. In this case the orifice of the prepuce is at the free extremity of a long ram's horn-looking growth springing from the front, generally of the upper part of the scrotum. The penis itself is not enlarged, but lies deep in the tumour firmly attached to the pubes by its suspensory ligament.

Elephantiasis of the Female Genitals.—Either both labia or one labium may be the seat of elephantoid growth, or the disease may affect principally the mons, or the clitoris. In the latter case the urethra is dragged down and courses for some distance along the upper and back part of the growth, and must be carefully dissected out before such a tumour is amputated. I have removed a tumour of this description weighing 7 lbs. Tumours of the labia may attain considerable dimensions, reaching, in some cases, as far down as the knees or even lower. In anatomical and histological characters they differ materially in no essential from elephantiasis elsewhere.

Tumours of the mamma are very rare, but, when they do occur, may attain large dimensions. Salmuth[3] refers to one which reached to the knees, and was associated with greatly enlarged axillary

[1] *Operative Surgery.*
[2] Chevers, *Diseases of India*, p. 269.
[3] *Compendium de Méd. Prat.* Bruxelles, t. ii. p. 125.

glands. Estienne is said to have removed a similar tumour which weighed 21 lbs. and reached to the pubes.[1]

I do not suppose that the diseases I have attempted to describe exhaust by any means the list of morbid affections produced by *F. nocturna*. Up to the present they are all we can with good reason attribute to it; but, doubtless, as knowledge of the subject extends, other diseases will be found to be caused by this parasite. A great variety of morbid affections have, at one time or another, been conjectured to be owing to the filaria,—including leprosy itself,—but on altogether inadequate grounds. It is reasonable, however, to suppose that the subject is by no means worked out, and that there are still many obscure diseases which in time will be shown to acknowledge the etiological cause which we may, with confidence, assign to the elephantoid group. Magalhães informs me that he lately had under observation a filarial patient suffering from amblyopia associated with retinal hæmorrhages, for which none of the usual causes for this affection could be found; and he speculates as to the possibility of a connection existing between the parasites in the blood and the grave ocular affection by which his patient had been afflicted for several years. It is evident that much work has still to be expended before we can hope to attain finality in our knowledge of this parasite and the diseases it gives rise to.

Pathology.

It is very certain that in the great majority of instances in which the blood is infested with filariæ, no harm whatever accrues. Host and parasite live together for years in perfect harmony. It is so in the case of many of the lower animals, and it is so in man. Manifestly, in view of the life history of *F. nocturna*,—as probably of all filariæ whose young make their first step in development and towards independent life by entering and circulating with the blood,—it is in the interest of the parasite that the health and longevity of its host should not be seriously impaired. Nature's plans are laid accordingly, and host does not injure parasite, and parasite does not injure host.

But that this harmony is sometimes interrupted is equally certain. This interruption, however, is not a necessary feature of the parasitism, or the result of imperfect adaptation of parasite to host; rather it is the result of accident—accident in the location of the parasite, disease in the parasite, traumatism, or other circum-

[1] *Bull. de l'Acad. de méd.* 1839, t. iii. p. 560.

stances not yet properly understood. Such misadventures to the parasite, though comparatively rare, do occur, and when they occur may result in disease to the host.

The lymphatic system being the habitat, at all events the usual habitat of the parent filaria, if any such accident as I refer to should occur, we would naturally expect that the part first to suffer would be this system. Accordingly, if we compare the physical condition of say a thousand individuals who have no filariæ, and never had filariæ in their blood or tissues, with that of a thousand individuals who have or have had filariæ in their blood or tissues, among the latter there will be found to be an undue proportion of certain forms of lymphatic disease which are rarely if ever encountered among the former. If we investigate these cases, we soon come to recognise that a principal feature which these lymphatic diseases have in common is varicosity of the lymphatic vessels.

That these various lymphatic diseases are from a pathological point of view identical in nature, is proved by the frequency with which they concur in the same individual, by the frequency with which one of them gives place to another, and, in a secondary way, by the fact of their having the same geographical distribution, and, of course, by the frequency with which *F. nocturna* is found in all of them. I shall base my argument about the pathology of the filarial diseases on four of the principal of these lymphatic affections, viz. chyluria, varicose groin glands, lymph scrotum, and chylous dropsy of the tunica vaginalis. I hold that *Elephantiasis arabum* also belongs to the same group; but, for reasons of convenience, shall defer the consideration of the pathology of this form of filarial lymphatic disease till that of those diseases I have just mentioned has been discussed and made clear.

If, in any of these diseases, we examine microscopically the contents of the varicose lymphatics after they have escaped spontaneously as in chyluria or lymph scrotum, or been liberated by puncture in chylous dropsy of the tunica vaginalis, or been aspirated from varicose groin glands, we find, in the great majority of instances, in addition to the usual elements of chyle or lymph, myriads of embryo filariæ. The frequent concurrence of lymphatic varix with filariæ in the blood is suggestive of some relationship between disease and parasite, which suggestion is further accentuated by such considerations as their corresponding geographical distribution; but when we find that the filaria not only concurs with the varix, but actually occurs *in* the varix, a very justifiable and obvious conclusion is that the one is the cause of the other;

and, as the varix cannot possibly give rise to the filaria, we are driven to the conclusion that the filaria gives rise to the varix.

It may be, and has been objected to this conclusion, that the filariæ find their way by a sort of diapedesis from the blood vessels into the varix after this had been produced by causes quite unconnected with the parasite. When we contemplate the structure of the filaria, its unplastic body, the masking of its boring apparatus by the loose sheath with which it is encumbered, such a migration through the walls of a double set of vessels is difficult to understand; but, for the sake of argument, admitting its possibility in the case of the free swimming embryo, such a feat is impossible on the part of the passive ovum and the relatively gigantic parent worm; these could not thus migrate. Yet they have more than once been found in the contents of the class of lymphatic varices we are considering. To explain their presence there we are forced to conclude that the parent filariæ live in the lymphatics. Consequently, believing in the uniformity of nature's operations, we are led to the inference that the filariæ we so constantly find in the lymph are born in it, and come from a parent living in it.

The question then comes to be, not, do the filariæ produce the varix, but how do they produce the varix.

It may be regarded as an axiom in pathology that noncongenital or acquired varix of lymphatics is the result of occlusion or obstruction of the lymphatics on the cardiac side of the varicosity. It is not produced, as sometimes happens in the case of the veins, simply by the distending force of a long column of superincumbent fluid; the lymphatic trunks are too short and too well guarded by valves to permit of this. Varix of lymphatics implies obstruction of lymphatics at a point above the varix; and, inasmuch as the contents of the majority of these filarial varices are chylous, the obstruction must necessarily in all such cases be situated in the thoracic duct, and at a point above the entrance of the vessels carrying the chyle. There are no observations as to the exact manner in which the filaria produces this obstruction; but we can understand that it might be brought about by the body of the parent worm simply plugging the thoracic duct, or causing inflammation in or about the walls of the vessel, or, perhaps, ulceration of its lining membrane and consequent stenosis, or by the ova or such masses of matted embryos as we sometimes see in the fluid of chylous dropsy of the tunica vaginalis, or by some other product of conception.[1]

[1] An interesting and suggestive illustration of a varix of verminous origin is supplied by the verminous aneurisms, so common in the mesenteric arteries of the horse, produced by the palisade worm, *Sclerostoma armatum*.

However produced, given the obstruction, it is easy to understand how the varix is brought about by the eccentric pressure of the accumulating lymph, and also how all the consequences which this involves come about.

This theory of the causation of lymphatic varicosity by the filariæ seems reasonable enough; but, reasonable though it may seem to be, unless supported by experimental, by post-mortem, or by further direct evidence, it cannot be regarded as thoroughly established, or as anything more than very plausible, fact-based speculation.

In medical literature, so far as I can find, there are but few recorded post-mortem examinations of chyluria subjects, and fewer still of cases of varicose groin glands, or of lymph scrotum, to which we might refer. Of the post-mortems which have been recorded, there are very few in which a detailed account is given of the state of the thoracic duct; and in many of them there is no mention even made of this vessel, far less any record of special dissection to ascertain its exact condition. So that as regards a very important point, these post-mortems afford, in most instances, little or no evidence one way or another. I am aware of only three or four dissections in which the state of the thoracic duct is described. This matter is of such vital importance in discussing the theory of filaria disease, that I propose to give in some detail the record of two of these cases and dissections.

Dr. Stephen Mackenzie records[1] the case of a European—born and resident in India for twenty-five years—who, a month after his arrival in England, developed chyluria, and in whose blood and urine *F. nocturna* was found in abundance. After seven months of persistent chyluria he was attacked with rigor, followed by fever, signs of lung trouble, and pain in the left shoulder. Abscess formed in the neighbourhood of the left sterno-clavicular articulation, and was opened fourteen days after the initial rigor, and pus with some blood evacuated. Some improvement followed, but fever, with signs of double pleurisy and congestion of both lungs, persisted, and, after a time, there was renewed swelling of the left shoulder and arm, terminating in the formation of fresh abscess in the neighbourhood of the acromion process. This was opened and drained, with temporary relief to the pain and swelling. Fever persisting, inflammation of the lungs and, later, cystitis supervened, and the patient gradually sank. He died six weeks after the second abscess was opened, about eighty days after the rigor issuing in his last illness, and about ten months from the first appearance of chyluria.

[1] *Trans. Path. Soc. Lond.* 1882.

The rigor referred to occurred at three in the morning; the same evening thirty filariæ were counted in one slide of finger blood, and it was observed that they were in a languid condition. On the following day, at 9 A.M., five filariæ were counted on one slide, and later, at midnight, twelve; but from that time, although the blood for at least ten days was systematically examined, no more filariæ were found in it. From the time of the rigor the urine began to

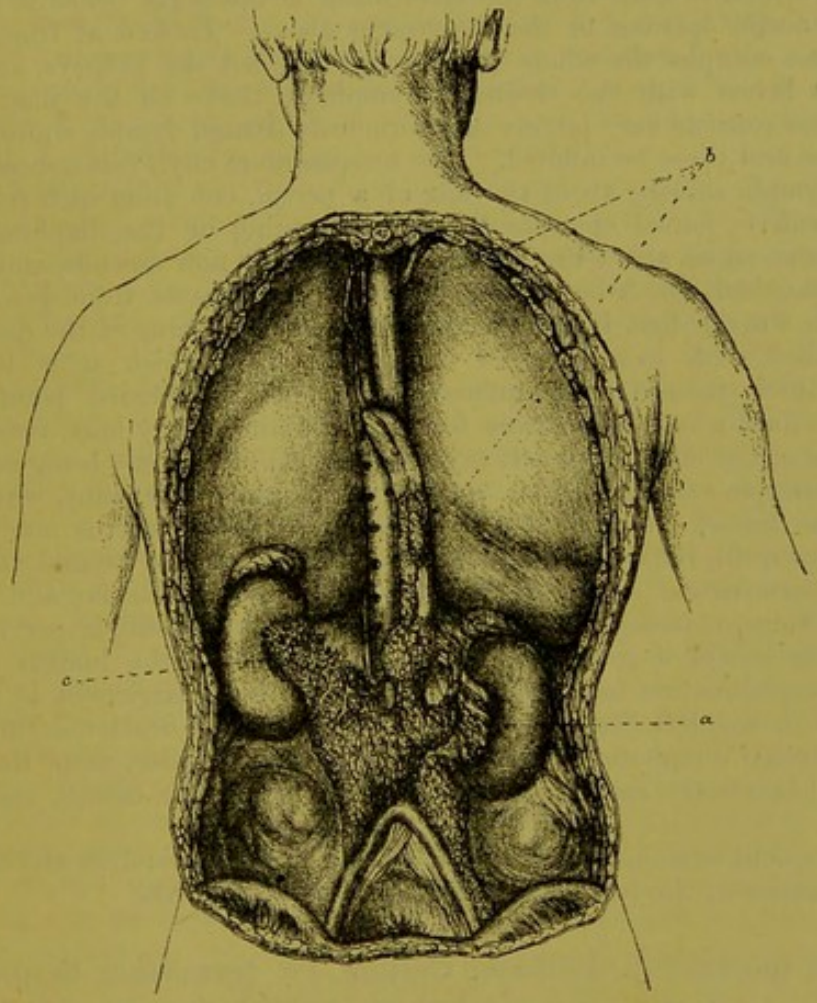


FIG. 74.—Dissection of the lymphatics in a case of chyluria. (Mackenzie, *Trans. Path. Soc. Lond.*)

clear; four days later it was found to be quite clear, with only a trace of albumen and no clots. Filariæ disappeared from it the day following the rigor; and on only one subsequent occasion were clots ever found in it, although albumen was always present in greater or less amount. Latterly it contained about half albumen, along with pus and other products of cystitis.

At the post-mortem examination an empyema was found on the right side, with corresponding lung collapse, and pus-plugging of

small bronchi. There was left-sided pleurisy and pulmonary œdema. The urinary bladder was inflamed, and the kidneys were in a state of incipient suppurative nephritis. Liver, spleen, and heart appeared healthy. A careful dissection of the lymphatics (Fig. 74) was made by Mr. E. H. Fenwick, and described as follows by the late Dr. James Anderson :—

The thoracic duct commences in a dense mass of lymphatic tissue and glands, which extends from the bifurcation of the aorta below to the level of the aortic opening in the diaphragm above. Looked at from behind, this mass occupies the whole of the space between the kidneys, and is continuous below with the chains of lymphatic tissue on the iliac arteries. The mass consists very largely of enormously dilated lymph sinuses, which can here and there be inflated. The receptaculum chyli commences by two large lymph sinuses about the size of a pencil, one from each side of the aorta, and is joined opposite the aortic opening of the diaphragm by a third large sinus about the same size. The duct now ascends, sinuous and much pouched, for 3 or 4 inches, varying in diameter from $\frac{3}{8}$ to $\frac{1}{2}$ inch, pervious for the first $1\frac{1}{2}$ inches above the aortic opening of the diaphragm, then filled with loose clot for $1\frac{1}{2}$ inches, after which it is lost in a tough thick mass (*query*, inflammatory). (The occluded point in the thoracic duct when opened was found to contain a very long, twisted clot, tapering at the end. It is left *in situ*, to be shown, before being submitted to microscopic examination.) About 4 inches above this point, when it can again be traced, although still involved in dense tissue, it is now the size of a crow quill, impervious, and tending to the left side behind the aorta. At its termination in the angle between the left subclavian and internal jugular veins, it passes through a mass of lymphatic tissue, is pervious, and about the size of a goose quill. As stated, the iliac, the lumbar, and the renal lymphatics are very much enlarged, and the enlargement is specially marked in the left iliac and left renal lymphatics. Scattered throughout the left renal lymphatics are numerous hard round masses, some the size of a pea, but mostly smaller. These masses manifestly occupy the lymph sinuses.

Subsequent examination of the coagula of the thoracic duct and the hard round masses in the lymphatics did not reveal any filariæ.

I am indebted to Professor Curnow for permission to quote the following interesting case, not hitherto published, and to Dr. Spencer, Seamen's Hospital, Greenwich, for the use of his notes in preparing the precis. I had the advantage of seeing the case on several occasions, and of being present at Professor Curnow's dissection of the thoracic duct, for the description of which I am responsible :—

J. M., a seaman, aged 32, was admitted into the Seamen's Hospital, Greenwich, under Dr. Curnow, on the 7th February 1891, suffering from cough, and pain in the right side. He stated that he was born in Burma of English parents, and that most of his life had been passed in the East Indies. Ten months prior to his admission he had an attack of what he called "malaria" in Calcutta. The illness for which he was admitted was

of three weeks' standing. On examination he was found to have signs of fluid in his right pleura extending as high up as the fourth rib, with friction râles above, and an irregular temperature occasionally as high as 102°. His condition did not improve much, and on 6th April he had a severe rigor, accompanied by pain in the back and limbs, and headache; there was no alteration in the physical signs, however, although the temperature rose to 103°·8. On 17th April he was considerably better; but it was noted on that day, and for the first time, that his urine, which previously had been quite clear and free from albumen, was chylous. On 23rd April the urine being still chylous, the blood was examined at night, and filariæ embryos were found in it; they were also found in the urine. On 28th April the urine ceased to be chylous, but filariæ persisted in the blood, and were frequently found there from time to time, and in considerable numbers, up to 28th May. After this date very few were found; two days before his death I succeeded in finding one or two specimens in a large slide of blood. Temperature still remained high though irregular. On 1st June he complained of severe and persistent headache, and by 8th June he was evidently very ill, being weak and lethargic, vomiting occasionally, and, at night, delirious. On the 10th he was unconscious, and there was slight left facial paralysis, and, at times, marked rigidity of both arms. He died on 12th June.

At the post-mortem examination a localised serous effusion was found in the right pleura, the upper part of the sac being adherent. The left pleura was healthy. Both lungs contained an abundance of what were pronounced to be miliary tubercles. Apart from this, and the condition of the thoracic duct about to be described, all organs appeared to be healthy. In the thorax the thoracic duct was felt to be very much enlarged, having a solid feel, as if it contained a number of hard lumps. Traced through the diaphragm, the duct was lost in a mass of firm tissue, forming an ill-defined tumour, about the size and shape of two superimposed hands, lying in front of and in contact with the vertebræ. This mass extended down into the right iliac fossa for some distance, and on the left side surrounded the ureter. On laying open the bladder, ureters, and kidneys, they were to all appearance healthy. The duct, along with the mass of new growth, were removed, and placed in spirit to await more detailed examination. On dissection some time afterwards it was found that the upper and terminal portion of the thoracic duct, for about 2 or 3 inches, was completely occluded; a probe could not be passed into it. Nor could it be traced into the left subclavian vein, the parts in the neighbourhood being much matted, and more or less implicated in a small, disc-shaped, flattened tumour about $1\frac{1}{2}$ inches in diameter and some $\frac{3}{4}$ inch in thickness. About 2 inches from the subclavian vein the duct was as thick as a goose quill; lower down it increased in diameter, but irregularly, some parts being quite as large as the little finger, others again being not much bigger than an ordinary lead pencil. On cutting into the duct, the cause of the hardness remarked at the post-mortem examination was apparent; the entire lumen was occupied by a firm core, or what appeared to be, and doubtless was, a sort of thrombus of inspissated lymph and chyle. This core, of a light brown colour, was firm, but not fibrous, for it could readily be broken into fragments and cut like a piece of cheese. It showed no lamellar structure, nor, indeed, structure of any sort, either to the naked eye or when placed under the microscope. At the upper and narrower part of the duct the core was intimately connected with the wall

of the vessel; but lower down, in the more distended part, there seemed to be no structural continuity between the two, for there the core could be easily shelled out, and fluid was seen to pass between it and the inner surface of the duct. This arrangement of core and vessel suggested to my mind that any fluid which the thoracic duct had transmitted must have passed downwards and between the wall of the vessel and the thrombus, and that fluid must have been so passing for a considerable time, and up to the day of the patient's death, else how account for the enormous size of the core, and the patency of the vessel on its outside. The appearance of the thrombus resembled somewhat the deposit on the inside of an aneurism, with this difference, that in place of being adherent to the walls of the vessel, and growing by additions to its interior, it was separated from the vessel by an appreciable interval, and probably grew by additions to its outside—by an exogenous rather than by an endogenous process of accretion. The large mass of matted tissue and new growth in front of the vertebræ in the abdomen, in which the dilated duct was lost, when cut into was found to be tough, dense, somewhat fibrous, greyish in colour, with here and there small gaping vessels scattered about on the surface of the sections; these vessels, for such they were presumed to be, had very thin walls, and may have been dilated lymph sinuses. The smaller mass, already alluded to as implicating the upper end of the duct, was of a similar nature. No ruptured vessel or dilated vessel was demonstrated anywhere in the urinary tract.

These two cases, inasmuch as in them the dissection of the thoracic duct was thoroughly carried out, are the most complete and convincing as yet recorded as to the mechanism of the production of chyluria. There are one or two others which, so far as they go, strongly support the theory advanced, and which I may allude to. In Esmarch-Kulenkampff's "Die Elephantiasis Formen" a case[1] is detailed in which filarial chyluria was complicated with diarrhœa—occasionally chylous, and which terminated in death. The patient was a woman, resident fourteen years in Brazil. Her urethra was so dilated by the passage of chylous clots that during life a catheter could be placed in a ureter, and it was observed on one occasion on which this was done that for two hours clear urine flowed by the instrument; on withdrawal of the catheter the urine again became chylous, proving that the chylous element it contained did not come as a secretion from the kidney. In the epigastric region there was a small patch of elephantiased integument. At the post-mortem examination a large sac filled with lymph, and having many divisions, was found reaching from the left side of the pelvis to the upper border of the kidney, and projecting somewhat to the right. Handling this, many hard lumps, abont the size of a walnut, could be felt, like enlarged lymphatic glands. On cutting into these thick, white, blood-streaked fluid flowed out. Thick white cords were seen extending from the mesentery to the small intestine. The

[1] Havelhing, *Virchow's Archiv*, Bd. lxxxix. p. 365.

left side of the upper part of the bladder was quite embedded in the recesses of the sac referred to, and when opened the bladder was found to be perforated in this situation.

Ponfick [1] relates a case, also in a resident of Brazil, of intermittent chyluria of eighteen years' standing. The filaria was not sought for until the post-mortem examination was made, and then was not found. In this case the lymphatics and abdominal cavity were filled with dark red clots, and long cords, like those in the preceding case, could be made out. The lymphatic glands downwards to the iliac glands were rendered conspicuous by their milk-like, dark bluish colour; the thoracic duct was as thick as a finger, rigid, with walls curled and wrinkled.

I might allude to one or two other dissections which have been recorded; but the descriptions of these are so incomplete that we are hardly justified in drawing positive conclusions from them. On the macroscopic conditions in chyluria, further evidence is very desirable, and it is to be hoped that, on the opportunity of post-mortem examinations of the subjects of this disease occurring, no pains will be spared to ascertain the exact condition of the thoracic duct and of the lymphatics of the abdomen and thorax, particular attention being given to the seat of obstruction and the way in which the filaria brings about the initial lesion of the vessel. Such investigation would be much facilitated by the injection of the lymphatic varix with some suitable material prior to the commencement of the dissection.

But, although the descriptions of the post-mortem evidences of the effects of filarial obstruction of the thoracic duct are somewhat scanty, there is abundant experimental evidence to show that when obstruction is brought about by ligature of this vessel, dilatation on the distal side of the ligature is produced; and that in animals surviving the operation for any length of time the flow of the lymph and chyle into the circulation is carried on through a system of anastomosis of dilated lymphatics. There is also abundant evidence to show that in cases of pressure by tumours to occlusion of the thoracic duct, or of its occlusion by growths in its lumen, or infarcts, a similar dilatation and anastomosis result. Filarial obstruction of the thoracic duct is, therefore, in harmony in its effects with long established experimental and pathological facts. In two, at all events, of the four cases just narrated, there can be no doubt whatever that the cause of the chyluria observed during life was the obstruction of the thoracic duct; nor can we doubt that the filaria which at one time was present was the cause of this obstruction, and

[1] *Deut. med. Wochen.* 1881, p. 624.

therefore, indirectly, of the chyluria. This, I maintain, is the pathology of all cases of tropical chyluria. I purposely say tropical chyluria. The cause of chyluria is obstruction of the thoracic duct, and this may be brought about in any climate by pressure on the duct, by tumours, by infarcts, by growths, and so forth; but in the form of chyluria endemic in the tropics, and which is the form of chyluria with which I concern myself here, the cause of the obstruction is the parent *F. nocturna;* and, I may add, for reasons already stated, this parasite is the cause of the obstruction which brings about the several diseases I have mentioned as pathologically associated and identical with chyluria.

Let the reader, with a diagram of the lymphatics before him, endeavour to follow out in his mind the effects of an occlusion of the thoracic duct, such as happened certainly in two, and presumably in all of the cases just related. There might be some little difference in the rapidity with which consequences developed, and their exact character, according as the occlusion was slowly or rapidly effected; but in any case they would be the same in character. The first effect of such an occlusion would be stasis of lymph all through the area draining into the thoracic duct. The next would be rise of pressure in the lymphatic vessels. As a consequence of this, at those points where the thoracic duct system of lymphatics anastomosed with those draining the upper part of the body, there would be a movement of lymph in the direction of least pressure, that is, towards the upper part of the body, and not any, as hitherto, towards the thoracic duct. This reversed movement of lymph, as it relieved pressure, would extend further and further in the direction of the seat of obstruction, until, as the anastomosis improved under the dilating influence of eccentric fluid distension, the pressure in the abdominal set of vessels fell to that in the vessels of the upper part of the body. But by the time this is effected the continued pressure of the accumulated lymph will have produced varicosity of some or many of the principal lymphatic trunks; so that, when the anastomosis is complete, the movement of the contents of the thoracic duct and of its branches will be completely inverted, being now along varicose vessels leading to those areas where anastomosis with the superior lymphatic system is effected.

Thus it will come to pass that the lymph coming from legs and scrotum will not enter the pelvis, but course upwards over the abdomen or back. The chyle, after entering the thoracic duct, will descend, and, traversing lumbar and pelvic glands, find a channel by the inguinal and femoral glands, scrotal lymphatics, and so to the

surface of the abdomen, and upwards to the higher lymphatics. Or the chyle may move by other routes; by the dorsal system of lymphatics, for example, or by the vessels following the aorta or portal vessels, or by way of the lymphatics of the stomach and œsophagus. In whatever direction there is least pressure to be overcome, in that direction the chyle will move. The lymphatics are subject to great variety of distribution, and their course to many irregularities—no two subjects being quite alike in this respect; so that it is impossible to say precisely by what course, in any given subject, the chyle will travel when an obstruction occurs in the thoracic duct.

Whatever route is taken it is a retrograde one, as can be proved by an examination of the contents of the various varices through which it flows. It is chyle which escapes from the varices in the urinary tract and produces chyluria; it is chyle which we aspirate from varicose groin glands, or from chylous dropsy of the tunica vaginalis; and it is chyle, as a rule, which escapes from the ruptured varices of a lymph scrotum—chyle on its way to the anastomosis which permits it to enter, and conducts it to the circulation. With the chyle will, of course, be carried the embryos of the filaria which was the original cause of all the mischief. The parent filaria, if not dead, is lying in the dilated thoracic duct, and the chyle, sweeping past her, will, in its stream, carry along the young filariæ as they are born into it. At certain points where the lymphatics are very imperfectly supported by lax tissues, as in the urinary tract, and more especially in the scrotum, superficial varices readily form, and frequently rupture, either spontaneously or as a result of a rise in the pressure of the lymph, or from a trifling traumatism.

The various degrees of milkiness, sanguineous tinting, quantity of discharge, and cessation or intermission of discharge in these diseases, are all readily explicable on this theory. The degree of milkiness is determined by the nature of the food consumed, and the stage of digestion any given sample represents. The sanguineous tinting, or even blood-like nature of the discharge in some cases, is explained by the length of time the lymph in question has been lying in the vessels. It is well known that lymph, under normal conditions, advances in development in the lymphatics, and that the lymph in the upper part of the thoracic duct is in consequence often red tinted, and contains corpuscles like those of blood; and also, that when the thoracic duct is ligatured, the lymph it contains after a time becomes sanguineous in character and appearance. This, of course, is the result of the ordinary progress in its evolution, which is not suspended during physiological or accidental delay in

the lymphatics. The intermission of discharge is explained by the alternate healing and rupture of a varix according as pressure rises or falls in the vessels from time to time. The cessation of chyluria, so frequently observable during acute disease and before death, is explained by diminished pressure in the varicose vessels permitting a rupture to heal; the limited and relatively small amount of food taken during fevers and as death approaches, and consequently the small amount of chyme absorbed under these circumstances, bringing this lowered pressure about. In these conditions the anastomosis which was sufficient, with an occasional relief by rupture, formerly to transmit all the chyle and lymph under high pressure, being now more than sufficient to transmit all the chyle and lymph at the greatly diminished pressure, and without the necessity of rupture.

The reader cannot fail to be struck with the remarkable parallelism existing between what happens in obstruction of the abdominal lymphatic circulation and in obstruction of the abdominal venous circulation, as, for example, in cirrhosis of the liver. In the former there is developed a compensatory anastomosis by various routes through which the lymphatic circulation is carried on at high pressure; there is the liability to lymphatic œdema—elephantiasis; there is varicosity of vessels—lymph scrotum, varicose groin glands; there is liability to rupture of varicose vessels—chyluria, chylous dropsy of tunica vaginalis, lymphorrhagia from scrotum; and there is the inverted circulation. In obstructed portal circulation there is a similar compensatory anastomosis following a similar route; there is a similar liability to œdema—ascites; there are the varicose veins in legs, rectum, and abdominal walls; there is a similar relief by rupture of gastric and hæmorrhoidal veins; and there is a similarly inverted circulation.

Some writers, without bringing forward any serious evidence for the position they take up, have suggested that the mechanism of chyluria is different from that which I have endeavoured to establish. For example, it is frequently stated that chyluria is produced by the embryo filariæ obstructing or wounding the lymphatic capillaries, and so permitting the contents to escape. Others again, fixing their attention on the sanguineous tint of the chyle in the urine, call the disease hæmato-chyluria, and, ignoring the well established facts of experimental physiology, suggest that in this disease the blood vessels must in some way be implicated—wounded, they say, probably by the filariæ. Apart from other considerations, that neither of these conjectures can be correct is proved by the not uncommon occurrence of instances of chyluria, evidently of filarian origin, in which the filaria having died, the chyluria still persists for

years perhaps, no embryos being present either in blood or urine. Moreover, the advocates of these views lose sight of the fact, that were the embryo filariæ the cause of the leakage from the lymphatics into the urine, by obstruction or wound of the vessels directly implicated, the case would be one of lymphuria and not chyluria; such a view gives no explanation of the regurgitation of the chyle. The fact, that chyluria may continue for years after the embryo filariæ have disappeared from the blood, is quite sufficient to prove that the healthy embryo filaria does not produce the disease. It is manifest that the parent worm in the case of chyluria is the offender; but even it, after it has once produced the obstruction of the thoracic duct, is not necessary for keeping up the disease; it may die and yet the chyluria will persist. Hence efforts to cure chyluria, founded on such theories of the pathological *rôle* of the embryo or parent worm as I have just alluded to, by attempting to kill the parasite, are as illogical as they are futile.

That this is the true pathology of chyluria, and of the majority of cases of the pathologically identical diseases, lymph scrotum and varicose groin glands, I feel sure; and I consider that it is thoroughly established by the evidence adduced here and elsewhere. There are, however, certain cases of lymph scrotum and varicose groin glands to which this pathology does not quite apply. In the majority of cases of these affections the characteristic fluid in the dilated lymphatics is chyle, or contains chyle, and the dilated vessels are a part of a system of anastomosis, along which the chyle, unable to pass an occluded thoracic duct, is travelling to join the lymphatics of the upper part of the body, and so get into the blood. But in some few cases the fluid in the scrotal and groin lymphatics is not chyle, but is simply a pure, transparent lymph without any chylous admixture whatever. If, then, the varices in these cases arise in consequence of an obstruction of the thoracic duct, and are channels for a diverted lymphatic circulation, how comes it that they do not possess all the elements and physical appearances of the contents of the thoracic duct?

There are two or three types of cases such as I refer to. In one of the types the lymph scrotum and varicose groin glands, while themselves containing only lymph, are combined with an ordinary intermitting milky chyluria with filariæ in the blood and lymph. In such cases the explanation of this apparent anomaly of chyle from one part of a varix and lymph from another, is to be sought for in the fact that, in these particular instances, the groin and scrotal lymphatics transmit only lymph from the legs and scrotum; whereas the anastomosis by which the contents of the thoracic duct are

transmitted, and which includes the lymphatics of, at least, part of the urinary tract, does not include the lymphatics of the scrotum and groin. So that in these cases to reach the circulation it is not necessary for the chylous contents of the thoracic duct to travel so far downwards as the scrotum, seeing that they may reach the blood by a shorter and more direct route. The extra and intra-abdominal anastomosis may be connected at their respective upper and lower peripheries, but it is not at all necessary that their contents should commingle throughout the varix.

In another set of cases the lymph in scrotum and groin glands is always quite clear and without chyle, but they differ from the preceding in that there is no chyluria present nor any history of it. Some of these cases admit of the same explanation as the type just referred to; there is thoracic duct obstruction, and consequent lymphatic varicosity, and the chyle gets into the blood by a higher anastomosis, and does not need to regurgitate by the roundabout inguino-scrotal route; the lymphatics of the urinary tract may or may not be involved, or, if they are not involved, they are not so distended as to rupture and produce chyluria.

Strange to say, in a proportion of these cases of lymph scrotum and of varicose groin glands with clear lymphous contents, although filaria embryos are found in the lymph, no filariæ can be found in the blood; or, if they are discovered in the blood, they are present there only in very small numbers. Twice, in investigating such cases, I have found in the lymph, not only the free embryo filariæ, but also, and along with them, unmistakable *ova*, containing active living embryos, about whose identity there could not be the slightest doubt. This circumstance, the presence of ova in the scrotal and groin lymph, combined with the fact of absence of filaria embryos in the blood, supplies, I believe, the explanation of this variety of scrotal varix, and, as I shall presently hope to show, possibly some other important phases of filaria disease. Seeing that the lymph in these cases contains embryo filariæ as well as ova, if a free passage existed to the blood the filariæ would certainly be found circulating therein as usual. Their absence from the blood proves that the scrotal lymph does not reach the circulation; that there is complete obstruction to its onward progress. The question then comes to be, Where is the seat of obstruction in such cases, and what produced it? Ova are found in the lymph, ova of an animal normally viviparous; therefore the parent filaria was, and perhaps is, aborting. Something abnormal, possibly of the nature of an accident, had happened to the mature parasite, usually so thoroughly in harmony with the organisation of its host that it gives rise to

no disease. In this premature expulsion of the products of filarial conception—this departure from the normal course of events—lies, I believe, the principal danger of filarial infection. From its bulk and shape, and passive, helpless nature, an ovum is a very different thing from the long, thin, sinuous, active, outstretched embryo filaria. The latter can easily traverse the fine capillary lymphatics into which the larger vessels divide before, and on entering the lymphatic glands; a broad globular ovum, the smallest diameter of which amounts to the $\frac{1}{750}$ inch, as compared to that of the outstretched embryo filaria, which is only $\frac{1}{3500}$ inch, cannot. Moreover, finding ova in scrotal lymph proves that the parasite which produced it lay between the lymphatic glands and the lymphatic radicles, either in the legs or scrotum, as it is manifest that the ova could not have traversed the glands and come from inside the abdomen or pelvis. Let us suppose a female filaria lying,—where she has been found more than once,—in the lymphatics of the integuments of the scrotum, and that she is aborting and expelling crowds of ova, what would be the effect on the lymphatic system of the part? The lymph stream in the natural course of things would carry the ova to a gland by the most direct and ordinary route; embolism, by the ova, of the minute afferent vessels of this gland would immediately ensue. The lymph, denied a passage in this direction, would seek a new channel, and in due course reach another gland; but, as it still carries with it the ova, embolism of this gland also would occur. A third route would then be tried, with the same result; then a fourth and a fifth, and so on until all the glands, directly or indirectly connected with the lymphatic vessel in which the filaria was aborting, would be plugged. Dilatation from rising pressure of lymph would, in due course, lead to varix (lymph scrotum); then there might be periodical relief by rupture of the attenuated walls, and the ova of the parasite would be found in the lymph if she were still aborting, or, if this had ceased, the embryos would be found in the lymph; but not a single embryo could enter, or would be found in the circulation. Hence it comes about that in some lymph scrota, and in some examples of varicose groin glands, the fluid is pure lymph and not chylous lymph; and that in a proportion of such cases no filariæ are found in the blood, although they are present in the lymph of the affected parts.

Pathology of *Elephantiasis arabum*.—Pathologists are agreed that the first step towards the production of non-congenital elephantiasis is lymph stasis; that this is usually the result of blocking of the lymphatics; and that this blocking is producible by

a variety of causes. Some of these causes are pandemic, and give rise to the sporadic forms of elephantiasis met with from time to time in all countries and in any climate. With these forms of sporadic elephantiasis we are not concerned. The cause of the lymph stasis leading to the *endemic* form of elephantiasis is strictly an endemic influence, something special to particular districts and climates, and also something which these districts and climates have in common.

Of the many things which from time to time have been cited as causes of *E. arabum*, such as erysipelas, lymphangitis, malaria, working in wet rice-fields, walking barefooted, injuries, particular articles of food, syphilis, and so forth, it is sufficient to say that such are either pandemic in their prevalence, or, if perchance existing in one locality where elephantiasis is endemic, are not to be found in other localities where the disease is equally common. Malaria is constantly cited as a cause of elephantiasis—as it is of almost all tropical diseases for which the cause is not very evident; we know, however, that there are districts in which malaria is particularly rife, but in which elephantiasis is rare, or entirely absent as an endemic disease, and *vice versâ*. And so it is with many other of the reputed causes of elephantiasis. There is no substantial body of evidence for attributing this disease to any of the things mentioned; all that can be said of their relationship to elephantiasis is that it is one of occasional and accidental coincidence; or, at most, that they determine the explosion of elephantoid inflammation in tissues already prepared by lymphatic congestion. No reasonable theory has as yet been propounded as to the way such supposed causes operate etiologically, in the proper sense of that term.

Corre has lately advanced the theory that residence in particular climates and countries gives rise to a state of constitution characterised by a special susceptibility, on the part of the lymphatic system, to various morbid influences. This condition of system, and the group of diseases he supposes it to lead up to, he calls "lymphathexie endemique"; and he suggests that when "lymphathexie" is present, any or many forms of irritation, which, under conditions of more perfect health would be innocuous, give rise to the various forms of elephantoid disease. I fail to see the necessity for the assumption of this condition of "lymphathexie" to explain these diseases; the only evidence of its existence is the diseases themselves. Corre is wrong, too, I think, in encouraging, as he does, the idea that race has anything to do with the manifestation of elephantoid disease. In my opinion, any difference we may remark

in the frequency with which one race of men is attacked with the elephantoid diseases, compared to other races of men, is attributable, not to differences of susceptibility, but to differences of opportunity. It is only necessary to quote the experience, as regards elephantiasis, of Europeans in the South Pacific Islands to prove this.

My own belief, arrived at after considerable study of the question, is that in an important section of the cases, if not in every case of endemic *E. arabum*—not sporadic elephantiasis—the cause of the lymph stasis is occlusion of the lymphatics, and the cause of the lymphatic occlusion leading up to the lymph stasis, and finally ending in permanent disease, is the *F. nocturna*. The following are the grounds on which I found this opinion :—

(*a*) As far as known, the geographical distribution of the parasite and the disease correspond.

(*b*) *F. nocturna* has been proved (proved as far as proof is possible in pathological problems) to be the cause of what I have, for convenience, termed the elephantoid diseases.

(*c*) A considerable number of cases have been recorded in which these diseases concurred with genuine *E. arabum*.

(*d*) Cases have been recorded of lymph scrotum gradually passing into *E. arabum*.

(*e*) A common history to receive from patients suffering from elephantiasis of the scrotum is, that the disease at first was accompanied at times by a lymphous discharge, *i.e.* that it was originally a lymph scrotum.

(*f*) Cases have been recorded in which a condition resembling lymph scrotum had supervened in elephantiasis of the scrotum, or been developed in the cicatrix or morbid tissues remaining after operation for *E. scroti*.

(*g*) The elephantoid diseases are essentially diseases of the lymphatic system ; so is *E. arabum*.

(*h*) The geographical distribution of the elephantoid diseases, as far as known, corresponds with that of *E. arabum*.

(*i*) The elephantoid diseases are associated with a peculiar type of inflammation and fever ; the same type of inflammation and fever is almost invariably recurrent in elephantiasis.

(*j*) On the supposition that the elephantoid diseases have a different etiology from *E. arabum*, we are driven to assume what is extremely improbable, viz. that there are two distinct diseases of the lymphatic system, which affect the same parts of the body, which are characterised by the same type of inflammation and fever, which frequently concur or follow each other in the same

individual, which are co-endemic, and which have the same geographical distribution.

(*k*) Were the filaria constantly found in the blood in cases of elephantiasis, which is far from being the case, its constant presence there would be considered sufficient proof—taking it in conjunction with the foregoing considerations—of its etiological relationship to the disease; but because the filaria is only occasionally present in the blood in these cases, in fact more often absent than present, it is illogical to infer from this circumstance alone—as so many have done—that the filaria cannot be the cause of the disease, or that it never had been present in the affected individual.

(*l*) Cases of elephantoid diseases occur in which the presence of the filaria cannot be demonstrated in the blood, although it is certain that the disease in such cases is attributable to the filaria. The absence of the filaria from the blood in such cases is attributable either—1st, to death of the parent worm; 2nd, to occlusion of the lymphatic area in which the parent worm is lying, so that it is impossible for the embryos, although their presence may be ascertained in the lymph, to make their way into the blood; 3rd, only male filariæ or unimpregnated females may be present.

(*m*) In the same way, in *E. arabum*, the absence of embryo filariæ from the blood is to be explained. The fact of elephantiasis supervening in a limb is evidence that the lymphatic system of that limb and the lymphatic systems connected with it by anastomosis, that is, the lymphatic systems of the scrotum and of the other leg, are completely cut off from the blood circulation. Therefore it is that the embryos of the filaria or filariæ which originally produced the occlusion cannot possibly enter and be seen in the blood. Consequently, in a filariated population, those affected with elephantiasis are less likely to have filariæ in the blood than those not so affected, the decrease of liability bearing some relation to the proportion the dimensions of the lymphatic system of the lower extremities bears to that of the total lymphatic system. In a filariated district, as I shall show, the subjects of elephantiasis are the least likely to have filariæ in the blood. This fact is constantly forgotten, and consequently we frequently read in reports of cases of elephantiasis that the filaria was sought for many times but was not found; the writer of the report, as a rule, concluding against the filaria being the cause of elephantiasis, and solely on this account. Properly interpreted, the absence of filaria embryos in the blood in elephantiasis is a strong argument in favour of the doctrine of the filarial origin of this disease (see p. 832).

(*n*) Seeing then that *E. arabum* frequently concurs with the

elephantoid diseases, often follows them, affects the same set of vessels and the same regions of the body, that it is associated with the same type of fever and inflammation, and is endemic in the same localities, it is reasonable to conclude that it depends on the same cause. This cause being proved to be the *F. nocturna* in the elephantoid diseases, the *F. nocturna* must be the cause of elephantiasis.

But although we may with considerable confidence assert that this is true, in the present imperfect state of our knowledge of the subject it is impossible to say possitively what is the exact way in which the filaria gives rise to elephantiasis. My view is, that those cases of lymph scrotum and varicose groin glands in which the contents of the lymphatic varix are lymphous and not chylous (that is to say, in which the lymph comes from the legs or scrotum, and is not regurgitating from thoracic duct and lacteals), and in which filaria embryos are found in this lymph whilst they cannot be found in the same case in the blood, — these cases, I hold, supply a key to part, at least, of the pathological problem which elephantiasis presents. The absence of filaria embryos from the blood in such cases manifestly shows that the glands may become so blocked that filaria embryos cannot pass them; and the varicosity of the lymphatics proves that the lymph circulation, if not completely arrested, is, at least, very seriously retarded. Then as to the way in which the occlusion is brought about, the discovery of filaria ova in the lymph in scrotal and gland varices supplies the requisite hint. It is unnecessary to repeat what I have already said (p. 822) as to the effect on the lymphatic system of the lower extremities of an aborting filaria; suffice it to say here that the weight of evidence, as far as it goes, is to show that the lymph stasis which leads to elephantiasis is owing, in some instances at all events, to occlusion of lymphatic glands by the ova of an aborting *F. nocturna*.

In elephantiasis of the legs or scrotum, in the great majority of cases, the parasite is, I hold, located below the lymphatic glands and in the legs or scrotum. A degree of lymph stasis would be produced by occlusion of the thoracic duct, and it is quite possible that a proportion of cases of elephantiasis have their origin in this, just as chyluria has; but if the point of occlusion in elephantiasis were generally in the thoracic duct, I think we would find the embryos of the filaria more frequently in the blood in this disease than we do, that, in fact, we would find them as frequently as we do in chyluria; this is far from being the case. I conclude, therefore, that the usual first step in the production of

elephantiasis is an aborting filaria on the distal side of the groin glands.

In an area of lymph stasis, such as precedes elephantiasis, and such as this occlusion would induce, slight causes suffice to bring about lymphangitis and forms of erysipelas. These inflammatory attacks result in effusions which, owing to the state of the lymphatics, are imperfectly absorbed. Consequently, there is gradual enlargement —partly inflammatory, partly passive—of the affected parts, and this is elephantiasis.

Experiment has shown that if the thoracic duct, or the lymphatics of any given area are tied, the lymphatics below the seat of ligature become gorged with lymph. No dropsy, however, ensues; the veins, apparently, are competent to remove all the fluids which formerly were, in part at least, taken up by the lymphatics. But should the veins become blocked as well as the lymphatics, or their free action be interfered with in any way, as by too dependent a position, pressure by tumours, phlebitis, thrombosis, or experimental ligation, then dropsy is sure to ensue. Or, if in a region of lymph stasis inflammatory effusion is thrown out, though the veins may continue patent and effective, the more solid part of the inflammatory effusion is not removed. These facts have over and over again been demonstrated experimentally. They have an important bearing on the pathology of elephantiasis, and explain many things in connection therewith.

The order of events in the production of a case of *E. arabum* I hold to be: occlusion of lymphatics by the filaria, or some product of the filaria, very likely by its ova plugging the glands; stasis of lymph; lymphangitis or erysipelas, readily induced by slight traumatism or other morbid influence in congested tissues; effusion of inflammatory products; imperfect absorption of these by the veins; permanent thickening of the involved area; recurrence of inflammatory attacks; remitting but gradual enlargement of the affected parts. Or—stasis of lymph from filarial occlusion of lymphatics; incomplete removal of unused balance of the nutritive plasma transuding normally from the blood vessels, in consequence of concurring imperfect venous circulation; gradual enlargement of the affected parts. Or there may be, and probably there usually is, in the course of the development of the majority of cases, a concurrence of both of these conditions.

There is no point connected with *E. arabum* which, so far as I know, cannot be explained by this hypothesis. As I have stated, the absence of embryos from the blood, so frequently observed in elephantiasis, has been a great stumbling-block to the general acceptance

of the doctrine of the filarial origin of this disease. This absence, however, as I have shown, is readily explained; indeed, it is inevitable in most cases. It would be as absurd to condemn, on this account, the doctrine of the filarial origin of elephantiasis, as it would be to condemn the doctrine of the filarial origin of what I have designated the elephantoid diseases, merely because the filaria cannot be found in every case. If Dr. S. Mackenzie's case (p. 812), for example, had survived the septic trouble which led to his death, he would still have had an occluded thoracic duct—still have been subject to attacks of chyluria, but his blood would certainly have been free from filaria embryos; the parent worm had died, the varix it gave rise to remained. It is a fact that not unfrequently cases of admittedly filarious disease are encountered in which, owing to antecedent death of the parent worm, embryos are necessarily absent from the blood. It is not, I repeat, the continuous action of a mature worm, or of the embryos, which produces disease, as some have suggested; it is the permanent occlusion of the lymph tract they have given rise to, and, as long as this remains, so long will the subject of it be liable to lymphatic disease. The parasite may die, but the mischief it wrought in the lymphatics survives. This is too obvious, and there are too many examples of the same thing in pathology to require further elucidation. But in developed elephantiasis, even supposing the parent worm is still alive, her brood cannot possibly, in most cases, reach the blood; the road is blocked at the glands. My opinion is, that the parent worm does not long survive the occlusion of the glands and stoppage of the lymph flow; her proper habitat is running lymph. If she happen to be in the thoracic duct, a free anastomosis permits the circulation of lymph; and if she happen to be connected with a lymph scrotum, the circulation of lymph in the anastomosis or the occasional escape of lymph by rupture of a varix will permit a flow of lymph past her, and so she will keep alive; but complete or nearly complete stasis, such as occurs in developed elephantiasis, is fatal to the worm; and I think the frequency of abscess about the scrotum and thighs in connection with elephantiasis often has its origin in this way in a dead filaria. I have found the remains of the parasite in such an abscess.

The rarity of elephantiasis of the upper extremities, of the breast and the upper part of the body generally, is explained by the readiness with which the blood and lymph return, assisted by gravitation, from these parts. That the filaria is often present in the arms we know,—the mature worm has been found in this situation more than once,—and undoubtedly gives rise to lymphatic stasis

there as well as in the legs; but the blocking of the lymphatics in this situation is readily compensated for by the greater facilities for venous return, and possibly a freer lymphatic anastomosis than exists in the case of the legs. The arm does not readily take on the elephantoid process, for the same mechanical reasons which render it slow to become œdematous in diseases involving the circulatory system. I believe that when elephantiasis develops in an arm or a breast, some imperfection in the venous system as well as of the lymphatics of the part is present, and conduces powerfully to the result.

The fact that in elephantiasis of one leg not only are the lymphatics of the affected side involved, but also the lymphatics of the healthy side, as shown by the enlargement of the glands, is explicable on the supposition that the lymph stasis is such as would be induced by an aborting filaria; for her ova would be carried, by way of the scrotal and pudic lymphatics, to the leg opposite to the one she was lying in as soon as all the glands were plugged in this. It is well known that the glands of both sides are enlarged in some cases, although the elephantoid manifestation in the shape of thickening of the integuments is apparent in one leg or part of one leg only. By a similar process of reasoning we may explain the appearance of the so-called metastasis which is so common in the early stages of elephantiasis; the well-known liability to the development of elephantiasis of the legs, after removal of an elephantiasis of the scrotum or of a lymph scrotum; all these circumstances are readily explicable on the supposition that an aborting filaria in the lymphatics is the original cause of the morbid lymph stasis, involving the entire lymphatic system below the pelvis.

Much might be done to support or confute the doctrine of the filarial origin of elephantiasis by the collection and compilation of reliable statistics. In the first place, the evidence showing that the respective geographical distributions of the parasite and of the disease correspond might be very much expanded. It would be an easy matter for medical men—if they were resident on the spot, or even as visitors only—to ascertain if centres of filariasis are also centres of elephantiasis, and *vice versâ*; or if in warm countries where the filaria is not to be found elephantiasis is also absent. It is manifest that if in any country it is found that elephantiasis is endemic, whilst the filaria is absent, or *vice versâ*, then the doctrine of the filarial origin of elephantiasis falls to the ground; either this, or we must conclude, and the possibility of this I would not deny, that there are two forms of elephantiasis, a filarial and a non-filarial.

The islands of the South Pacific offer a splendid opportunity for investigations of this sort. There, in one island, elephantiasis may be extensively endemic, whilst in a neighbouring island it is almost unknown. It would be an easy matter to ascertain if there is a corresponding caprice in the distribution of *F. nocturna*. The collection and examination of 100 slides of night blood prepared in the simple manner I recommend (pp. 847–851), from the inhabitants of any country, is all that is necessary to settle the question of the presence or absence of filarial hæmatozoa there. It is a pity that so important a point in geographical pathology should be left undetermined.

I have myself lately made an attempt in this direction in the case of two countries—one selected because elephantiasis is not endemic in it, the other because elephantiasis is particularly prevalent there. The two countries I refer to are South Africa, where there is no or very little elephantiasis, and Cochin (India), where, as already pointed out, elephantiasis is excessively common. About neither of these countries, so far as I am aware, was there any previous knowledge as to the presence in them or absence of the *F. nocturna*. This is what I found. Through the kindness of Dr. Newton Burns I received 74 slides of blood taken at night (I had also corresponding slides of day blood) from 74 South Africans from different parts of the coast, and comprising Kafirs of different tribes, Zulus, Fingos, Basutos, Mashonas, Hottentots, and other races. These slides reached me in good condition, and they were carefully stained and examined. In none of them were filariæ embryos found. I conclude, therefore, that in South Africa *F. nocturna* is practically if not entirely absent. Through the kindness of Surgeon-Major Elcum I received 88 slides of night blood taken from 88 natives of Cochin. In 21 of these slides I found *F. nocturna* in great abundance. So that as regards these two countries—South Africa and Cochin—filariasis and elephantiasis have a corresponding geographical distribution.

In the collection sent me by Dr. Burns there were really 75 slides from 75 individuals. On examining one of them I discovered in it *F. nocturna* in abundance. Looking at the label on this slide, I found it marked "Zanzibar"; so that the blood-giver in the case of this slide came from equatorial Africa, and from a place where elephantiasis is notoriously common. The circumstance is not without significance.

Another point in furtherance of the evidence for or against the doctrine of the filarial origin of elephantiasis might also be cleared up by the statistical method I suggest, and that is: Does the filaria,

as I have advanced, cause elephantiasis by obstructing that part of the lymphatic system in which she is lying? If this view be correct, then in countries in which elephantiasis and filariasis are co-endemic, the proportion of individuals among the healthy general population having filariæ circulating in their blood at night must be greater than it is among those affected with elephantiasis. The *rationale* of this I have already explained. The slides I received from Cochin, so far as their numbers go, have an important bearing on this point. Of the 88 slides, 74 were marked from "healthy" individuals, and 14 were marked "elephantiasis." Of the former, 20 contained filariæ; of the latter, 14 in number, only 1 contained filariæ. Assuming that these slides fairly represent in their relative proportions the healthy and the sufferers from elephantiasis in Cochin, we are to conclude that the proportionate liability of individuals of the general population to have *F. nocturna* circulating at night in the blood to those not so infected is as 1 in 3·7; whereas in those affected with elephantiasis it is as 1 in 14. Assuming that elephantiasis is caused by the *F. nocturna*, the liability of the Cochinese to filariasis is as 34 in 88; for, to arrive at a correct estimate of this, we must add the number of individuals affected with elephantiasis—a filaria caused disease, but in which the filaria does not appear in the circulation—to the number having filaria embryos in the blood.

These figures are small, it is true, but, so far as they go, they bear out all I have advanced as to the filaria being the cause of elephantiasis, and also as to the particular way the parasite brings about this disease.

The reader may think that I have devoted too much space to the consideration of the etiology and pathology of these diseases. My apology is the complexity of the subject, the necessity for detailing work not generally accessible and much of it hitherto unpublished, and the fact that unless our knowledge on cardinal points be exact and sufficient we are not in a position to advance the subject or to advise with regard to the very important points of treatment and prophylaxis. Apart from the biological interest of the life history of the filaria, which constitutes one of the most marvellous of "the fairy tales of science," there can be no question about the practical importance of the subject; the fact I have just mentioned, that nearly half of the inhabitants of some, probably of many, parts of the world are the subjects of filiariasis, attests this. If we reflect upon the vast amount of suffering, deformity, and even mortality which in the aggregate is produced by *F. nocturna*, it is evident that no zooparasite attacking man approaches it in importance.

The Treatment of Filaria Nocturna Disease.

As yet no means are known by which, once established in the human body, *F. nocturna* can be killed or removed. The locality occupied by the parent worm cannot even be guessed at until the parasite has given rise to disease; even then it is rarely possible to locate it with sufficient precision to justify an attempt at removal, and even in the very few instances in which removal might be possible, interference would come too late, as the damage would have been already done. Our efforts, therefore, have to be confined to prophylaxis and to the mitigation of established disease.

Prophylaxis.—The knowledge we already possess of the life history of *F. nocturna* enables us to indicate with precision the proper steps to take to prevent its entry into the human body; and our knowledge of the pathology of the diseases it gives rise to enables us to indicate means which may contribute to prevent the development of these diseases, and to mitigate their effects when they do arise.

The time may come when communities shall endeavour to protect themselves against the ravages of this parasite; the methods they shall then employ will have reference principally to water supply. A pure water supply, uncontaminated by mosquitoes, is the first essential; given that, the elephantoid diseases and elephantiasis will disappear from a community. Tank water or stagnant water of any kind ought not to be used for drinking purposes; wells and reservoirs ought to be protected by a fine-meshed netting or effective cover of some sort to keep out mosquitoes; vessels used for storing water must be frequently emptied, cleaned, and always kept covered up; drinking water, unless beyond suspicion, ought to be boiled or filtered. Persons known to harbour the filaria ought to be looked on as sources of danger to the community, and shunned or compelled to sleep under suitable mosquito curtains. By these means *F. nocturna* could be stamped out, and the diseases it causes made matters of history. Their ultimate disappearance is entirely a matter of personal and municipal education—in other words, of civilisation. It is too early in the day, and somewhat optimistic, to look for special efforts on the part of governments in this direction, but if any municipal or other body is in want of one more argument for a pure water supply, here is one ready made to their hands.

If an individual should know that he himself harbours *F. nocturna*, he ought to endeavour to avoid all sources of lymphangitis, such as wounds of the extremities; excessive exposure to sun, rain, or chills;

prolonged walking or standing—especially in water, as in paddy fields; alcoholic stimulants, and excesses generally. He ought to wear shoes, and have his legs properly protected from thorns, blows, and insect bites. If the locality he lives in is very malarial, he ought to leave it if possible, and seek out a cool, dry climate in preference to a hot and damp one. Such skin diseases as scabies, ringworm, phthiriasis, and eczema ought to be promptly treated, and by non-irritating means, and every effort ought to be made to keep the skin clean and wholesome, and the general health good. Should he succeed in avoiding lymphangitis, he will probably escape elephantiasis.

Treatment.—*Lymphangitis* must be treated on general principles when it occurs. Rest, elevation of the affected part, salines, fomentations, and anodynes are indicated; and, if malarial complication is suspected, the free use of quinine. Where effusion is great, and in circumstances where proper antiseptic dressings are available, great relief will be afforded by pricking or scarifying the inflamed part, and wrapping it in salicylic or boracic wool. In this way large quantities of stagnant lymph will escape, and much subsequent deformity be avoided. When the acute symptoms are over, a short course of mercury or of iodide of potassium might be beneficial, either alone or combined with carefully applied and graduated elastic bandaging, and gentle massage with some bland ointment or oil to prevent undue friction. By these means absorption of effused lymph is promoted, and the almost inevitable progress of the disease somewhat retarded.

Abscess, when it forms, must be freely incised and drained. *Orchitis* should be dealt with on ordinary surgical principles; enlargement of the testis after orchitis should be treated by strapping and suspension, and, in suitable cases, by mercury.

Chyluria.—The principles which ought to guide us in the treatment of chyluria are clearly indicated by the pathology. Our efforts ought to be directed to getting the ruptured varix, by which the chyle is leaking into the urine, to heal. The most important and potent of the means to this end at our disposal are—1st, rest in bed, by which a certain amount of hydrostatic pressure is taken off the leaking, ruptured lymphatic, and the necessity for full diet obviated; 2nd, an albuminous and farinaceous diet, with a minimum of fluid and fatty food, by which the amount of chyle passing through the lacteals and thoracic duct is diminished.[1] By these

[1] The fatty element in chylous urine can be very quickly caused to disappear by such a diet. I have many times effected this. But although the milky appearance of the urine may pass away the presence of a clear gelatinous clot, lymph corpuscles, and albumen too often

means the rupture of the lymphatic is afforded the best chance of healing. Certain drugs have been recommended in chyluria, and have enjoyed a perhaps not very well deserved reputation. Amongst these may be mentioned benzoic acid, benzoate of soda, glycerine, tannic acid, gallic acid—all in very large doses; salts of iron, chromic acid, quinine, decoctions of mangrove bark (*Rhizophera racemosa*), and of the seeds of *nigella sativa*. Lately, Surgeon Lieutenant-Colonel Lawrie[1] has advocated thymol in 2 grain doses four times a day, maintaining that it is not only curative as regards the chyluria, but also fatal to the filaria. As his conclusions are drawn from the experience of one or two cases only, and as chyluria is well known to get well temporarily spontaneously, and as Dr. Lawrie gives us no details of the methods he employed for determining the absence of the filaria in the blood of his patients, we must wait for further experience of this drug before we can say that thymol is as efficacious against *F. nocturna* as it is against *Anchylostoma duodenale*.[2] Personally, I have no belief in the power of any drug either to cure chyluria or to kill the filaria. I think that, as a general rule, our care should be to protect rather than to irritate or kill the parasite. A dead filaria in the thoracic duct, or a dying and aborting filaria in the crural lymphatics, are much more dangerous in my opinion than a healthy, living parasite. On the other hand, I have a strong conviction that by rest and suitable diet, chylous leakage into the urine may be, at least temporarily, suspended.

Chylous Dropsy of the Peritoneum and Tunica Vaginalis.—In these, if they are causing inconvenience, tapping is indicated; followed, in the case of the tunica vaginalis, by injection with

remain to testify that leakage is still going on from the lymphatics of some part of the urinary tract. Dr. de Arze (*El Progresso Medico*, March 1893) advocates this dietetic treatment of chyluria.

[1] *Lancet*, February 14, 1891; November 26, 1892.

[2] I have tried this drug unsuccessfully as an anthelmintic in the case of *F. diurna* and *F. perstans* (*Lancet*, October 1, 1892); and in a recent *Lancet* (August 15, 1892), Surgeon Lieutenant-Colonel Crombie proves that, even in very large doses, it is quite powerless to kill the filaria in the blood, or to cure chyluria. Similarly negative results have been arrived at by Mr. Williams, of Norwich (*Lancet*, June 17, 1893), and by myself (*Lancet*, February 18, 1893). Magalhães informs me that he, too, has been equally unsuccessful with thymol in Rio de Janeiro. He gave the drug to two filaria patients—to one, a woman suffering from lymphatic varix of the thigh, for over three months; to the other, a man who had been operated on for chylocele, for over two months, and until his stomach was upset by the drug. Slides of evening blood, taken from these patients after this prolonged course of treatment, Magalhães, very kindly, sent to me; they contained many filariæ. I fear, therefore, that the expectations we were led to entertain from Lawrie's experience of the power of thymol as an anthelmintic in filariasis must be abandoned. Similarly, Magalhães' suggestion of glycerine has had to be given up; and I suspect the same fate is in store for M. Zune's recommendation of rectal insufflation with sulphuretted hydrogen.

iodine, or, this failing, free aseptic incision. If the swelling is not causing inconvenience it had better be left alone.

Varicose Groin Glands.—The question of removal of these may arise. The experience of French surgeons—Nélaton, Trélat, and others—has shown that in pre-Listerian times interference with these tumours was attended with very great danger from septic lymphangitis. It may be different in this respect nowadays, but still the operation is one which should not be lightly undertaken. It is well to remember that the dilated vessels constituting these swellings are but part of an enormous varix through which the chyle and lymph are finding their way, very probably over the surface of the abdomen, to the lymphatic system of the upper part of the body. Therefore to excise them is to interfere more or less with a physiologically necessary anastomosis, damage or curtailment of which at one point must be compensated for by fresh developments elsewhere, and, for a time at least, increased pressure in the remaining vessels. Moreover, to excise one of these tumours risks the starting of a chyluria, of a lymph scrotum, or of an elephantiasis, in addition to the immediate dangers from the operation. Nevertheless, circumstances may arise which justify operation. Such a case was successfully dealt with lately by Surgeon-Major Maitland. In this case the tumour gave rise to so much pain and inconvenience that operation was considered justifiable. The immediate result was excellent; but, Dr. Maitland informs me, three years afterwards the disease is appearing in the other groin. In some cases of varicose groin glands, relief might be afforded by a carefully adjusted bandage. Azéma says that in time they subside spontaneously; I presume, when in the course of years the lymphatic anastomosis has become so perfect and sufficient that all eccentric fluid pressure is removed. I have never myself seen one of these varices subside spontaneously.

Lymph Scrotum.—The same rules, with regard to operative interference, which apply to varicose groin glands apply to lymph scrotum. If it appear to be an efficient part of a compensatory varix, and be giving rise to no particular distress, it ought to be left alone. But if it be the seat and starting-point of frequent attacks of lymphangitis, be damaging the general nutrition by repeated and prolonged attacks of lymphorrhagia, and be seriously in the way of the patient's making a livelihood, it ought to be removed. This is easily effected. The mass of the scrotum is pulled well down with the left hand, whilst an assistant presses the testicles upwards and keeps them out of the way; the redundant scrotum may then be transfixed as near the testes as may be and, by an upward and downward cut, rapidly

removed. Sufficiently large flaps, free from superficial varices or disease of any kind, are generally available, and can be stitched together by closely set sutures so as to completely cover in the testicles. The wound, if properly managed, heals rapidly. I have frequently practised this operation, and never saw any bad result, so far as the life of the patient was concerned; but I have seen chyluria follow, and, also, elephantiasis commence in a leg. When in lymph scrotum the varices have ceased to discharge for some time, and the tumour begins to grow rapidly, and to take on the characters of elephantiasis, it shows that the lymphatic varix is no longer available for lymph or chyle circulation; in this case it ought to be removed as soon as possible. If the discharge from a lymph scrotum be lymphous and clear, and contain filariæ, whilst, at the same time, filariæ are absent from the blood, there is a strong presumption that in such a case the parent filariæ are lying in the scrotum. In such a case the parent worms ought to be carefully sought for in the amputated scrotum. As much has still to be learned about the zoological characters of the male worm, surgeons ought not to neglect the chances afforded by such operations of supplying the important details which are still wanting.

Elephantiasis of the Leg.—Rest, elevation, massage, and elastic bandaging constitute the most generally approved treatment for elephantiasis of the leg. They do not cure, but they alleviate in a marked degree; other methods of treatment, sometimes credited with curative powers, really owe much of their supposed efficacy to their being combined with these common-sense measures.

Of the many other methods which from time to time have been tried, I may mention the exclusively milk diet treatment of Richards, the mercurial course recommended by Bentley, electrolysis as extolled by Silva Araujo,[1] and ligature of the femoral formerly practised by many surgeons. I have no experience of any of these methods. Ligature of the femoral artery is now generally condemned as being not only useless and opposed to pathological indications, but often fatal.

Several times I have endeavoured to lighten the burden of an unusually heavy leg by dissecting off, in the direction of the long axis of the limb, strips of thickened integument. This affords some relief at the time, and the subsequent contraction of cicatricial tissue tends to prevent further enlargement.

Elephantiasis of the Scrotum.—If the scrotum be affected with elephantiasis to any great extent, and especially if it be growing rapidly, it ought to be removed.

[1] *Atlas des maladies de la peau.* Rio de Janeiro, 1887.

Before operating there are certain points which, as a matter of routine, the surgeon ought to attend to. These are—1st, in malarial countries, the administration of quinine in 5 grain doses three times a day for three or four days; 2nd, the elevation of the tumour to drain it of fluids and render it lax and manageable; 3rd, the localisation by palpation of the testes, and inquiry about the possibility of non-descent of one or both; 4th, the presence or absence of hernia; 5th, thorough purifying of the parts with an antiseptic.

Formerly, before the introduction of the elastic cord for controlling bleeding, the hæmorrhage at these operations was often very alarming; nowadays, with a properly applied constricting elastic ligature, very large tumours may be successfully removed with the loss of only a few ounces of blood. The entire blood in the tumour may be readily forced out of it and into the general circulation by pressure with an Esmarch bandage, and almost all loss of blood be completely prevented; but the propriety of this, considering that it is liable to surcharge the circulation and perhaps embarrass the heart, is questionable; unless, perhaps, in cases of marked anæmia.

It is well to provide, in the case of large tumours, some means, such as a block and tackle, or a triangular supporting board hooked on to the end of the table, to facilitate the moving of the mass; and, on account of the great weight which has to be manipulated, no operation of this description should be undertaken without plenty of assistants.

Before applying the constricting ligature to the neck of the tumour it is well to map out by a shallow cut in the skin the line at which it is proposed to amputate, and, if flaps are to be made, to define in this way their outline.

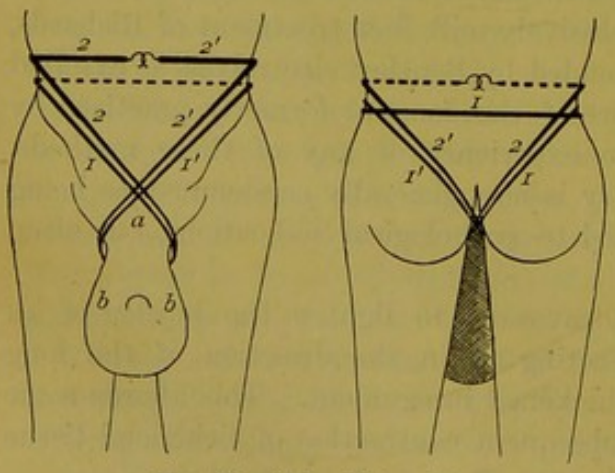


FIG. 75.—(After McLeod.)

There are two good methods of applying the constricting ligature. McLeod[1] recommends the following procedure (Fig. 75):—Laying the middle of the cord across the small of the back, bring the two ends above the crests of the ilia, crossing them over the pubis, the left end of the cord passing to the right side of the neck of the tumour,

[1] *Operative Surgery.*

the right end to the left; stretching the cord, wind the ends round the sides of the neck of the tumour, crossing again in front of the anus, and passing to their respective sides round the buttocks and over the crest of each ilium, down again across the pubis, round the tumour once more, again crossing in front of the anus, and so up over the buttocks and iliac crests, finally uniting their ends across the abdomen. A modification of Partridge's method is the one which I generally practised; although perhaps not the best, it is quite effective. It is as follows:—Tie a strip of bandage cloth somewhat loosely round the waist. Underneath this slip the upper ends of four strips of bandage, each about 3 feet long, two in front—one piece falling over either groin and on to the front of the tumour, and two behind—one piece falling over either buttock and under the tumour. Keeping these strips of bandage close to the mass, wind the rubber cord firmly over them and round the neck of the tumour several times, uniting its ends in front. Next tie firmly the two ends of each bandage together over the waist belt, so that they shall slightly yet decidedly drag the cord upwards against the pelvis, and thus prevent it from collapsing or slipping down when the final cuts of the operation are made in its neighbourhood. Having thoroughly satisfied himself with the efficiency of the constricting ligature, the surgeon proceeds with the operation.

A long incision over one spermatic cord is carried from the guide mark on the skin near the pubis down to the lower end of the testicle, passing well through the thick rind; the loose blubbery tissues are partly torn and partly cut through down to the testicle; a finger is then slipped round the strong fibrous band—the remains of the gubernaculum testis—which will be found running between and binding the testicle to the bottom of the scrotum, and hooking this band up, it is divided with scissors. The testicle is then easily thrown up along with the spermatic cord. The other testicle and cord are dealt with in the same way. The penis is then dissected out by slitting up the tunnel leading to the glans and prolonging the incision to the guide mark in the skin above; the prepuce is divided all round the penis close to the glans and, dragging on the glans with one hand, the penis is easily shelled out as far up as its attachment to the pubis. The flaps are then dissected up if it is thought desirable to preserve any, and the guide mark across the perineum, uniting the posterior ends of the flaps, deepened to about an inch; the upper ends of the incisions for penis and testicles are united by a transverse cut; and the penis and testicles and flaps being held up and well out of the way of the knife, the remainder of the neck is divided as high up as possible

without wounding the perineal fascia. The preliminary shallow cut is a valuable guide at this step of the operation, insuring that sufficient, and yet not too much tissue is removed. Those vessels which can be seen are tied; the constricting ligature is then removed, and any further bleeding promptly dealt with. If hydroceles are present they are now slit up, and redundant tunica vaginalis cut off. If there are no flaps, or if these are too small to cover the testes, these glands are united by bringing their inner borders together with a stitch or two carried through the remains of the tunica vaginalis, and they are further secured in a suitable position on the face of the wound by stitching the remains of the outer edge of the tunica vaginalis to the skin or to some convenient tag of tissue, or pockets may be dissected out for them on the face of the perineum. If flaps have been made they are now to be stitched together over the testes, a T-shaped wound resulting with the penis emerging from the point of junction of the horizontal with the perpendicular limb.

The dressing ought to consist of a piece of protective silk, with a hole cut in it to take the penis; the penis ought to be wrapped in a separate strip of the same material. Fibrous dressings, such as lint, if applied to the wound, adhere and cause much bleeding and pain when the parts come to be dressed again. Over the protective is laid a massive antiseptic dry dressing, and over this an eight-tailed bandage with a hole for the penis. The two outer tags—front and back of one side—of this bandage are tied together over the corresponding thigh; the two front inner tags are tied over a piece of bandage encircling the waist, and the two back tags are similarly disposed of behind. This makes a firm, easily adjusted, and easily removed dressing. When the wound begins to granulate, healing ought to be encouraged by skin-grafting. Especial care must be taken that the penis does not become adherent to the other parts of the wound or retracted in any way, and that it shall preserve its proper proportions; it is well, therefore, to apply numerous skin grafts at as early a period as possible around it, especially at the root.

In these operations the penis and testes, unless very gravely diseased, ought always to be preserved. Serious mutilations, such as were formerly practised, only add to risk, and are never necessary, besides being for other reasons exceedingly undesirable.

The mortality from this operation, when properly performed, and with proper antiseptic appliances, ought not to exceed 5 per cent.[1]

[1] In 136 operations, on tumours ranging in weight from 7 to 80 lbs., Dr. Turner, of Samoa, only lost two cases. (*Glasgow Med. Journ.*, June 1882.) In 120 cases operated on in Amoy, China, I lost five cases—four of them from septic disease, one from tetanus. Since the introduction of antiseptic methods the mortality in India has fallen from 17 or 18 per cent. to about 5 per cent.

Elephantiasis of the vulva, clitoris, or breast ought to be removed by operation before it has attained inconvenient dimensions.

FILARIA SANGUINIS HOMINIS PERSTANS. (Figs. 76, 78.)

Nothing is known of the mature form of this parasite. The embryo as it is seen in the blood I have already described. Its peculiar anatomical and physiological characters, and the entire absence of periodicity in its habits, are quite conclusive as to its

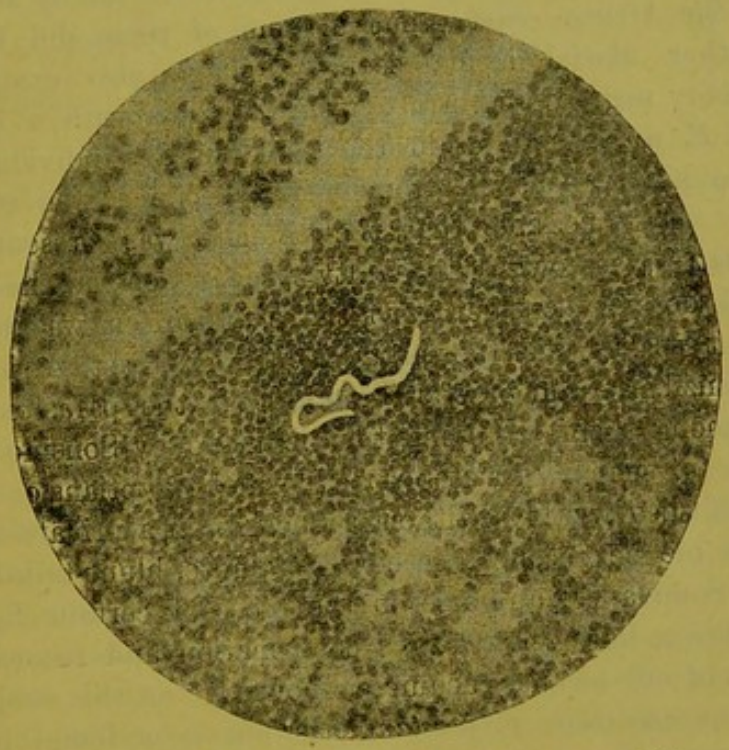


Fig. 76.—*Filaria sanguinis hominis perstans.* × 160.

being a species of hæmatozoon totally distinct from both *F. diurna* and *F. nocturna*, although it may be, and is occasionally, associated in the same individual with one (Fig. 78) and, probably, sometimes with both of these.

Geographical Distribution.—*F. perstans* is further characterised by its singularly limited geographical distribution. It appears to be confined to Africa, and, most probably, to the central part of the West Coast and adjoining regions of this continent. Here, in certain districts, it is present in more than half the population. I have examined the blood of 98 negroes from the Lower Congo and from Old Calabar,—kindly sent to me, for the most part, by Dr. Small, Congo, and Mr. Richard Henshaw, Old Calabar,—and

found this parasite in no fewer than 55 instances. Judging from these observations, in some places two-thirds of the people are affected, whilst in neighbouring districts the proportion is very much smaller, so that the distribution, even in the endemic area, is more or less capricious. With the view of ascertaining if it is present in the inhabitants of other parts of Africa or in West India negroes, I have examined, in a manner which to my mind and according to my experience is absolutely conclusive as to the presence or absence of filarial hæmatozoa in the blood-givers, the blood of 74 Zulus, Basutos, Mashonas, Bechuanas, Hottentots, and other South Africans, —most of the slides having been collected for me by Dr. Newton Burns on the African coast,—but in none of these did I find this or any other filarial hæmatozoon. I have also examined the blood of many negroes and Arabs from Zanzibar with a like result as regards *F. perstans;* and in the blood of 23 individuals which Dr. Sharpe kindly sent me from Sierra Leone I was equally unsuccessful. It is possible that the parasite was present in some of these latter, as, owing to faulty preparation of the slides on my part, I do not consider that the examination was quite conclusive; but as regards the natives of South-East Africa, as far north as Zanzibar, I am practically sure that the parasite is not to be found among them. No mention is made by Sonsino or other investigators of such a parasite in the blood of Egyptians or Algerians in their published writings, although Sonsino, in a private communication, tells me he recollects seeing a minute, blunt-tailed, embryo filaria in a Soundanese and also in the blood of certain Egyptians: this observation, however, requires confirmation and reconsideration in the light of our recently acquired knowledge on this subject. As far as I can ascertain, *F. perstans* has not been found in Brazil, India, China, or any other part of the tropical world where the blood has been scrutinised for filariæ by competent observers. Hence I think we may conclude that *F. perstans* is confined to the African continent, and, most probably, to the central part of the West Coast and adjacent hinterland. How far it extends into the interior I cannot say; it is certainly to be found in districts 200 miles up the Congo.

The following table of the results of my examinations of the blood of Africans for this parasite may prove interesting to readers, and a guide to future investigators. With the exception of those relating to Sierra Leone they are practically reliable:[1]—

[1] Since this table was prepared I have had an opportunity, through the good offices of Mr. Beaumont of Upper Norwood, of examining the blood of 51 natives of Dahomey, who were giving a performance at the Crystal Palace, Sydenham. In two I found *F. nocturna*,

	Place.	Number examined.		*Filaria perstans* present in—	
Congo District.	Old Calabar,	31	93	17	53
	Cameroons,	1		1	
	Banza Manteka,	48		34	
	San Salvador,	13		1	
	South-East Africa,	74	127	0	0
	Zanzibar,	18		0	
	West India Negroes,	12		0	
	Sierra Leone,	23		0	

Longevity of the Parasite.—Our present data are not sufficient to determine this, but that it may live for a very long time is certain. I know of one instance in which the parasite was found six and a half years after the negro host had quitted Africa. I have also found it in others who had not been in Africa for over three years. Probably in this respect it is like *F. nocturna*, and may live for an indefinite time, provided it does not meet with an accident.

Life History.—This is still quite unknown. There are certain features, however, about the anatomy and habits of this embryo filaria which, to say the least, are suggestive as to what probably occurs during the earlier period of its existence, and while in the human body. We know for a fact that *F. nocturna* does not leave the blood vessels by its own efforts, but that it is fished out from the circulation by the mosquito; its structure renders such intervention on the part of some blood-sucker, such as this insect, necessary. The structure and habits of *F. perstans* suggest that similar extraneous aid is not required by it, but that, unlike *F. nocturna*, it leaves the vessels by virtue of its own efforts. This is the interpretation I would put upon the retractile beak (Fig. 59, p. 749), the powers of elongation and retraction, the truncated tail, and the absence of a sheath, as already described. All these anatomical and physiological details have certainly reference to the welfare and life history of the parasite. The unmasked retractile beak I

in one *F. diurna*, and in one *F. perstans*. The latter was the interpreter of the troup, and as he had travelled much in Africa, from the complete absence of *F. perstans* in his 50 companions, I suspect he must have acquired his parasite elsewhere than in Dahomey, where this examination of a considerable number of natives would seem to show that *F. perstans* is not indigenous.

regard as a provision for penetrating the walls of the vessels and the tissues; the naked extensile body, endowed with locomotive powers, as fitted for travelling through the tissues; and the blunt tail, as designed for a sort of foot to push against during efforts at progression. The blunt tail of *F. perstans* is like the blunt tail of the embryo of *Trichina spiralis*, whose first step in life is a journey through the tissues of its host. Filaria embryos, which have sharp tails, it is to be presumed do not thus locomote.

Considering these things, I believe that the parent *F. perstans* lives in or in close connection with the circulation; that after its embryos have been launched into the blood stream, they seek to leave the vessels by piercing the walls of the capillaries, and that they then start on a journey through the tissues. As to the next step, I have thought that we may have some clue to this in the unique observations of O'Neil on the parasite of craw-craw, in conjunction with similar observations by Professor Nielly on the analogous disease which he has described under the name "dermatose parasitaire," and in certain cutaneous helminthiases of the lower animals.

It would occupy too much space to discuss this question fully. I would refer the reader to a paper in the *Transactions of the Seventh International Congress of Hygiene and Demography* for a fuller statement. Suffice it to say, that it is possible that the filaria-like parasite found by O'Neil in the pustules of the disease he calls "craw-craw,"—a disease which is endemic, and apparently confined to the region of Africa, in which *F. perstans* is endemic and also confined,—may be an advanced developmental form of *F. perstans*, which, having left the vessels and travelled through the tissues, has taken up its temporary abode under the skin, where it undergoes developmental changes fitting it for further advances towards its destiny. These changes completed, it may be that by the irritation of its presence it brings about the pustulation of the skin, known as craw-craw, a condition which is accompanied by excessive pruritus. Scratching, and liberation thereby of the parasites then ensue; and in this way, I conjecture, the young filariæ get into water, or by some other way obtain access to an intermediary host; for, judging by analogy, the services of such an intermediary are necessary for the maturation of this as of similar parasites. Such a life history would be in harmony with what is known of the life history of *F. nocturna*, *F. medinensis*, *cucullanus*, etc. In the limited geographical distribution of this hypothetical intermediary host we must seek for the explanation of the limited geographical distribution of this filaria, and of those pathological

conditions which, in the future, may be found to be associated with it.

This sketch of a possible life history of *F. perstans* is in great measure purely speculative. I do not commit myself to it in any way; but I would submit it as a possible explanation of the facts of the case, and as a working hypothesis by which to direct and stimulate further investigation.

Pathological Relations.—In the article on Negro Lethargy, I have discussed briefly the possible relationship of this parasite to the sleeping sickness of the West Coast of Africa, and to what I have there said I would refer the reader. I have seen no reason to associate *F. perstans* with any other pathological condition, and there is abundant evidence to show that its presence in the human body is, in the vast majority of instances, quite compatible with perfect health.

Treatment.—It is not likely that there are any means of killing this parasite other than such as would at the same time kill the host. But even if it were possible to influence the filaria in any way, it is very questionable if this influence would be one in the right direction, and in the interests of the host; for an irritated, sickly, dying, or dead parasite in the blood vessels or lymphatics is to my way of thinking an infinitely more dangerous guest than one which is healthy and performing its various functions in a normal manner. Of course it is very undesirable to have such a guest; but I maintain that, as in the case of *F. nocturna*, once such guests are in the house, it is too late to try to turn them out; that it is a wise thing to take care of them, not to irritate them, but to put up with them, and make the best of an unfortunate circumstance which, fortunately, in nine cases out of ten, leads to no bad result. The only opportunity for medical science, as regards this and similar parasites, is in the direction of a wise prophylaxis. To direct this properly and with precision, a more thorough knowledge of the life history of the parasite is desirable; but, pending the acquisition of this fuller knowledge, as we may be fairly sure that *F. perstans* passes one stage of its existence in water, and that it is probably through the agency of drinking water that it obtains access to the tissues of its ultimate human host, we are justified in saying that a pure and properly conserved water supply is all that is necessary to secure immunity from *F. perstans* and any diseases it may produce.

The Demonstration of the Filariæ Sanguinis.

The Determination of the Presence or Absence of Filaria Embryos in the Blood.—It is owing in great measure to ignorance of suitable and convenient methods of preparing the blood for examination for the filariæ, and the vast amount of trouble entailed by any examination—deserving to be called reliable—made in the usual way, that progress in our knowledge of these parasites has been so slow, and that their presence has been so frequently overlooked or denied in cases in which in all probability they would have been found if the examination had been properly conducted. Apart from any scientific interest which may attach to the matter, it is often of the first importance in clearing up diagnosis in certain tropical affections, that the presence or absence of filariæ in the blood should be determined; just as important as the determination of the presence or absence in the sputum of the bacillus tuberculosis in lung disease, or of casts and albumen in the urine in kidney disease. Experience has shown, however, that unless methods of clinical investigation are simple, and can be easily and rapidly applied, they are neglected by the majority of practitioners. It is a very tedious business the examination of the blood for filariæ in the usual way; six or more slides may have to be searched through, bit by bit, before certainty, in a negative sense, can be said to be attained. This means a great expenditure of time. Moreover, owing to the periodicity observed by two of the filariæ, which is not regulated in accordance with the convenience of the physician, it may so happen that, though at other times present in great profusion, at the time of the physician's visit the parasite is normally absent from the general circulation. It is therefore a matter of importance that the method adopted for demonstrating presence or absence of filaria embryos should not only be one which is simple, easily, and rapidly carried out, but that it should be one which can be applied to blood which has been drawn at any time of the day or night. Of the many different methods I have tried, I shall confine myself to describing the one I have found the most convenient.

In examining blood for filariæ, "filarial periodicity" must never be ignored. Examinations made regardless of this phenomenon are valueless. Millions of filariæ may be present in the general circulation at midnight, and yet not one can be found at mid-day, and *vice versâ*, according to the parasite. In examining for *F. nocturna*, the blood must be drawn during the night; any time between 8 P.M. and 7 A.M. will do, but the nearer to midnight the

better. If *F. diurna* is sought for, the blood must be drawn between 10 A.M. and 6 or 7 P.M., but best about 1 P.M. *F. perstans* may be sought for at any time in the twenty-four hours.

A supply of blood is most conveniently obtained from the pad of a finger. If the finger be lightly ligatured, so as to compress the veins only but not the arteries, on pricking it with a sharp needle a drop of blood speedily wells up. The whole of this should be transferred to an ordinary microscope slip before coagulation has had time to occur. The blood is then spread out with the needle, in a fairly uniform layer, over an area of about an inch square. The slide is then laid flat—blood surface upwards, covered to keep away dust, and allowed to dry. When dry it may be labelled and stored away to wait the convenience of the practitioner; or, if time permit, the examination may proceed at once. The slide does not spoil by keeping, provided it is not allowed to become damp; I have examined many such slides which had been prepared for months, and others which had come from the tropics and were covered with cryptogamic growths; but even in these have had no difficulty in finding the filariæ in them. To demonstrate the parasites in slides so prepared, all that is necessary is to immerse the slides in a very weak solution of fuchsin—one drop of the saturated alcoholic solution to an ounce of water. The slide ought to remain in the stain from one to two hours. If on removal from this the blood seem to be deeply stained, it should be washed for a few seconds in weak solution of acetic acid—three or four drops to the ounce of water. If the blood be not seen to be deeply stained, it is not necessary to use the acetic acid; but old slides, especially such as have been prepared in the tropics, usually need to be washed in the acid solution. After washing off the acid, a cover glass may or may not be applied to the wet slide, and the preparation be at once placed under the microscope. Search is then made with a low power—an inch or two-thirds of an inch objective is best. A high power must not be employed for searching; it includes too small a part of the slide at a time to permit of rapid and exhaustive examination. In slides so prepared, all colour is found to have been discharged from the red corpuscles; a few of the white corpuscles and any filariæ which may be present are the only objects which are deeply coloured. The filariæ stain very deeply, and at once catch the eye. The preparations from which the photomicrographs (Figs. 77 and 78) were made were prepared in this way. If there be any doubt about the nature of any coloured object encountered, it should be centred and examined with a quarter or eighth of an inch objective; its real nature will then be at once apparent. A

double nose piece is therefore of great service; and by using a mechanical stage the observer can work accurately, and be sure that no part of the slide escapes examination.

Prepared in this way, *F. nocturna* usually preserves its graceful curves; but, strange to say, in the case in which I more particularly studied *F. diurna*, the parasites were generally considerably distorted. The specimens of the latter were shrunk by a third or a half of their normal length, and correspondingly thickened. Their tails were often doubled under their bodies, and the graceful curves in which they are disposed during life gave place to awkward,

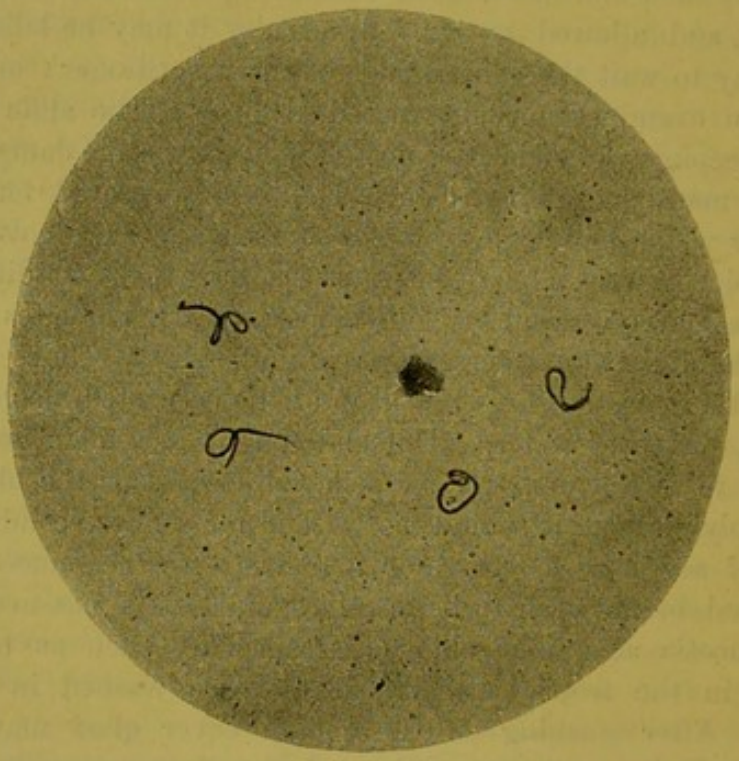


FIG. 77.—*Filaria nocturna.* × 60.

ungraceful curves and kinks. In neither parasite does the sheath take the stain, and consequently is hardly visible; nevertheless the filariæ are readily detected if present in such preparations, and, with a little experience, *F. perstans* can be easily diagnosed from *F. diurna* or *F. nocturna*.

Unfortunately in such preparations the fuchsin stain fades very quickly—in a very few days. If more permanent preparations are desired, weak picrocarmine, or, better still, ½ per cent. eosin solution may be used. They are to be employed in exactly the same way as the fuchsin stain. They are not suitable for old slides, however, as in such they stain corpuscles and liquor sanguinis as well as parasites, and are not discharged by washing in weak acetic acid.

Slides of blood prepared in this way, and stained by eosin, are best mounted in glycerine jelly or Farrant's solution.

A very few minutes are sufficient to thoroughly search such a slide. If no filariæ be found, seeing that the slide contains a large quantity of blood, it may be confidently asserted that there are no filariæ in the circulation.

I would strongly recommend that an examination of this sort be made in all cases of obscure tropical disease, and that it be systematically practised on all patients admitted into hospitals in

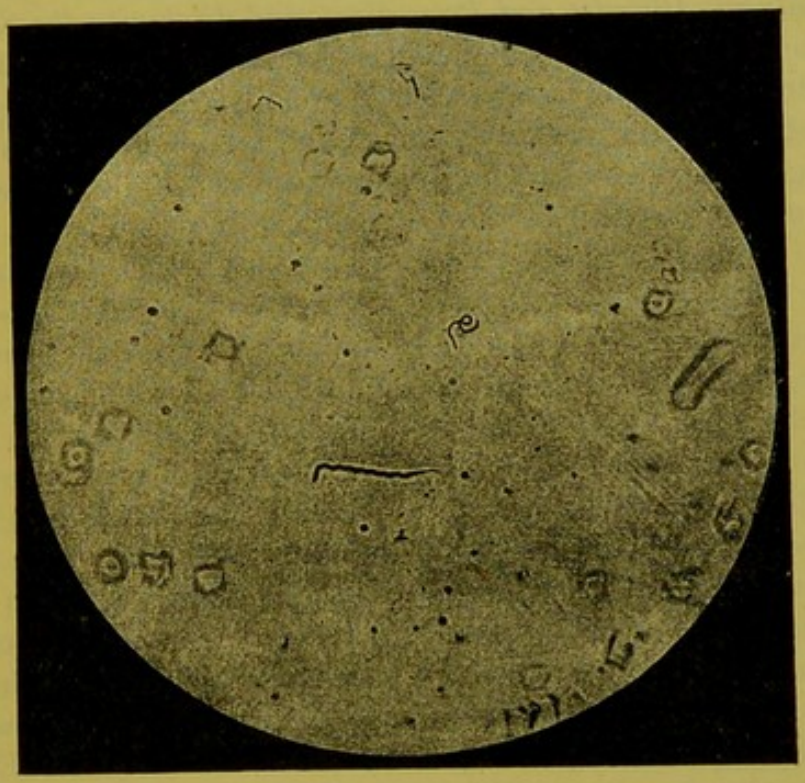


FIG. 78.—*Filaria diurna* and *Filaria perstans*. × 60. In the patient from whose blood the specimen represented was prepared, both parasites were present.

tropical and subtropical countries; and, further, that medical men and naturalists travelling in new countries should make collections of slides of blood so prepared from the natives. It entails very little trouble, and often throws unexpected light on cases otherwise obscure; and, if practised systematically, would, in time, yield a large body of valuable information. A native assistant, or dresser, or the patient himself, can easily be taught how to spread the blood on the slide, and, if necessary, stain and examine it. Fig. 78 is a photomicrograph from a slide collected by a negro in Africa, and subsequently stained in London.

To demonstrate the Sheath of F. nocturna and F. diurna.—Mix a droplet of blood with some solution lower in specific gravity than the blood. Urine is recommended by Mackenzie; it is always at hand, and answers very well. This produces an endosmosis into the sheath, which then becomes distended, separated from the body of the contained embryo, and very manifest.

Permanent preparations, in which the sheath is beautifully displayed, can be obtained by following the directions given in the next paragraph.

To make permanent Preparations of the Filariæ, showing them in their natural Attitudes and Proportions, and displaying the Sheath.—Into each of half a dozen watch-glasses or small glass capsules pour a few drops of strong acetic acid. Carefully clean as many microscope slips. After cleansing the tip of the finger, ligaturing and pricking it in the way already described, touch lightly with the centre of a glass slip the drop of exuding blood, so as to transfer only a minute quantity to the glass; over this lay, crosswise, another glass slip, and, as soon as the blood has spread out in a very thin layer between the slips, rapidly pull or glide them apart. A very thin film of blood is thus obtained. Immediately, and before they dry, invert the slips over glasses containing the acid, so that the fumes of the acid, but not the fluid itself, may impinge on the drying blood. At least six slides should be so treated, as, in consequence of the small quantity of blood on each slide, there is some chance that some of the slides contain no filariæ. As soon as the blood has dried, pour a few drops of a ½ per cent. solution of eosin on the slips, and allow it to remain in contact with the blood for two minutes. Then wash the slide in water, dry slowly, and mount in the usual way in balsam. In slides so prepared (Figs. 51, 52, and 53), it will be found on examination that the red blood corpuscles and the liquor sanguinis are stained—the former deeply, the latter lightly—by the eosin, the filariæ and a few of the white corpuscles remaining unstained, and the former showing up quite white with a beautiful pearly lustre. In places where the layer of blood is very thin, the dimensions and beautiful graceful curves of the parasite are perfectly preserved, and have a very natural appearance. Where the layer of blood is rather thicker, the body of the filariæ is seen to have shrunk somewhat, but the sheath retains its normal dimensions, and thus shows up very distinctly. The fumes of the acetic acid have the singular property of inverting the ordinary action of the eosin on the corpuscles and filariæ respectively. In these, as in all preparations of filarial blood, the filariæ must be sought for, in the first instance, with a very low power;

when one is found it can be centred and examined with any power to suit the purpose of the observer.

Permanent preparations, in which the filariæ are stained, are easily and satisfactorily made from thin cover-glass blood films. Fix the films by passing through the flame, stain by floating in eosin, wash, dry, and mount in balsam in the usual way.

CHAPTER XXII.

DISTOMUM RINGERI VEL PULMONALE.

BY PATRICK MANSON, M.D., M.R.C.P., LL.D.

History.—Helminthologists have long been aware that the lungs of many of the lower animals are liable to be infested by various species of trematodes, but it is only comparatively recently that it has been shown that man himself is liable to a similar infliction. Working independently, both Professor Baelz[1] and the writer, the former for Japan and the latter for Formosa, described in 1880 a peculiar form of hæmoptysis, affecting a considerable proportion of the inhabitants of certain districts of these countries, which appeared to have for its cause a parasite lodged in the lungs. The diagnosis of pulmonary helminthiasis was arrived at from the discovery of the ova of an unknown parasite in the sputum characteristic of the class of cases referred to, and not long afterwards this diagnosis was confirmed by the discovery by Dr. Ringer, of Tamsui, Formosa, of the parasite itself. This proved to be a species of distome, which was named by Cobbold, *Distomum Ringeri*, and, subsequently, by Baelz, *D. pulmonale*. Since that time our knowledge of this parasite and of the diseases it gives rise to has been considerably extended, although there are still many important gaps to be filled in both as regards its geographical distribution, its life history, and its pathological relations.

Leuckart—who has given a very elaborate and the most accurate description of the anatomy of *D. Ringeri*—points out that it is probably identical with a distome found by Kerbert in 1878 in the lungs of a tiger, and named by him *D. Westermanni*. Raillet[2]

[1] Baelz, *Cent. f. d. ges. Med. Wissen.*, No. 39, 1880. *Deutsche Ges. für Natur und Völk. Ostasien*, Tokio, Oct. 1882. *Berl. klin. Woch.* 1883, p. 234. *Lancet*, 1880, vol. ii. p. 548. Remy, *Arch. gén. de méd.* 1883, p. 525. Manson, *Chinese Cust. Gaz., Med. Rep.* No. 20, 1880; No. 22, 1881. *Lancet*, vol. i. 1883, p. 532. *Filaria Sang. Hom.* 1883, p. 134. *Trans. Hongkong Med. Soc.* 1889. Wallace Taylor, *Chinese Cust. Gaz., Med. Rep.* No. 27, 1883-84. Leuckart, *Parasiten des Menschen*.

[2] *Trans. Seventh Internat. Cong. of Hyg. and Dem.* vol. iii. p. 75.

mentions that the dog also, at all events in Japan, is occasionally its host. These are important facts from a practical as well as from a scientific point of view; for, if the lower animals are subject to this form of helminthiasis, any prophylactic measures which in the future may be devised as against this parasite must be planned in accordance.

Geographical Distribution.—*D. Ringeri* has been found in Japan, in Formosa, and in Corea, but we do not as yet know of its occurrence in any other country. In some localities in the countries mentioned it is exceedingly common. For example, in the mountain village of Tama, Okayama Ken, Japan, situated several thousand feet above the level of the sea, and with a long and very severe winter, according to Yamagiwa and Inouya,[1] nearly every one is affected with hæmoptysis from pulmonary distomiasis; and in some parts of North Formosa, I have been assured that from 10 to 30 per cent. of the inhabitants are similarly afflicted. As the climate of North Formosa is almost tropical, and that of some of the mountain districts in Japan in which this distome is endemic is almost arctic, it is evident that the geographical range of the parasite may be very extensive and, in great measure, independent of climatic conditions. A curious fact about its geographical distribution is, that even in the affected countries it occurs only in particular and often very limited districts, resembling in this respect the ordinary liver fluke, *D. hepaticum*. For example, although very common in North Formosa it is unknown in South Formosa, and it is also unknown as an endemic affection on the neighbouring Chinese mainland, scarcely a hundred miles to the westward, and in constant communication with the affected districts. It is safe to conclude that this apparent caprice of distribution depends, as in the case of the liver fluke, on the distribution of the animal or animals which play the part of intermediary host to the parasite, rather than to any peculiarity of climate, or of the habits of the people. The peculiar and capricious distribution of this unknown intermediary host is, doubtless, in its turn dependent on certain local peculiarities in the chemical and hydraulic conditions of the soil. It has occurred to me that as Japan, Formosa, and Corea, or those parts of them where the parasite is found, are highly volcanic in their geological structure, and the neighbouring mainland of China, where the parasite is not found, is not of a volcanic nature, that, possibly, in this circumstance lies the explanation of the caprice of distribution; the unknown intermediary host of *D. Ringeri*—most probably a mollusc—requiring a volcanic soil, among

[1] *Sei-i-kwai*, 1891, p. 28.

other conditions, for its wellbeing. Relying on this hypothesis, I venture to predict that, as observations extend, the parasite and the peculiar hæmoptysis it gives rise to will, in the future, be found in many of the volcanic islands lying off the east and south-east coasts of Asia, and, possibly, also in the volcanic districts of America and Europe.

Description of the Parasite.—*D. Ringeri* usually inhabits the lungs, although it has also been found in the sub-peritoneal fascia and in the brain. When turned out of the burrows or tunnels it occupies it exhibits leech-like movements, and is seen to be a small, very thick, reddish-brown, oval fluke, measuring about $\frac{1}{3}$ inch in length by $\frac{3}{16}$ inch in breadth (Fig. 79, *m*). It possesses two suckers, the oral (Fig. 79, *c*), which is terminal, being slightly the larger (Wallace Taylor), smaller (Leuckart); the ventral (Fig. 79, *b*) is placed about one-third of the entire length of the animal posterior to the oral. The œsophagus (Fig. 79, *d*) is very short, and bifurcates immediately behind the oral sucker into two simple tubes (Fig. 79, *f*), which, running along the borders of the body of the parasite, terminate in blind extremities close to the caudal end. The genital pore opens close to and behind the ventral sucker.

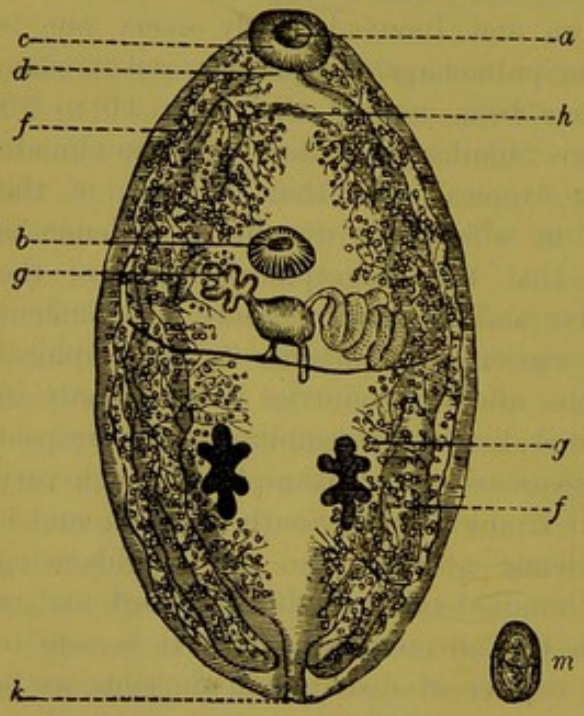


FIG. 79.—*Distomum pulmonale.*

The Ova, which can be readily found in the contents of the burrows or in the sputum brought up from the lungs after coughing, are dark-brown oval bodies, measuring about $\frac{1}{300}$ inch by $\frac{1}{500}$ inch. The shell is devoid of markings or spines, and is moderately thick, one end—the broader—being closed in by a circular operculum. When newly expectorated, and for some time afterwards, no embryo can be detected. The entire interior of the egg is occupied by an irregular, ill-defined mass, which, on pressure being applied to the cover-glass, is readily broken up and escapes through a rent or fracture of the shell as a crowd of variously sized, globular, oil-like

bodies, the more minute of which indulge for a time in active molecular movements.

The Embryo.—If some of the characteristic sputum from a case of endemic hæmoptysis be shaken up with water and allowed to stand so that the ova it contains may settle to the bottom of the vessel, and if after a day or two the supernatant fluid be removed and fresh water added, and this process of washing the ova be repeated several times, it will be found that after one or two months—longer or shorter according to temperature—a ciliated embryo is developed in the interior of each ovum. As the embryo approaches maturity it becomes very restless, and is seen to be constantly endeavouring to obtain its liberty by butting against the operculum closing in the end of the shell. By a little pressure, carefully applied to the cover-glass, the operculum may be forced back, when the embryo immediately escapes into the surrounding water, its covering of cilia starting at once into increased activity. In the water the little animal swims about very briskly, rotating on its axis, gyrating about, or rushing across the field and changing its shape in harmony with the character of its movements. It is evidently quite at home in the new medium into which it has been so suddenly launched. From this circumstance, and also from the fact that development does not proceed unless the sputum is freed from mucus by washing in water, we may safely conclude that water is the proper habitat for the time being of the little animal. These experiments, which I have made many times, and always with the same result, have since been confirmed by Nakahama.

As regards the future progress of development, we have no positive information. Analogy, however, leads us to believe that in the water the embryo distome seeks out an intermediary host in the shape of some mollusc or other fresh water animal; and that after entering this, either by being swallowed or by penetrating its integument, it undergoes the complex metamorphosis peculiar to the distomes. When this is completed it is either swallowed by man while still in its intermediary host; or, escaping from this, it attaches and encysts itself on some vegetable or other matter, or enters another animal, and there awaits the chance of being transferred to a human stomach, from whence it afterwards works its way to the lungs of its definitive host.

Prophylaxis.—Although our knowledge of the life history of *D. Ringeri* is still very defective, enough is known to indicate the direction which an intelligent prophylaxis should take. This, obviously, should consist in a pure water supply, in filtering and boiling this where the source is at all suspicious, in avoiding

uncooked fresh water animals and uncooked vegetables as food, and in preventing the subjects of endemic hæmoptysis, whether men or animals, from expectorating near or otherwise contaminating the sources of water supply.

(*a*) Endemic Hæmoptysis or Pulmonary Distomiasis.

As will be gathered from the foregoing, this disease is by no means a rare occurrence in the endemic area of *D. Ringeri*, and it may be one of considerable gravity.

Symptoms.—It is found at all ages, in both sexes, and is peculiar to no occupation. An individual affected with endemic hæmoptysis has a chronic cough of varying degrees of severity and frequency. Each attack of coughing results in the expectoration of a considerable pellet of thick, glutinous, rusty-brown mucus of the colour and consistence of the rusty sputum of acute pneumonia. Such sputum can usually be produced by the patient at will, but generally in greatest abundance on waking in the morning. The cough and sputum are permanent symptoms; in addition to these, at irregular intervals of weeks or months, the patient may be attacked with an ordinary hæmoptysis of pure, bright red blood, in quantity varying from a drachm or two to many ounces. As a rule, this active hæmoptysis is neither profuse nor of long continuance, but it may happen that it is so profuse and so long continued that life is endangered. Frequently the ordinary rusty sputum contains minute specks and pellets of pure blood.

Physical examination in the earlier stages and in the milder cases of the disease can detect no sign of pulmonary mischief; later, signs of consolidation with absence of respiratory murmur in patches may develop.

Prognosis.—Usually, endemic hæmoptysis is unattended with risk to life; but as violent hæmorrhage from the lungs is always a possibility, and as it may happen that frequent and prolonged bleedings, distressing cough, and habitual dyspnœa gradually, sooner or later, induce a cachectic condition which may end in death, it must be looked on as by no means a disease to be made light of. It is also more than probable that the peculiar condition of lung, to be presently described, may tend to induce bronchitic, pneumonic, and tubercular states, and thereby indirectly shorten life. The disease may continue for many years, probably for life.

Diagnosis.—When the presence of pulmonary distomiasis is suspected a diagnosis is easily arrived at by a microscopical examination of the sputum. On placing a little of this under the

microscope,—not the bright red blood brought up during one of the intercurrent attacks of active hæmoptysis, but the rusty, sticky material already described,—besides epithelium, Charcot's crystals, mucus and blood corpuscles, it is seen to contain a number of comparatively large, oval, operculated, egg-shaped bodies of a dark-brown colour—the ova referred to above. These are absolutely diagnostic, and are readily discovered and recognised if present. The examination ought, in the first instance, to be made with a low power—a 1 inch or ½ inch objective is the best to employ.

Pathological Anatomy.—A few cases of endemic hæmoptysis have come to the post-mortem table in Japan. Externally the lungs may look quite healthy; but on cutting into them several or many specimens of the parasite already described may be found, either free in the bronchi, or, more usually, occupying what have been termed "burrows." These burrows, for the most part about the size of a filbert, communicate with the bronchi by one or more small openings. In some instances they seem to spring directly from the dilated end of a bronchus, or from a lateral dilatation of one of these tubes, these appearances probably being brought about by the septa separating the burrows from the bronchi breaking down, and burrow and bronchus thus becoming one cavity.

The walls of the burrows or cavities are very irregular in thickness and shape, and often extend, as a sort of infiltration surrounded by a congested area, for some distance into the parenchyma of the lung. Neighbouring burrows communicate by tunnels, and as it sometimes happens that adjacent burrows open into each other by the breaking down of the intervening septa, a cavity of some size may result. All the burrows and cavities contain a viscid, reddish secretion, corresponding in appearance and constitution to the sputum already described; and, in addition, one, several, or many specimens of *D. Ringeri*. As many as twenty of these distomes have been found in one body.

Treatment.—I have tried many kinds of parasiticide inhalations in these cases, but I have never succeeded, so far as I know, in procuring the death or expulsion of a distome, although in one case I believe the inhalations did some good. Wallace Taylor mentions a case in which what appeared to be a distome was spontaneously expectorated. It is to be feared, however, that this is quite an exceptional occurrence, and that the only thing which can be done for the benefit of cases of endemic hæmoptysis is to attend to the general health, and by rest and the usual treatment for hæmoptysis to mitigate the severity of this when it occurs.

(*b*) Cerebral Distomiasis.

Although the lungs are the usual seat of *D. Ringeri*, and one well adapted to its habits, inasmuch as the air passages afford a ready route by which the ova may leave the body of the human host, recent investigations in Japan prove that other organs may sometimes be affected by this parasite.

Yamagiwa [1] relates a case in which a form of Jacksonian epilepsy resulted from the lodgment of distomes or of their ova in the cortex of the right cerebral hemisphere. The patient, 29 years of age, and without syphilitic history, suffered from March to December 1887 from a convulsive affection of the left side of the body. The attacks would commence in the arm and then extend to the side and leg. At first they occurred two or three times a day, each attack being followed by a period of unconsciousness. At the end of 1887 the epileptiform seizures ceased, but it was then remarked that the field of vision of the left eye was surrounded by a circle of colour, and that walking was rendered difficult by giddiness. Two months later the convulsive attacks recurred, and were now associated with left-sided paresis and paræsthesiæ (face and arm), and there were also violent right-sided headache, together with irritability of temper and weakness of memory. A short and temporary improvement was followed by a final relapse in December 1888, when the patient was admitted to hospital. The paretic condition of the left arm and leg was then very marked, and there were noted also exaggerated left knee-jerk and, among the subjective symptoms, dimness of sight and the coloured areola already referred to, although the ophthalmoscope revealed nothing amiss with the fundus oculi. About 22nd March he passed into the *status epilepticus*, and died on 2nd April 1889, about two years from the onset of the first cerebral symptoms. At the post-mortem examination a distomum tumour containing many ova was found in the left lung; the parent flukes were not discovered. With one exception the other organs were healthy. In the right cerebral hemisphere, more especially over the lateral and posterior regions and about the central convolutions, the pia mater was adherent to the cortex, which was found to be very firm, and in places distinctly sclerosed. On cutting into the grey matter, here and there groups of dark grey dots, each dot being surrounded by a white and sharply defined circle, were displayed. On microscopical examination it was seen that these grey dots with white surroundings were produced by embolic occlusion of vessels, from clusters of minute, brown, oval bodies, which were readily recognised to be

[1] *Virchow's Archiv*, 1890, Band cxix. p. 447.

distomum ova. Some of the ova preserved their normal contour and brown appearance; others, again, were reduced to collapsed and crumpled up shells. Around the clusters of ova the walls of the vessels were thickened, and outside of these again was a zone of hypertrophied connective tissue with round cell infiltration.

In commenting on this remarkable case Yamagiwa suggests that possibly, as no mature distome was discovered in the brain, the ova he found there had been carried from the lungs by the blood. But had this been the case, I might suggest that the ova would have been more generally diffused throughout the entire encephalon, occurring singly and not, as was the case, in groups. I am inclined to think that in this patient the parent worm lay in the brain, or in communication with one of the larger arteries of the brain,—the middle cerebral, for example,—into which it discharged its ova as, under normal circumstances, it discharges them into a bronchus.

In his very valuable and suggestive paper Yamagiwa refers to another and somewhat similar case of distomum infection observed by his friend Otani. The patient was 26 years of age when, in the spring of 1886, he began to expectorate characteristic distomum sputum. In May of the following year, 1887, he had his first epileptic seizure. Fits recurred about once a month, and on 9th September he was admitted into hospital very ill, apparently as a consequence of a succession of epileptic fits. On 12th September he had ten epileptiform seizures, followed by a prolonged state of unconsciousness, from which he gradually recovered by 19th September. On 25th September he again became insensible, and on the following day he died. At the post-mortem examination two tumours were discovered in the right cerebral hemisphere. One, about the size of a hen's egg, involving the fore part of the first and second frontal convolutions, was visible on throwing back the dura mater; the other, consisting of two larger cysts about the size of a pigeon's egg, and a number of smaller intercommunicating cysts, was only revealed on section of the occipital lobe. The tumours were made up of connective tissue, in which lay a number of cysts varying in size from a grain of rice to a pigeon's egg. The cysts communicated with each other and contained a dark brown, viscid fluid laden with distomum ova. A mature distome was found in one of the occipital cysts, and a second was discovered close by, but apparently outside the tumour, and in healthy brain tissue. Distomum tumours were also found in the lungs and sub-peritoneal tissue.

Some years ago I was consulted by a Corean, 39 years of age, about certain œdematous swellings which for some days had been

coming and going on the left side of his face, notably around the eyelids, which were puffy, on the malar region, which over an area the size of a half-crown was also puffy and swollen, and behind the left ear. There was no redness, tenderness, nor pain in any of the swellings; they disappeared in a day or two. About two months previously this man began to expectorate characteristic distomum sputum, and he was expectorating it freely when I saw him. Unable to find any ordinary explanation for the singular facial swellings, it occurred to me at the time that they might in some way be of verminous origin, and possibly produced by wandering distomes or their ova. Since reading Yamagiwa's paper I am still more inclined to this opinion.

It is evident that *D. Ringeri* is at times a highly dangerous parasite, not only on account of the damage it may produce in the lungs in the course of the normal evolution of its life history, but also on account of cerebral and, possibly, other forms of disease, not yet recognised as depending on it, produced in the course of the abnormal blind wanderings which, judging from the cases just cited, it appears at times to indulge in. Henceforth, in the endemic area of this helminthiasis, the possibility of such a cause for the symptoms in obscure cerebral cases must be considered in attempting an exact diagnosis; and the concurrence of a history of hæmoptysis and distomum sputum with Jacksonian epilepsy or other form of one-sided cerebral disease, must be regarded as significant of the possibility of this grave accident.

Treatment.—In cases of cerebral distomiasis, such as the two just related, in which life is threatened, and in which the position of the parasite in the brain can be fairly well localised from a study of the symptoms, trephining and excision of the implicated area would be a justifiable proceeding, and one which might be undertaken with some hope of permanent benefit to the patient, otherwise doomed, if not to certain death, at least to a chronic, painful, and disabling affliction.

CHAPTER XXIII.

INTESTINAL, HEPATIC AND PORTAL ENTOZOA, AND ASSOCIATED DISEASES.

BY PROSPERO SONSINO, M.D., PISA.

Intestinal Zooparasites, particularly Anchylostomiasis and Rhabdonemiasis.

Intestinal Entozoa, their importance in warm countries, and their diagnosis.—As an introduction to this chapter we think it desirable to present a synopsis of those entozoa which up to the present time have been met with only in warm climates, the more so as little attention has been bestowed upon them, as a rule, in treatises on general pathology. They constitute, however, almost two-fifths of the total number of those met with in man, and among them there figure some which are of great importance on account of their pathogenetic capacity, and their injurious effects upon the host. It will be seen from this synopsis that only a relatively small number of these parasites belong to the intestinal class with which we are immediately concerned in this chapter.

Indeed, the greater number of intestinal worms are cosmopolitan, or almost cosmopolitan, and are found more or less frequently in different countries, without any strict relation to climate or race, their prevalence being rather in relation to the food and sanitary habits of the different races. This is the case in respect of *Ascaris lumbricoides*, *Oxyuris vermicularis*, *Trichocephalus dispar*, among the nematodes, and *Tænia solium*, *T. mediocanellata*, *T. nana*, *T. elliptica*, *T. leptocephala*,[1] and *Bothriocephalus latus* among the cestodes. These parasites are no more peculiar to warm than to temperate climates. Perhaps even some intestinal entozoa, which up to the present have

[1] *T. flavo-punctata*, found in man in America by Weinland, and also in Italy by Parona, is now generally believed to be nothing else than *T. leptocephala*, or *T. diminuta*, found ordinarily in the intestine of rats and mice. For full particulars about this cestode, and about the akin species, *T. nana*, see Blanchard, *Histoire zoologique et médicale des Téniadés du genre Hymenolepis, Weinland.* Paris, 1891.

SYNOPSIS OF THE ENTOZOA OF MAN, EXCLUSIVELY PROPER TO WARM COUNTRIES, WITH THEIR GEOGRAPHICAL DISTRIBUTION.

NAME OF THE ENTOZOON.	ASIA.	OCEANIA.	AFRICA.	AMERICA.	OBSERVATIONS.
(1) TÆNIA MADAGASCARIENSIS, *Davaine* (*)	Bankok (*Leuckart*)	..	Mayotta (*Grénet*).—Mauritius (*Chevreau*)	..	..
(2) BOTHRIOCEPHALUS MANSONI, *Cobbold* (*?)	China (*Manson*). — Japan (*Scheube, Ijima*)	..	..	..	Known only in the larval stage. Probably the same species of larval form found in *Canis aureus* (Jackal) in Egypt by Sonsino.
(3) DISTOMUM RINGERI, *Cobbold* (**)	Formosa (*Manson, Ringer*). —Japan (*Baelz, Taylor, Ijima*). — Corea (*Baelz, Manson*)	..	..	..	Found by Yamagiva, encysted in brain, having caused Jacksonian epilepsy. According to Leuckart, *D. Ringeri* is identical with *D. Westermanni* found in Tigre.
(4) DISTOMUM HETEROPHYES, *von Siebold* (*)	..	..	Egypt (*Bilharz*)	..	..
(5) DISTOMUM BUSKI, *Lankester*, or D. CRASSUM, *Cobbold* (*)	China (*Busk* in a Lascar, who died at Greenwich; *Kerr* at Canton; *Cobbold* in persons from Ningpo). — Straits Settlements (*Sinclair*).—Assam (*Giles*). —Borneo (*Walker*)	..	..	..	..
(6) DISTOMUM SINENSE, *Cobbold* (**)	China and Corea (*MacConnell* at Calcutta, in Chinese — *Macgregor* at Mauritius, in Chinese). — Japan (*Baelz, Scheube*).—Tonkin (*Grall, Vallot*).—Bengal? (*Pfihl*)	..	..	..	Found, too, in cats in China—identified by Sonsino.
(7) DISTOMUM CONJUNCTUM, *Cobbold* (**)	India (*MacConnell*)	..	..	..	Found by Cobbold in an American fox which died in London, and by Lewis in dogs at Calcutta.
(8) BILHARZIA HÆMATOBIA, *Cobbold* (*?)	Arabian Coast of Red Sea (?)	..	Egypt (*Bilharz* and others).—Cape Colony (*John Harley*).—Kaffraria and Natal (*Cobbold*).—Nearly all the east coast of Africa, with the adjacent isles. —On the Gold Coast in Western Africa (*Eyles* and *Eden*).—White Nile between 6° N., and the Albert Nyanza (*Felkin*). —Chad and adjacent countries? (*Nachtigal*). —South Tunisia (*Villeneuve, Brault, Cahier*).	..	Nachtigal's verbal communication only attested the existence of endemic hæmaturia at Lake Chad and adjacent countries; the existence of *Bilharzia* in those districts has not been actually demonstrated. Berkeley Hill, in 1888, spoke of *Bilharzia* in two persons living wholly in England. *Bilharzia* was found by Cobbold in *Cercopithecus fuliginosus*, which died in London. *Bilharzia crassa* found by Sonsino at Zagazig (Egypt) both in oxen and in sheep; but it is still doubtful whether it is a different species from that of man. *Bilharzia* in oxen was found, too, at Calcutta by Bomford, and in sheep, near Cattania (Sicily), by Grassi.

(9) AMPHISTOMUM HOMINIS, *Lewis and MacConnell* (*)	India (*Lewis and MacConnell*).—Assam (*Giles*)	..	..	..	..
(10) DRACUNCULUS MEDINENSIS, *Lin.* (**)	Arabia, Persia, Turkestan, India	..	Guinea, Senegambia, Darfoor, Senaar, Abyssinia, Nubia, Egypt	West Indies, Brazil, Guiana	..
(11) FILARIA SANGUINIS HOMINIS, *Lewis* (*)	India (*Lewis*).—China (*Manson*). — Japan (*Scheube, Beukema, Baelz*)	Queensland (*Bancroft* of Brisbane)	Egypt (*Sonsino*, 1874)—Soudan — Algeria? (*Cauvet*, 1876).—Shores of the Zambesi and Lake Nyassa, Zanzibar coast (*Felkin*).—Mauritius, Mayotta, West Africa (*Manson*)	West Indies (*Demarquay, Creveaux*, and others). Brazil (*Wucherer, Silva Araujo, Magalhães*, and others).—Southern portion of the United States, Charleston, South Carolina (*Guitéras*).—Mobile, Alabama (*William M. Mastin*). — Guiana (*Winckel*).—Buenos Ayres (*Wernicke*)	The first description of the embryos of this filaria we owe to Demarquay, who found it in a chylous hydrocele of a man from Havana, 1863. Lewis discovered it at Calcutta in 1872 *in blood*. Adult worm found almost at the same time by Bancroft, Carter, and Lewis.
(12) FILARIA DIURNA, *Manson* (*)	..	..	Congo, Old Calabar (*Manson*)	..	The zoological specificity of this and of the following filaria rests on the recent observations of Manson, and it is founded only on the characters of the embryos, the only stage known at present.
(13) FILARIA PERSTANS, *Manson* (*)	..	..	Congo, Old Calabar, (*Manson*)	..	Possibly the cause of sleeping sickness and perhaps of craw-craw (*Manson*).
(14) FILARIA LOA, *Guyot* (*)	..	..	West Coast of Africa, Guinea, Angola, Gaboon, Congo	Guiana and West Indies, only in African negroes	Supposed by Manson to be the adult stage of *Filaria diurna*.
(15) FILARIA VOLVULUS, *Leuckart* (*)	..	..	Gold Coast	..	Found in verminous tumours in the integuments of negroes.
(16) PENTASTOMUM CONSTRICTUM, *von Siebold* (**)	..	.	Egypt (*Pruner, Bilharz, Fenger*). — Bathurst, Gambia, and other places of Western Africa? (*Crawford, Kearney*)	..	Known only in the larval stage. Found in Cairo only in necropsies of negroes, and probably indigenous to the Soudan and not to Egypt; also found in the giraffe (*Pruner*).

(*) One asterisk indicates that the entozoon was found in man only.

(**) Two asterisks indicate that the entozoon was found in man and animals.

(*?) One asterisk and interrogative point indicate that it is doubtful whether it was found only in man, or in both man and animals.

been found only in certain warm countries, and which we will mention hereafter, owe their restricted distribution rather to the special kinds of food used by the inhabitants, and to neglect of cleanliness in certain particulars, than to the influence of a high temperature. From all that is known of the way in which the body becomes infected by entozoa, even the greater liability to intestinal parasites observed in the negro in Africa and in the West Indies may be ascribed to the above mentioned causes, rather than to any particular susceptibility peculiar to that race. In the same way, the great frequency of *T. mediocanellata* among the Abyssinians is entirely due to their habit of eating raw meat, and not to any special predisposition. While all this is true, it is nevertheless necessary in warm countries to bestow special consideration to the intestinal entozoa as a cause of disease, first, because a high temperature is generally favourable to the free stage of some of them, as in the case of *Anchylostoma duodenale*, and consequently determines their introduction in greater numbers into the human body; second, because the human organism in tropical and subtropical climates is more sensitive to the action of certain morbid causes, and reacts reflexly with greater intensity; third, because gastric and intestinal catarrh, which are so frequently produced by worms, and which give rise to abnormal fermentations and hinder absorption, give rise to more pernicious effects in warm than in temperate and cold countries.

Many pathologists and writers on the diseases of warm countries have spoken incidentally of the great frequency of intestinal worms in man, and of the important part played by them in the pathology of such countries. The reader will find interesting remarks on this subject in Davaine's and Hirsch's classical works.[1] But we cannot omit mentioning among the recent observers who have contributed to extend the knowledge of tropical and subtropical helminthology, the names of Bilharz and Griesinger for Egypt; Grénet for Mayotta; Wucherer, Silva Araujo, Magalhães, and Lutz for Brazil; Lewis and MacConnell for India; Manson for China; Baelz, Scheube, and Ijima for Japan, and Bancroft for Queensland. Bilharz,[2] speaking of Egypt, says that that country is a field specially adapted for researches about helminths, as it is common there to find in the dead body hundreds of anchylostomes, along with numbers of ascarides and trichocephali, and thousands of oxyurides; and I have myself often had occasion

[1] See Davaine, *Traité des entozoaires*, Paris, 1877, in several parts of the work; and Hirsch, *Handbook of Geographical and Historical Pathology*, London, 1883, especially in vol. ii. p. 280.

[2] "Ein Beitrag zur Helminthographia humana," in *Zeitung für Wissenschrift Zoologie*, 1852, iv. p. 53.

to dwell on the important part played by *Anchylostoma, Bilharzia* and *Filaria sanguinis*, in the morbidity and mortality of man in this and other warm countries. Recently, Pasquale,[1] treating of the fevers peculiar to Massowah, assigns great importance to intestinal worms, and especially to *Ascaris lumbricoides*, and *Trichocephalus dispar* as factors in the genesis of those fevers, and asserts that their evacuation by suitable treatment is followed by the best results, and often by complete recovery. Erni,[2] in Sumatra, has likewise drawn attention to the importance of the injuries produced both by *anchylostoma* and *trichocephalus* in the wall of the intestine; and though he has exaggerated the importance of the latter in referring to it the origin of beriberi, he has verified its prevalence among the natives of the Malayan Archipelago. Kynsey says that *trichocephalus* is common in the bodies examined in Ceylon, and he is inclined to believe that its influence in the production of disease has been, on the whole, underestimated; and as for *anchylostoma*, he has established upon solid facts that the so-called beriberi of Ceylon (wet beriberi) is generally nothing else and nothing more than anchylostomiasis. Giles[3] more recently, referring to Assam, says that this country is a perfect hotbed for parasites, man and animals being alike affected with an universality that can hardly be surpassed elsewhere, and adds that "it is excessively rare to find an Assamese entirely free from parasites." Speaking particularly of *ascaris*, the same author states that "this parasite, as is well known, often causes serious symptoms, and its extreme prevalence in Assam is no doubt one of the causes of the low average of physical health of the inhabitants." He is inclined also to find a causal connection between trichocephali and dysentery, and, at all events, he says that trichocephali cannot fail to do something to contribute to the destruction of the digestive powers initiated by *anchylostoma*. To anchylostomiasis, as a main factor, he refers a disease that under the colloquial name of *kála-ázar* has caused a great increase in the mortality of Assam in recent years.

The great importance of intestinal worms as a disease factor is still more clearly shown by the recent discovery that some of them produce, by their metabolism, noxious substances of the nature of leucomaines or toxalbumins.

For these reasons it is important that the physician should always

[1] Pasquale, "Nota preventiva sulle febbri di Massauah," *Estratto dal Giornale medico del R. esercito e della R. marina*. Roma, 1889.

[2] Erni's Report on *Trichocephalus dispar* and Beriberi in Sumatra, as an appendix to Kynsey's Report on the Anæmia or Beriberi of Ceylon. Colombo, 1887.

[3] A Report of an investigation into the causes of the diseases known in Assam as Kála-ázar and Beriberi. Shillong, 1890.

bear in mind this source of disease when treating patients in warm climates. The practitioner ought never to postpone the investigations necessary for detecting the presence of intestinal worms in his patients,—I mean, the direct examination of the fæces, which alone can supply objective evidence of the presence of worms in the intestinal canal,—seeing that the practice of administering anthelmintic remedies in the absence of positive evidence of the presence of worms must be absolutely condemned. Anthelmintics generally are not innocuous remedies which can be used without clear indication.

In the case of some worms, such as the large *tæniæ* and *Bothriocephalus latus* (this latter cestode is now known even in Japan), a simple macroscopic examination of the fæces will afford sometimes this objective evidence, as we may find portions of them (proglottides) in the stools; but for the greater number of parasites a microscopical examination is necessary, as by it alone can the eggs or embryos of the worms be discovered. Sometimes this evidence may be obtained from a microscopical examination of vomited matter, which also ought never to be neglected.

Although the characters of the different eggs, which may be recognised in the dejecta as belonging to intestinal and liver worms, are described in handbooks of helminthology, and even in some recent treatises on pathology, it may not be out of place here to give a brief description of them, since an adequate knowledge of their characters is indispensable for the diagnosis of the diseases to which worms give rise. To facilitate our exposition, we submit to the reader the figures of the more important eggs as they may present themselves in the stools,—figures which we reproduce in part from the works of Leuckart, Bizzozero, and other writers, and in part from our own original drawings. (See Plate IV. Figs. 1–13.)

It may be as well to state at once that the examination of the stools for the detection of eggs is very easy and simple. The best method is simply to put a small portion of fæces on a slide, spreading it out uniformly, by applying and gently moving the cover glass to and fro. The fæces may be unmixed, or, if too consistent, they may be softened with a drop of water. The preparation is then placed under a low magnifying power, not exceeding 100 diameters. This answers best for the simple detection of eggs. A higher power will afterwards be useful in determining the particular characters which they present, when they are once in the field. When the fæces are liquid, they may be put into a conical glass; and after having been left to deposit, a drop of the sediment may be removed with a pipette and placed on the slide,

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 5.

Fig. 7.

Fig. 4.

Fig. 9.

Fig. 4

Fig. 13.

Fig. 6.

Fig. 10.

Fig. 12.

Fig. 8.

Fig. 11.

OVA FOUND IN FÆCES.

—the eggs, being heavy, fall to the bottom. The custom of mixing the fæces with carbolic acid solution, with the object of rendering the examination less disagreeable, is not always advantageous, as we then lose the opportunity of detecting the movement of any living organisms that may be met with, as embryos of *rhabdonema*, or protozoa, such as *amœbæ* or *cercomonas*. For these it is also necessary, if we wish to discover them by their movements, to examine the fæces while quite fresh, as they very soon die and become deformed, especially with cooling, in such a manner as not to be recognisable if not suitably fixed and stained. It is thus preferable to make at least the first examination without having recourse to the use of any reagent or antiseptic. Staining the preparation when our object is simply the detection of eggs is generally useless, and only in case of uncertainty is it to be resorted to.

With the simple method above indicated it is rare, if eggs were present in the fæces, that I have failed to detect them in a first, second, or third preparation examined systematically. When fairly abundant, as in ordinary cases of anchylostomiasis, they will seldom escape discovery in the first preparation.

As all eggs do not present themselves in every case with their more characteristic features, there may be some difficulty in certain instances in deciding by the examination of a single specimen the precise nature of the particular object under observation; in such cases it may be necessary to examine several specimens before pronouncing on the nature of the body, and, if it is found to be an egg, in deciding to what worm it belongs.

The eggs of *Ascaris lumbricoides*, though colourless when within the uterus of the worm, as seen in the fæces have a brown colour caused by the bile acting on the outer shell, which is, moreover, characteristically nodulated. They often present the form of a barrel rather than of an oval, and are of variable length, between 60 and 75 μ, with a breadth of from 36 to 55 μ, different specimens varying very much in the proportion between their length and breadth. The interior layer of the shell is transparent, colourless, and rather thick, and shows a multiple outline. (See Plate IV. Fig. 1, *a* and *b*.)

The *Oxyuris*, or thread worm, has colourless eggs, from 50 to 55 μ in length, and about 24 μ in breadth. They have a rather thin shell, but with double outline, and are unsymmetrical, the periphery being more curved on one side than on the other. They may contain a well developed embryo. In the solid fæces they are found more frequently in the superficial layers than in the centre of the mass; but are still more frequently to be detected in the

mucus coating the interior of the rectum. When the *oxyuris* infests a person, it is seldom that some one specimen of the adult worm does not appear in the alvine dejection. (Plate IV. Fig. 2.)

Trichocephalus, or "the whip worm," has eggs so characteristic that it suffices but once to have seen them (Plate IV. Fig. 3) in order to recognise them again at first sight; it is not possible to mistake them for any other egg found in human fæces. Their colour is accidental, as in the case of *ascaris*, and may vary between yellowish brown and red. Sometimes they are very small, not more than 36 μ in length by 26 μ in breadth.

The eggs of *anchylostoma* vary in size between 50 and 69 μ in length by 30 to 40 μ in breadth. They are oval in form, and present a very thin and transparent shell with a simple contour, enclosing a yolk of a greyish colour, more or less segmented, and separated from the shell by a zone of clear transparent fluid. (Plate IV. Fig. 4, *a* and *b*.)

The eggs of *tæniæ* occurring in the dejecta of man are very different from those of the nematodes mentioned. An important characteristic of them is presented by the hooklets of the contained embryo, which are more or less distinguishable. Eggs of *Tænia solium* and *T. saginata* (or *T. mediocanellata*) are distinctly characterised both by their thick and firm radially striated shell and by their smallness; they differ, however, very little from each other, those of *T. solium* being somewhat roundish, and those of *T. saginata* rather oval, the diameter of the former being generally no more than 33 μ (Plate IV. Fig. 9), whilst the long diameter of the latter may reach even 36 μ (Plate IV. Fig. 10). Eggs may not be present in the fæces of individuals affected with *tæniæ*, because the ova of these parasites are not set free within the alimentary canal, unless there is accidental rupture of a mature proglottis. The diagnosis of the two *tæniæ* is generally made by the detection of the proglottides, which escape either by themselves (especially in the case of *T. saginata*) or along with the fæces.

The eggs of *T. nana* are oval, with a thick, but not radially striated shell, and consist simply of a double membrane enclosing abundant amorphous matter containing a few granules (Plate IV. Fig. 11). They measure from 30 to 50 μ in length by 33 to 40 μ in breadth. Under a low magnifying power they appear as roundish bodies, with the central part more shining than the large outer rim.

The eggs of *T. leptocephala* (*T. flavopunctata* of Parona) are very large, attaining a diameter of from 60 to 70 μ, or even more. They are also distinguished, according to Grassi, by a radial stria-

tion of the outer covering, which is to be made out only with oil immersion lenses. (Plate IV. Fig. 12.)

The eggs of *Bothriocephalus latus*, according to Bizzozero, are oval and large, having a long diameter of 70 to 84 μ, with a rather thin and slightly brownish operculated shell, and coarse granular contents, the embryo being still undeveloped. (Plate IV. Fig. 13.)

The eggs of *Fasciola hepatica*, measuring from 130 to 145 μ by 80 to 90 μ, are the largest, after those of *Bilharzia hæmatobia*, of any ova found in the excreta (for those of *amphistomum* have not hitherto been met with in the fæces). They possess a thin and brownish shell, and are operculated. (Plate IV. Fig. 6.)

The eggs of *Distomum lanceolatum* have a thin brown operculated shell, and measure from 40 to 45 μ in length by about 20 μ in breadth. They generally contain an already formed embryo, which is covered with cilia at its anterior part. (Plate IV. Fig. 5.)

The opercula of the eggs of the three last mentioned worms are best displayed by treating them with a weak solution of potash.

The characters above given of the eggs of the three last mentioned worms, as well as the figures representing them, refer to the eggs as they are found within the worm, rather than to those they may exhibit after mingling with the contents of the intestine of man.

Of the eggs of *D. sinense* (Plate IV. Fig. 7) and *Bilharzia hæmatobia* (Plate IV. Fig. 8), and of other eggs, we shall speak in another place.

Rarer Intestinal Parasites.

Before proceeding to a somewhat detailed consideration of anchylostomiasis and rhabdonemiasis, conditions which, although not exclusively peculiar to warm climates, nevertheless play so important a part in the pathology of many tropical countries, we think it necessary to refer briefly to some entozoa which, having until now been met with only in tropical and subtropical regions, are of peculiar interest to the physician practising in warm countries. These are: *Distomum heterophyes*, *D. crassum*, *Amphistomum hominis*, and *Tænia madagascariensis*.

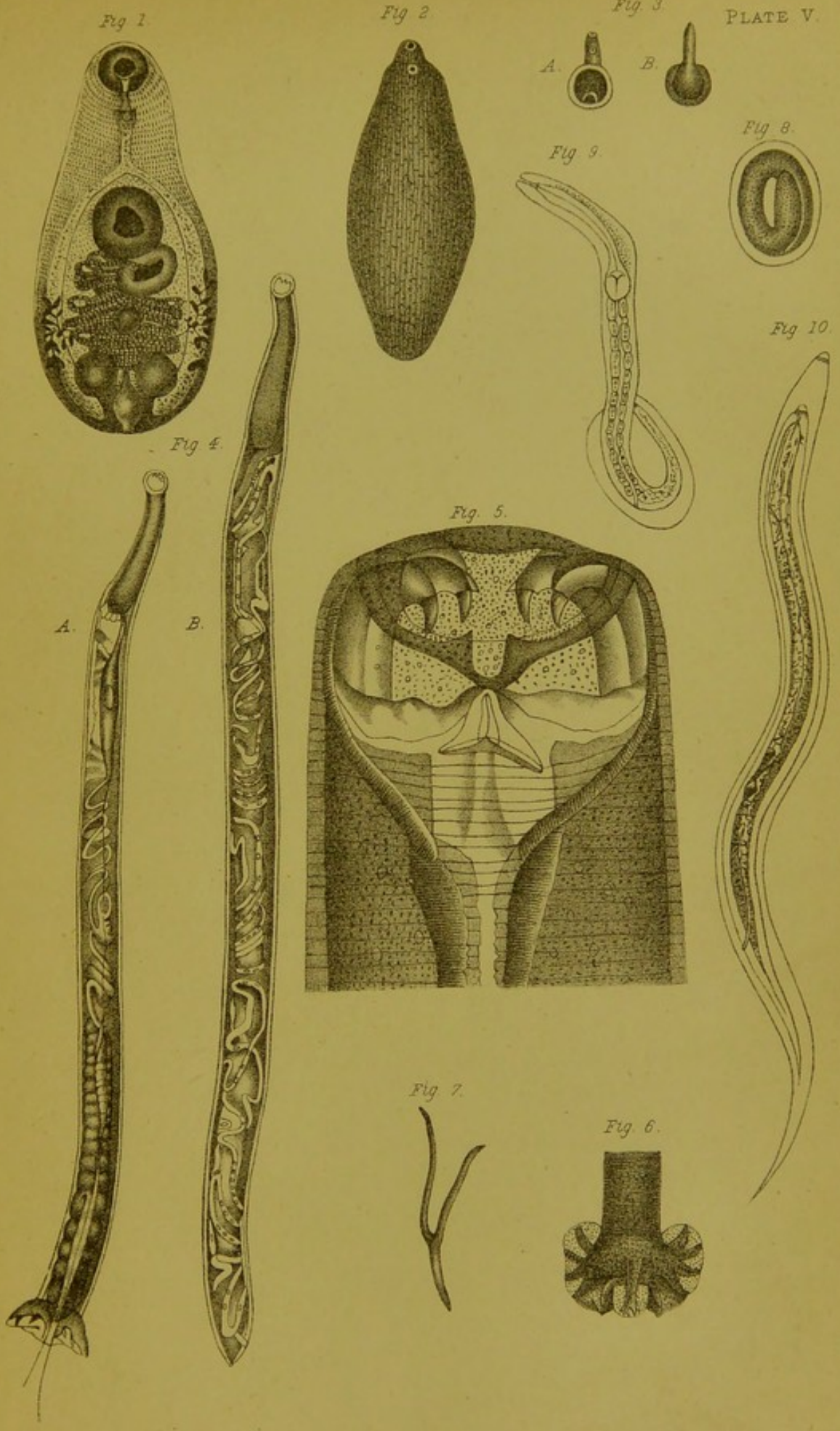
D. heterophyes (Plate V. Fig. 1) is a very minute fluke, measuring in length no more than from 1 to 1½ mm. It has been found only twice—in dead bodies—at Kasr-el-ain Hospital in Cairo, by the late Dr. Bilharz. As yet we know nothing as regards its pathological and clinical importance; it is well, however, to know the characters of its eggs, so that in future its presence may be recognised in the living by means of the microscopical examination of the stools. These eggs are very small indeed, the smallest yet met with in human

stools, measuring, so far as is known, about 26 μ by 15 μ. They have a thick shell, are oval in form, and in colour—which they communicate to the entire parasite—reddish-brown.

D. crassum, or *D. Buski* (Plate V. Fig. 2), is the largest fluke hitherto found in man, measuring about 6 or 7 cm. in length by from 1·5 to 2 cm. in breadth. It is distinguished from the common liver fluke (*Fasciola hepatica*) principally by its simple non-ramified intestinal cæca. This worm was originally discovered by Professor Busk in the duodenum of a Lascar who died at the Seamen's Hospital, Greenwich (1843), and has subsequently been observed several times in persons living in or coming from China. Its ordinary seat seems to be the duodenum; in some of the observed cases it was found in the stools of the patients. The eggs are large, being about 125 μ in length by 75 μ in breadth, and are thus only a little smaller than those of *F. hepatica*, and more than twice the size of the eggs of *anchylostoma*. Their other characters are as follows: operculated; regular, distinct, oval outline; walls thin; contents—completely filling the cell wall—roughly granular, containing numerous highly refracting large granules. In the cases observed by the late Dr. Cobbold[1]—a missionary, his wife and daughter, who had been resident four years in Ningpo (China)—there was a history of diarrhœa and hepatic complaints. According to Cobbold, there were grounds for suspecting that in these cases the infection with *D. Buski* took place from the use of a peculiar Chinese oyster as food. Although the clinical features of this infection are not yet well known, it seems that *D. Buski* is not harmless, and that it deserves further attention from physicians practising in China and the East. In these countries its presence should be suspected, especially in obscure cases of liver and intestinal complaints. Recently cases have been observed in Selangor, in Assam, and in Borneo, which leads us to infer that the range of this worm is not limited to China. A full account of one of these cases was given by Dr. Sinclair of Selangor in 1889, and two other cases were described last year by Dr. Walker of Sandakan, Borneo.[2] The case in Assam, recorded by Dr. Giles, was that of a little girl of Chaygaon, who passed a single specimen of this fluke, along with several other species of worms, after the administration of thymol.[3] As regards the treatment of this parasite, we may also mention that in one of Walker's cases the fluke was expelled by means of thymol and santonine, administered for anchylostomiasis. Thymol, in

[1] Cobbold, *Parasites*, London, 1879, p. 20 and following.
[2] *Brit. Med. Journ.* of 5th and 12th December 1892.
[3] Giles, *loc. cit.* p. 125.

PLATE V.



INTESTINAL FLUKES AND ANCHYLOSTOMA.

small doses of a few grains, having succeeded in this case (although its action may have been increased by the santonine), we may hope that this drug may hereafter prove a remedy for this parasite, as well as for *anchylostoma*. We may observe that in one of Cobbold's cases some specimens of the worm were expelled during the use of a milk diet simply, after drastics had failed to remove it.[1]

Amphistomum hominis (Plate V. Fig. 3, *a* and *b*).—The first account of this worm was published in 1876 by T. R. Lewis and J. F. P. MacConnell,[2] who received the parasite from Dr. O'Brien and Dr. Curran, who found it at Gauhati in a post-mortem of an Assamese. Lewis and MacConnell discovered that a specimen of this worm (though not classified) existed already in the Pathological Museum of the Calcutta Medical College, and had been received from Dr. Simpson, who had found it, so far back as 1857, in a post-mortem of a patient who died at the Tirhoot Jail Hospital. Until now it has been only found in India. In one case, in which it was present in great numbers, red spots were observed, corresponding to the points where the worm had been attached to the mucous membrane of the cæcum and ascending colon. It has recently been repeatedly met with by Giles,[3] but not in numbers sufficient to constitute a danger to the patient. In no case did he observe more than a dozen or so. It is probable that this parasite will not prove to be of great clinical importance, as it belongs to a rather innocuous class of worms. The eggs, which possess a shell with operculum, are oval shaped, and measure 150 μ in length by 72 μ in breadth. It is curious that Giles assures us that he had never met with the ova in the dejecta, even in the case of patients who were afterwards proved to harbour it. According to the same observer, thymol appears to expel this worm with tolerable certainty.[4]

Tænia madagascariensis.—Another intestinal parasite, which has hitherto been met with only in warm countries, whose range, however, has lately been found not to be so restricted as was at first supposed, is *T. madagascariensis*. This cestode, so far as we know up to the present time, reaches a length of at least 24 cm., and a breadth of no less than 2·6 mm. It is provided with a rostellum, armed with numerous hooklets (about ninety) of a peculiar

[1] Specimens of *Distomum crassum* may be seen at the Hunterian Museum, as well as in those of King's College, Middlesex Hospital, and Charing Cross Hospital. *D. rathouisi*, evacuated by a Chinese woman suffering from liver disorders, and described as a distinct species in France, is probably the same as *D. Buski*.

[2] Lewis and MacConnell in *Proc. Asiatic Society of Bengal*, August 1876.

[3] Giles, *loc. cit.* p. 124.

[4] *A. hominis* resembles much in its form an amphistomum I found in horses in Egypt (*Gastrodiscus Sonsinoi*, Cobbold), and which has since been found in horses in Senegambia, in Guadeloupe, and in Assam.

form, and presents to the naked eye, in the ripe proglottis, numerous well-marked oval bodies, which are balls of eggs. According to Leuckart, it approaches in its characters to *T. tetragona*, a *tænia* proper to fowls. *T. madagascariensis* was found first in Mayotta by Dr. Grénet, then in Bankok (Siam) by Dr. Deuntzer (Leuckart's[1] case), and by Dr. Chevreau[2] in Mauritius. Thus its range comprises both Africa and Asia. It is important to note that in all the observations (seven cases) the subjects were children not more than 5 years of age; and that in some instances they suffered from nervous disorders, and even from convulsions. Filix mas, as for *T. nana*, is the only remedy that is known to have effected its complete expulsion.

It is very probable that the four worms of which we have just spoken will in future be found in other places, especially in countries hitherto imperfectly explored from a medical point of view. Cobbold[3] justly observes "that we possess very little knowledge of the parasites which take up their abode in the viscera of savages; and that several of the human parasites, which we now consider to be rare, would be found to be abundant, if, by means of post-mortem examination and other methods of investigation" (and, we may add, particularly microscopical examination of the excreta), "we search for them especially amongst the raw flesh- and fish-eating savage tribes."

Other intestinal cestodes have been from time to time described as new, and as especially proper to hot countries, as *T. capensis* (Küchenmeister[4]), *T. nigra* (Davaine[5]), *Bothriocephalus tropicus*, (Schmidmüller[6]); but they are not yet recognised as distinct species, and will probably be found to be only abnormal forms of already known species.

Some parasites of the class of protozoa are sometimes found in the intestine of man. Amongst them must be noted some *Cercomonas*, *Balantidium coli*, and *Coccidium perforans*. As these have not been found to be special to warm countries, and as their pathogenetic importance has not as yet been well established, it is unnecessary to treat of them in this place; we only refer to them in order to remind pathologists of their existence, and as

[1] Leuckart, "Ueber Tænia madagascariensis," *Separat Abdruck aus Verhandlungen d. Deutsch. Zool. Gesellsch.* 1891.

[2] Blanchard, "Note sur quelques vers parasites de l'homme," *Comptes rendus de la Societé de Biologie.* Séance du 18 Juillet 1891.

[3] Cobbold, *Parasites*, p. 27.

[4] Küchenmeister, *Parasiten*, Leipzig, 1855, Erst. Abth. p. 93, and its English translation by Dr. Lankester.

[5] Davaine, *Entozoaires*, p. li.

[6] Gervais et Van Beneden, *Zoologie médicale*, t. ii. p. 243.

subjects deserving special investigation in the future. Importance has been given to certain *amœbæ* as being the cause of dysentery and of the liver abscesses consecutive to it; some account of them will be found in the chapters dealing with those diseases. Although we were the first to signalise the presence of *amœbæ* in dysentery in Egypt,[1] we do not share the opinion of those who hold that they are the cause of that disease, seeing that they are not constantly present in all cases of dysentery, and that they are also found sometimes apart from dysentery.

Anchylostomiasis.

History and Geographical Distribution.—As *Anchylostoma duodenale* is now known to exist in so many countries, it may be placed among the proximately cosmopolitan entozoa infesting the human body. Of those countries, some, like the European ones, belong to temperate climates. It is only in warm climates, however, that we find it generally diffused among the population, and becoming the cause of a very common, and sometimes grave anæmia, which must be regarded as a scourge to many tropical and subtropical countries.

The first account of *A. duodenale* was published by its discoverer, Dr. Dubini, in Milan, in 1843, though he had observed it for the first time in 1838.[2] Dubini had a clear conception that this entozoon must have an injurious effect on the organism; but he did not dwell much on the clinical aspect of the subject. Bilharz and Griesinger[3] (1851–53) not only confirmed the existence and great frequency of *anchylostoma* in Egypt, which was first seen there by Pruner in 1846, but Griesinger came to the conclusion that this entozoon was the direct cause of Egyptian chlorosis.[4] The term "Egyptian chlorosis" is only the local name for a form of anæmia occurring in many warm countries, in which *A. duodenale* has been found to infest the body.

The detection of the parasite on the other side of the Atlantic was first effected by Wucherer, at Bahia in 1866.[5] Very soon after this others found *anchylostoma* at Rio de Janeiro; Grénet dis-

[1] See Leuckart, *Parasites of Man*, i. p. 187.

[2] See "Nuovo verme intestinale umano (*Anchylostoma duodenale*, etc.)," in *Annali Univers. di Medicina di Omodei*, 1843, cvi. pp. 5–13; and *Entozoografia umana*, Milano, 1850.

[3] Griesinger, 'Anchylostomem Krankheit und Chlorose," in *Archiv f. phys. Heilkunde Jahrg.* 1854, xiii. p. 555.

[4] According to Joachim's translation of Eber's *Papyrus*, dating from the earliest historical times, a disease is therein described, which, from its symptoms, must be referred to this Egyptian chlorosis caused by *anchylostoma*. See *Brit. Med. Journ.*, May 13, 1893.

[5] Wucherer, *Gazetta Medica da Bahia*, 1869, No. 65.

covered its existence at Mayotta (one of the Comoro Islands); and Riou Kérangal at Cayenne. We may add that since then the existence of *anchylostoma* has been ascertained in many places in all parts of the world. So, not to speak of Europe, but only of warm countries, it has been discovered in Egypt and Tunis,[1] in Mayotta, and in other islands appertaining to Africa; on the Zanzibar Coast, in Senegambia, Sierra Leone, Guinea, and the Gold Coast. In Asia, it has been met with in India, particularly in Lower Bengal, Travancore, Ceylon, Assam, in the Malay Peninsula; also in Cochin-China, Tonquin,[2] Japan, Java, and Borneo. In Australia, it has been found in Queensland;[3] and in America, besides Brazil, it has been observed in the West Indies, Colombia, Venezuela, Guiana,[4] Peru, Bolivia, and in some districts of the United States, as Louisiana, Alabama, and Georgia. The known northern limit of *anchylostoma* is now about 51° 31′ N., at Dortmund (Westphalia); and the southern limit at about 30° S., at Gudna in Queensland, and in the province of Santa Caterina in Brazil. But it is probable that the southern limit will be extended to higher latitudes by further research.

Important facts in the history of *anchylostoma* are:—The discovery (Grassi and Parona, 1878) that the diagnosis of the existence of the worm in the human body can be made from the presence of its eggs in the dejecta, and the finding of the best remedy to expel it ("thymol," by Bozzolo, 1880); then, the Saint Gothard epidemic (1880), which specially directed attention to the entozoon, and led to its discovery in many parts where before it had passed unobserved; lastly, the discovery of anchylostomiasis in Ceylon and Assam, where this parasitic disease was formerly associated with the colloquial terms of *beriberi* and *kála-ázar*.

Characters of the Parasite and its Natural History. —*A. duodenale*, or *Dochmius duodenalis*, belongs to the order *Nematodæ*, and to the family of *Strongylidæ*. We will only indicate those characters of this entozoon that suffice for distinguishing it from other parasites of man. Its colour is whitish when its alimentary canal is empty, and reddish or brown when this is filled with blood more or less recent. Its length is from 7 to 18 mm., and its breadth about $\frac{1}{20}$th of its length. Both males and females (see Plate V. Fig. 4, *a*, *b*) are cylindrical, with conical pointed head and a peculiar bulging mouth-capsule; but while the

[1] I have found *anchylostoma* frequent in Gabes, South Tunisia.

[2] See de Santi, *De l'entérite chronique paludéenne ou diarrhée de Cochinchine*, Paris, 1892, p. 153.

[3] See *Lancet*, 1889, i. p. 750.

[4] Grieve, "Endemic Disease in British Guiana" (quoted by *Brit. Med. Journ.* 1st March 1890, p. 470), speaks of *anchylostoma* having been found in that country by Dr. Ozzard.

females are pointed at the posterior end, the males present in this situation an enlargement, due to a bell-shaped *bursa copulatrix* characteristic of this family of worms, and which serves to distinguish the males from the females, even to the naked eye. The mouth-capsule (Plate V. Fig. 5), whose aperture is directed to the dorsal aspect, is provided with two strong, claw-shaped teeth on each side of its upper ventral margin; and also with similar but not so strong tooth-like projections, one for each side, on the opposite dorsal margin.

Male.—Length up to 12 mm. and even 15 mm. The bell-shaped bursa (Plate V. Fig. 6) is trilobate, and is provided with eleven ribs, five on each side, and one median, which has a common origin with the two contiguous lateral ones. The median rib bifurcates at the end, and each bifurcation presents three small digitations. The two spicula are long and slender.

Female.—Length up to 18 mm., and breadth not more than a millimetre—a little more than that of the male. It has the vulva between the anterior two-thirds and the posterior third of the body.

When male and female are conjugated they present the form of a γ (see Plate V. Fig. 7). From the characters given it is scarcely possible to mistake *anchylostoma* for other human entozoa. From *Dochmius trigonocephalus* and kindred species infesting dogs and cats it is differentiated, in that these latter have three instead of two claw-shaped hooks for each side in the upper ventral margin of the capsule.

The adult animal lives generally in the upper part of the small intestine of man. The jejunum seems to be its favourite site rather than the duodenum. The eggs of the parasite are expelled with the fæces, and may be detected by microscopic observation of the stools. Their characters have been already described on page 868 (Plate IV. Fig. 4, *a* and *b*). The segmentation of the vitellus has hardly begun when the eggs reach the vagina of the worm, and is never completed within the body of the parasites. In fresh stools, eggs are sometimes seen still unsegmented, but more generally the vitellus presents two, four, or more spherules of segmentation. After remaining some time in the fæces, segmentation is completed, reaching the morula stage. It seems that the embryo is never found developed in the fæces up to the time of their evacuation. In the cold season of temperate climates, the lapse of some days is necessary for the formation of the embryo; but in summer, and when the temperature is about 30° to 35° C., twenty-four hours, or even less, is found to be sufficient for its development. According to Perroncito,[1] a temperature above 35° C. retards the develop-

[1] *I parassiti dell' uomo e degli animali utili*, Milano, 1882, p. 343.

ment of the embryo; and this author explains in this way the fact that the embryo is not formed in the interior of the human body. It is more probable, however, that the hindrance to the formation of the embyro in the interior of the human body is due to other circumstances inherent to the surroundings, among which may be want of air.

The embryo (Plate V. Fig. 8) in the egg has a short cylindrical form, which gradually increases in length. As soon as it has attained its full development, it makes this manifest by movements within the shell. Finally, it bursts its covering and slips out, generally head first. The newly hatched worm (Plate V. Fig. 9) is very different from the adult, and shows a typical rhabditic form, characterised by a spindle-shaped œsophagus ending in a chitinous bulb provided with three chitinous ridges resembling an anchor, and an abruptly pointed tail. When emerging from the shell the embryos have an average length of a little more than 0·20 mm., and a breadth of 14 μ. They then grow more or less rapidly in relation with the degree of temperature and as other circumstances are more or less favourable. During growth the young larvæ undergo an incomplete kind of moulting, or process of ecdysis, which gives them an appearance as if they were enclosed in a kind of case, which is constituted by the detached transparent skin. The larva may fill the case completely, so that the existence of the latter is only recognised by the double contour of the worm; at other times the case is larger than the parasite, so that it projects very considerably either beyond the tail end, or head end, or both, whilst the larva moves inside (Plate V. Fig. 10). This is what has been called by some observers a kind of encystment. But the generality of observers now agree that this appearance is due simply to moulting, which is, however, incomplete, as the detached skin, instead of being thrown away, remains to clothe the larva. What has been described as a stage of calcification of the capsule was misinterpreted, the appearance being really brought about by the death of the larva.

Until recently, all observers were agreed in this, that the larva having attained a certain length, and having undergone some modifications in its digestive canal, especially as regards the loss of its chitinous bulb, is capable of developing directly into the parasitic stage, requiring only an opportunity of being introduced to the digestive canal of man to grow there into the adult *anchylostoma*. But, lately, Giles'[1] researches in Assam have cast doubt on this simple theory of development. According to Giles,

[1] A Report on Kála-ázar and Beriberi. Shillong, 1890.

anchylostoma, like other nematodes, presents what zoologists call dimorphobiosis, or heterogenesis; in other words, it accomplishes its life history in two different and complete sexually mature stages, one of which is parasitic and the other free.[1] Thus the embryo born of the parasitic worm reaches an adult free stage, but only the progeny of the latter, viz. the grandchildren of the parasitic worm, are capable, after having reached a certain degree of development, of assuming the parasitic life whenever introduced into the human organism. Giles, having made experimental cultures in earth, assures us that he has succeeded in obtaining an adult free stage. Of this he gives drawings representing both the male and the female forms, which certainly are very different from the *anchylostoma* parasitic in the human organism.

We restrict ourselves to mentioning this result of Giles' interesting researches, without pronouncing on their definitive value, as it seems to us that, notwithstanding his assurance that he has avoided all sources of error, it is yet possible that the rhabditic adult form he observed in his cultures may prove to be only one of those numerous species of free nematodes that live in mud, as *Rhabditis terricola* (Dujardin), and which are very difficult to eliminate in experimental cultures of both *anchylostoma* and *Rhabdonema intestinale*. Lately, Dr. Macdonald, of Colombo, has advanced that the free stage of *anchylostoma* described by Giles is nothing other than the free stage of *R. intestinale*.[2]

Notwithstanding the uncertainty still existing about the life history of *anchylostoma*, we may consider it as settled to some extent that man actually takes the infection of *anchylostoma* from earth that has been soiled with dejecta containing the eggs of this worm. Eggs left in undiluted fæcal matter develop embryos; but these do not live a long time in such a medium, as they require for their further growth, besides a certain temperature, other conditions which are better fulfilled when the fæcal matters are deposited on the ground. The principal of these conditions are a limited amount of humidity, and a free exposure to the air.

In consequence of the infection taking place through the soil, it comes about that certain classes of persons are specially liable to contract anchylostomiasis, viz. those who handle earth in which dejecta containing the eggs of *anchylostoma* have been deposited. This in European countries happens especially to brickmakers and those

[1] Leichtenstern in 1886 had already advanced the dimorphobiosis of *anchylostoma*; but he very soon recognised that he had been misled, and then rejected the new theory, now again sustained by Giles.

[2] See *Ceylon Medical Journal*, January 1892, p. 30.

engaged in mining and tunnelling, and also to rice planters, and, more rarely, to those employed in other agricultural operations. But in warm climates, where the free stage of the *anchylostoma* finds more favourable conditions for development owing to the constant high temperature, the parasite is much more widely spread among all those who have to labour on the soil; and as this class comprehends a great part of the total population, it follows that anchylostomiasis is in such countries a very widely diffused disease.

It follows from all this that there is no particular disposition associated with race or age to infection by *anchylostoma*, all those being equally subject to it who are engaged in handling contaminated earth or mud, and who do not pay proper attention to avoid swallowing the larvæ.

When the larval form has been introduced into the digestive canal, it has been supposed by some that its first parasitic development takes place while in a state of encystment in the walls of the digestive canal. This supposition has arisen from the fact that very young worms have never been found in the interior of the intestine in the dead body, and also because young specimens are absent in the dejecta after the administration of thymol. But till now such an encystment, as that which has been described for *Strongylus tetracanthus* in the horse, has never been proved in the dead body for *anchylostoma*. It is true that *anchylostoma* has been found in blood effusions in the connective tissue under the mucous membrane, as was first observed by Bilharz and Griesinger, and as I have myself seen several times in dead bodies in Egypt. But this is not a true encystment, seeing that the worm still maintains communication with the intestinal canal by means of the perforation through which it had migrated. The smaller immature specimens found once in the intestinal walls by Grassi measured between 3·6 and 4 mm. in length, and they were both male and female. It is possible, though till now this has not been confirmed by observation, that the very young anchylostomes have their ordinary abode in the walls of the intestine, instead of in the lumen of that organ.

According to Leichtenstern's last observations, supported by feeding experiments on man, the larvæ of *anchylostoma* introduced into the digestive canal lose their covering, not in the stomach, but only when they reach the intestine. There they grow *on the surface of the mucous membrane*, and become sexually mature animals, after having undergone a true process of ecdysis, but without passing through a stage of encystment in the intestinal wall. *Five weeks* at

least are necessary, according to this observer, for them to attain full sexual maturity, after their introduction into the digestive canal. In fact, Leichtenstern in one case found 250 *anchylostoma* still quite small and not yet sexually mature in the *fourth week* after infection. The female genital tubes were without ova (in the oviduct), and the male without formed seminal elements.

As for the duration of the life of *anchylostoma*, there is yet no accord among observers. Shulthhess and Giles hold that it is short, not exceeding several months. If anchylostomiasis is a disease of very long duration, they explain this by assuming several reinfections. But I am inclined rather to the opinion that ascribes to *anchylostoma* a very long life. Indeed, I had a case of a girl who suffered from her childhood with anæmia, which I found to be due to *anchylostoma;* for nine years she had not visited the place in which, I presume, she had become infected, whilst in her new residence the presence of *anchylostoma* in other individuals has not yet been reported.

Symptoms. — Under the term "anchylostomiasis" is now understood the disease produced in man by the presence of *anchylostoma.* It consists especially in a peculiar progressive anæmia, often associated with dyspepsia and other functional and painful disorders of the intestine, and which, in the course of time, leads to fatty degeneration of the heart and other organs, with serous effusions, and even to death.

The symptoms point especially to digestive and circulatory disturbances, and to malnutrition, which is expressed more by anæmia than by emaciation, whenever the patients can obtain sufficient food. The first symptom is generally a feeling of pain in the pit of the stomach, which sometimes extends towards the navel. This pain, which is augmented by pressure, is rarely wanting. It is generally accompanied by dyspepsia, and often assumes the characters of colicky pains with borborygmi. Sometimes there is anorexia, but more often the appetite is ravenous, the feeling of pain being alleviated by eating. Generally over indulgence in food is not easily supported. Sometimes there is a depraved state of the appetite, manifested by what is called allotriophagy, pica, or geophagia, as happens in ordinary chlorosis and in pregnancy. The geophagia, which has been pointed out as frequent in negroes, is probably sometimes itself a means of increasing the disease, causing fresh introductions of the *anchylostoma* larvæ. This geophagia was noted by Macdonald as of common occurrence among labourers on coffee estates in Ceylon, and by Kynsey, particularly in children.[1]

[1] See the *Ceylon Medical Journal*, July 1890, p. 294; and Kynsey's *Memorandum on Anchylostomiasis, or the Anæmia of Ceylon* (revised; issued originally in 1886).

According to Cobbold and others, geophagia is a common symptom of helminthiasis of all kinds, and is not peculiar to man. The same habit has been noticed in elephants affected with flukes (*Masuri*), as well as in horses affected with *Strongyli* and other parasites.

The bowels generally are constipated; only when the disease is advanced is there sometimes diarrhœa alternating with the constipation. When diarrhœa is marked, it may be due to the complication of *Rhabdonema intestinale*. The stools examined macroscopically often present no alteration. But sometimes a peculiar reddish-brown colour of the fæces indicates that they are mixed with blood. Unmixed blood is rarely seen in them; not so rarely, however, according to Lutz's experience in Brazil.[1] Generally the blood from the bites of *anchylostoma* exudes little by little, and may be altered in its slow passage through the intestines in such a manner as not to be recognisable in the stools, even by the microscope. But frequently mucus tinged with blood is observed in the stools.

Finding ova in the fæces by the microscopical examination of the stools proves the presence of the worms in the bowel. According to Grassi and Parona, 150 to 180 eggs in one centigramme of fæcal matter correspond to 1000 worms (viz. about 750 females and 250 males—the ordinary proportion of females to males being reckoned at three to one). Although the eggs are, as a rule, uniformly distributed throughout the fæces, and there is no doubt that the proportion of eggs in a given quantity of fæces is in relation to the number of females present in the intestine, there are other and variable conditions, such as the consistence of the fæces, and the time occupied by the alimentary matters in passing through the first part of the intestine, which must affect any such calculation and make it unreliable. It is not, therefore, always possible to judge the number of worms from that of the eggs.

In order to determine approximately the proportion of the eggs in the fæcal matter, Lutz recommends the following plan:—One part of fæces is uniformly mixed with three parts of water, and of this mixture 1 decigramme (about three drops) is placed on the slide, and flattened by laying a special glass plate over it. This latter must have a surface area of 25 mm., divided in squares of 1 mm. each. In each of these squares there appears 4 milligrammes of the fluid, or 1 milligramme of fæces. When every square offers an average of from 15 to 18 eggs, we may infer that the patient

[1] See Lutz's "Ueber Ankylostoma duodenale und Ankylostomiasis," in Volkmann's *Vorträge*, Nos. 255, 256, 265; or the translation by J. D. Macdonald, M.D., in the *Ceylon Medical Journal*, 1890–91.

is infected by 1000 worms, according to Parona's and Grassi's reckoning.

Eggs of *oxyuris* may be mistaken for those of *anchylostoma;* and I think that the allusion to the frequency of the occurrence of great numbers of the eggs of *oxyuris* in the fæces, made by some observers previous to the discovering of *anchylostoma* eggs in dejecta, is attributable to their having referred both *anchylostoma* and *oxyuris* eggs to *oxyuris* alone. According to my observations, although thread worms (*oxyuris*) may often be present in the bowel, yet we may not, and often do not, find their eggs in the fæces, especially if the latter are solid, and the central part only of the mass is examined. Moreover, *oxyuris*' eggs, as we have said, are a little smaller than those of *anchylostoma*, and present a double contour, and are unsymmetrical (Plate IV. Fig. 2). The eggs of *ascaris* (Plate IV. Fig. 1, *a* and *b*) are so very different as not to be easily mistaken for those of *anchylostoma*, being larger, and having a nodulated and yellow-brown outer shell; but when, as may happen, they present themselves under the microscope, having lost their outer shell, they may at first sight be confounded with those of *anchylostoma;* in this case they are distinguishable by their transparent shell, with a double or multiple outline.

The frequency of intestinal *coccidia*, signalised by Giles in patients affected by *anchylostoma*, seems to be peculiar to Assam; at least we do not know that this condition is endemic in any other country.

In the microscopic examination of the dejecta of persons whose food is principally composed of vegetables, it is usual to meet with great quantities of vegetable cells, which certainly could not be mistaken for eggs; but often these vegetable cells so cover the field as to obscure the latter. Thus, in difficult cases, it is desirable to examine the stools after having placed the patient on a simple diet of milk, eggs, and meat only for two or three days. But this, generally, is only possible in hospital practice.

Very often we find, in the stools of patients affected by *anchylostoma*, detritus from undigested food in greater abundance than in the stools of individuals in good health. Octahedral crystals, like Charcot's, also occur; and, according to Leichtenstern, they are found also in the fæces of all individuals infested with intestinal worms of other species.[1]

To revert to the symptoms and course of the disease. The patient begins by and by to become pale, and feels weak and weary. He is subject to palpitation of the heart, especially on exertion.

[1] See "Ueber die Charcot-Robin'schen Krystalle in den Fæces nebst einer Bemerkung über Tænia nana in Deutschland," in *Deutsch. med. Wochenschrift*, 1892, No. 25.

Auscultation elicits the same symptoms as are found in chlorotic and other anæmic subjects, such as murmurs in the heart, in all the larger arteries, and in the jugular veins. The pulse is often abnormally quick and small, sometimes weak and slow—features which differ in relation to differences in the age, constitution, and habits of patients. Slight œdema of the feet or face points to a high degree of anæmia, and to a lowered state of the heart's action.

The observations respecting the microscopic alterations of the blood have not hitherto given uniform results. Generally it appears that these alterations consist more in a deficiency in the number of the red corpuscles (oligocythæmia) than in the want of colouring matter in them (oligochromæmia), or in alterations in the form of the individual corpuscles, like microcytosis or poikilocytosis. The number of red corpuscles may be reduced to one million and even less per cubic millimetre. The proportion between red and white corpuscles is not altered in ordinary cases; only in the far advanced stage is there an increase of white corpuscles. The alterations of the blood found in anchylostomiasis, together with the prevalence of fat in the body, are indicative of anæmia from loss of blood rather than of anæmia from defective assimilation.

When the disease is advanced, the patient is often subject to small elevations of temperature, and to very slight fever. In far advanced cases it is common to observe a subnormal temperature. Giles had a case in which the temperature did not rise to normal for a week together. In this case, in which an attack of intercurrent pneumonia supervened, which proved fatal, the temperature ran up to 103° F., and remained above normal for some days. But it became again subnormal (93°·8 F.) for several days before death. In another case (Case 32), Giles observed the temperature persistently subnormal, and two hours before death it was only 90° F. in the rectum—a low temperature that reminds us of those met with in anæmia, from the abuse of leeching in tropical invalids, as recorded by Sir James Ranald Martin.[1]

Pallor is especially noticeable in the mucous surfaces of the mouth and eyelids, where the bloodlessness reaches such a degree as to be comparable to that observed in states of syncope. The legs feel heavy, especially in ascending; walking becomes difficult, and causes painful dyspnœa and palpitation; giddiness occurs frequently, especially on rising up, or on stooping down. Often, too, there is tinnitus aurium, and dimness of vision. Sometimes fainting occurs. The spirits are depressed, the patient apathetic; he becomes lazy and unable to work, feeling better in that state of *dolce far*

[1] Influence of Tropical Climates, etc., 2nd ed., London, 1861, p. 654.

niente to which the inhabitants of warm climates are already inclined.

Vomiting of blood as well as severe gastralgia, looked upon by some observers as symptoms of anchylostomiasis, should rather lead us to suspect a complication like gastric ulcer. It is even doubtful if dilatation of the stomach, so common in such patients, can be referred simply to the anchylostomiasis, but rather to the quality and bulky nature of the food used generally by the class of persons most subject to this infection. While vomiting, flatulence, and pain point rather to gastric catarrh than to bowel complaints, it is possible that these symptoms may be due to the presence of anchylostomes in the stomach, a thing which may happen, though it is not frequent. To ascertain the fact, it is necessary to examine the vomited matters microscopically, in which may be found the eggs of the worm. It must not be forgotten, however, that their presence in the vomit may be due simply to a regurgitation of the contents of the duodenum during the efforts of vomiting.

Lately, Fischer, of Dortmund, has verified in some cases of anchylostomiasis the existence of retinal hæmorrhages, which had already been signalised by previous observers (Grassi), and which are also met with in simple, pernicious, and other forms of anæmia. This fact is rather difficult to explain, because hæmorrhages secondary to anæmia produced by simple loss of blood (considered by many pathologists to be the condition met with in anchylostomiasis) are not of common occurrence. They may be a consequence of marasmic thrombosis.

According to Lutz, children suffering from anchylostomiasis, for years during the developmental period, are small and puny, and at 25 years of age look like children of 10 or 12. In accordance with this statement, I had a case of a girl, 18 years old, whose infection went back perhaps to 9 years, and who looked as if she were no more than 13 or 14. She was not emaciated; although extremely pale, weak, and not well developed, she was rather plump; and I may add that I have never seen emaciation in cases of anchylostomiasis uncomplicated by wasting diseases, unless in persons constrained to suffer from deficiency of food. In a case[1] of simple and genuine anchylostomiasis in a girl, which ended fatally, and at whose necropsy I was present, I noticed that the body presented as much adipose tissue as is usually observed in a well-nourished and healthy young person. Yet there is no doubt that in some cases of chronic and long standing

[1] See "L'Anchilostoma duodenale in relazione coll' anemia progressiva perniciosa Nota," etc., in the journal *L'Imparziale*. Firenze, 1878.

anchylostomiasis, especially in persons of rather advanced age, the impaired digestive powers, and consequent diarrhœa, may give rise to emaciation, as has happened in the experience of Lutz and others.

A patient suffering from anchylostomiasis may sometimes fall into a very pitiable condition in a relatively brief time, viz. in a few weeks. When infection is brought about by the simultaneous introduction of enormous numbers of larvæ, the disease runs a very acute course. These are the cases that are so dangerous, if not relieved in time. Leichtenstern explains the sudden appearance of anæmia and grave symptoms in the acute cases in the following manner. By his feeding experiments he has assured himself that *anchylostoma* during the first *four weeks* after its introduction is innocuous, and that it is not until the fifth week, that is, when it has attained full sexual maturity, that it begins to give trouble. In fact, a patient who died of *phthisis pulmonum* in the *fourth week* after infection, exhibited no intestinal symptoms, nor signs of marked anæmia, and in the dead body only small and immature *anchylostoma* were found, as we have already observed. When sexual maturity is reached, according to Leichtenstern, *anchylostoma* becomes very active, moving about a great deal and inflicting numerous bites on the mucous membrane, thereby giving rise to violent pains with hæmorrhagic stools, and a sudden appearance of a high degree of anæmia. Leichtenstern adds that this lively locomotion of the parasites is only observed during the periods of copulation, which occur in an intermittent manner. Thus he explains the intermittent character of the pains and hæmorrhages observed in certain cases, and the absence of such symptoms in those cases of long standing anchylostomiasis in which there are only *old* worms, which have become more or less permanently fixed and do not locomote. This ingenious explanation is only available for the rare cases in which a large number of larvæ have been swallowed at once or at short intervals, so that many worms become sexually mature simultaneously. But as the infection is effected in ordinary cases by the ingestion of a small number of larvæ at a time, the occurrence of distinct periods of contemporaneous lively movement of many worms together, bent on copulation, cannot be invoked to explain the periodical exacerbations of symptoms. The disease more generally runs a chronic course; and when the patient has abundance of nourishment, this state may last for years, the patient continuing more or less pallid, sickly, and miserable, till an acute affection, as pneumonia, or some chronic ailment, like tuberculosis, malarial cachexia, beriberi, carcinoma, supervenes and causes death. In other cases in which the disease remains uncom-

plicated, but in which new infections do not permit a spontaneous cure by the natural death of the parasites, the patient lingers many years, until the fatty degeneration of the heart reaches such a stage as to give rise to failure of the heart's action. From this arises a more pronounced dropsical state, and then death follows, either from œdema of important organs, as brain or lungs, or directly by syncope from exhaustion.

Giles, speaking of one of the rare cases in which death occurred from uncomplicated anchylostomiasis, says that "the patient died in the most typically asthenic way it has been my lot to witness, the temperature falling, and the lungs and heart, hampered by dropsical effusions, acting more and more weakly, until the functions of life came to a stop; much in the same way that a machine is brought to a standstill by the dying out of the fire used to originate its energy."

Post-mortem Examination and Pathological Anatomy.—It is not difficult to detect *anchylostoma* in the intestine of the dead body; but in order to find it, as was said by Dubini, we must *look* for it. In fact, when the necropsy is made many hours after death, anchylostomes assume a grey colour, and being hidden by mucus easily pass unobserved, if they are not searched for. Moreover, the usual manner of washing the intestine under a water-pipe, or in a basin with great quantities of water, which is thrown away without examination, certainly retarded the discovery of the parasite.

A better system for the discovery of the parasite is the following:—Begin by removing the small intestine by separating it along the mesenteric attachment, in order to allow its coils to be spread out; then open it in successive tracts (of about 50 cm.), spreading out each tract upon one of the thighs of the corpse. By this process, taking account of the tracts opened, we may locate precisely all the points where worms are found. Leichtenstern[1] uses this method to indicate the number of anchylostomes found in each successive metre of intestine. Water is then poured by a small siphon to remove gently the mucus from the surface of the mucous lining, care being taken to raise with a spatula one by one the *valvulæ conniventes*, in order to discover the anchylostomes that may be hidden under them.

In many warm countries, such as Egypt, India, Brazil, anchylostomes are met with very frequently in post-mortem examination. Bilharz said that he scarcely made a necropsy in Cairo without

[1] "Eineges über Anchylostoma duodenale," in *Deutsch. med. Wochenschrift*, 1887, Nos. 26–32.

finding this parasite. I found it in no fewer than in sixteen out of nineteen necropsies made in Cairo, at the Kasr-el-ain Hospital, in the year 1877. In the majority of the cases the number of entozoa was small, and there were no signs of any disturbance produced by them. Even when severe symptoms of anchylostomiasis preceded death, the fatal termination of the case may have been caused by some complicating disease.

In the small number of cases of genuine and uncomplicated anchylostomiasis ending in death, in which a necropsy has been made and an exact record kept, the alterations found have been very simple. The body generally does not appear much emaciated, but rather plump, and the connective tissue, especially that of the wall of the abdomen and of the omentum, is far from being deficient in fat. The high degree of anæmia is shown by the pallor of the muscles, meninges, brain, lungs, and other organs. This anæmia is more noticeable on opening the abdomen from its contrasting with the ecchymoses scattered over the mucous lining of the jejunum, and which are often to be recognised through the walls of the gut.

The worms, which in rare cases may reach the number of two or three thousand, are generally found in the upper part of the small intestine, viz. the duodenum and jejunum. More than once I have found many anchylostomes in the jejunum without finding one in the duodenum. This is in accord with the results of many other observers. In early necropsies a great number of anchylostomes are found still attached to the mucous lining, and still living, and with their intestine red from containing unchanged blood. But whenever the necropsies are made many hours after death, the entozoa are generally found detached; being of a whitish colour, they may pass unobserved. Anchylostomes have been found exceptionally, too, in the stomach (Parona), very rarely in the ileum, although sometimes they have been found below its middle (Roth and Baümler), and even in the cæcum (Riou Kérangal).

Small ecchymoses, several millimetres in diameter, sometimes raised and constituting nodules, having a very small central aperture, corresponding to the insertion of the parasite, may be met with. The number of ecchymoses is not always in proportion to the number of *anchylostoma*. In a few cases anchylostomes are found to penetrate more or less deeply into the walls of the intestine, or they may be found entirely concealed under the mucous lining, in a cavity full of extravasated blood that forms a thickening or swelling of the size of a filbert or larger. These swellings always display, as we have said, the aperture of entry of the worm on the mucous surface. Sometimes two worms are concealed entirely in

one of these swellings; and, as we said before, Grassi once found in these swellings very young specimens of the worm that did not exceed 4 mm.

Besides the ecchymoses and swellings, there is often seen a punctiform pigmentation, which must be regarded as a residuum of old hæmorrhages. At other times, as a result of old standing catarrh, we meet with a thinning or thickening of the wall of the first part of the intestine. Sometimes very small vesicles with transparent fluid are met with, indicative of a still present intestinal catarrh.

The surface of the intestine is smeared with a certain quantity of mucus more or less tinged by blood. In certain cases, blood in quantity has been found in the intestine. Griesinger relates a case in which, at the necropsy, he found the duodenum, jejunum, and the first part of the ileum full of recently extravasated blood, with anchylostomes in thousands. In this case death was caused by the sudden hæmorrhage. And it must be noted that it was just this case that struck Griesinger so forcibly as to suggest to him the pathogenetic connection between the loss of blood due to the parasite and the characteristic anæmia. Similar cases, though rare, have been recorded by others (Wucherer).

The same alterations which we have described as occurring in the intestine, may sometimes be found in the stomach, produced probably by the direct action of the worms. But the ectasia or dilatation of the stomach, which is spoken of by some authors, is rather to be regarded as a complication independent of the presence of *anchylostoma*, as we have said elsewhere.

Besides signs of anæmia and intestinal alterations, even in cases of uncomplicated anchylostomiasis, the heart is found flabby, relaxed, and thin, with a pale and yellowish coloration that indicates not only its anæmia, but also a fatty degeneration of its walls. Ante-mortem clots in the right side, extending into the pulmonary artery, are noted in two necropsies performed by Giles[1] in cases of simple anchylostomiasis.

Some degree of fatty degeneration may be observed, too, in other viscera, as the liver and kidneys; and, finally, it is common to find serous effusions in some of the serous cavities, or in the tissues of the brain and lungs.

I am inclined to regard certain other alterations, found in some cases only, as being independent of, or not directly connected with, the associated anchylostomiasis: as those in Peyer's patches, enlargement of mesenteric glands, amyloid degeneration of the duodenal villi

[1] See Giles, *loc. cit.*, Cases 31 and 32.

—noted in a case complicating *phthisis pulmonum* (Baümler), and enlargement of the pancreas. In the same manner, it seems probable that it is only to casual association that are due atrophy of spleen, and cirrhosis of liver; the latter I noticed in one of my patients who died some months after being freed from anchylostomes. The enlargement of the spleen, noted as a very common occurrence in Assam, is evidently due to malarial cachexia. Adhesion of the folds of the small intestines by means of a gelatinous exudation (Wucherer), when not due to tuberculosis, is possibly the effect of an inflammatory process originating in the mucous lining, and caused by the presence of parasites. In some instances it might be the result of a peritonitis circumscripta, such as was observed during life by Lutz in some cases of anchylostomiasis. Dilatation, or hypertrophy of the heart, and even valvular insufficiency, have been noted too frequently in cases of anchylostomiasis, by Lutz and others, to admit of their being regarded as simply accidental; though really it is not easy to explain the connection that may exist between those heart affections and the anæmia from *anchylostoma*.

It remains for further post-mortem observation to determine whether amyloid degeneration and atheroma may or may not be a consequence of the anæmia from *anchylostoma*, just as fatty degeneration is.

What has, up to the present, been well ascertained may be recapitulated in the following conclusions:—Post-mortem alterations due to simple anchylostomiasis are—1st, ecchymosis in and under the mucous lining, with effusion of blood into the lumen of the small intestine, and changes of the lining membrane due to chronic catarrh; 2nd, general anæmia; 3rd, fatty degeneration of the heart and other viscera; 4th, serous effusions and œdema, principally from cardiac insufficiency.

Pathogenesis and nature of Anchylostomiasis, with some considerations about Tropical Anæmia.—If, at the present time, there is no pathologist who would totally deny the pernicious action of *anchylostoma* upon the system, still we must acknowledge that there is not yet a general accord regarding the manner in which this pernicious action is brought about. The first opinion expressed by Griesinger, namely, that *anchylostoma* produces anæmia by the direct abstraction of blood from the system, is not shared by all observers, and cannot, perhaps, be accepted as the sole cause of the anæmia and other symptoms. No less than three different modes of action are now put forward to explain the supervening anæmia—1st, direct loss of blood; 2nd, catarrh of the stomach and intestine, causing altered digestion and impeded

absorption; 3rd, blood poisoning, produced by materials originating in the metabolism of the parasite, or in the impaired digestion of the bearer.[1]

Against Griesinger's opinion, sustained recently even by Leichtenstern, it is urged that the anchylostome is too small to cause important hæmorrhages. But it may be replied that although this may be true when only a few parasites are present, it is otherwise when there are hundreds or thousands of them. Granting that each may cause the loss of only a few drops of blood in twenty-four hours, the loss is a daily loss, which, continued for a long time, cannot fail to produce important effects. Moreover, although the anchylostome may be very small, it seems that it consumes more blood than might be anticipated from its size, as it utilises only the plasma of the blood, ejecting the corpuscles unaltered. Add to this, that there is the loss of blood from many points left bleeding after detachment of the worms, and which sometimes amounts to a great quantity. Indeed, any one who has ever seen dead bodies infested by hundreds of worms attached to the duodenum and jejunum, with cavities full of blood containing *anchylostoma*, and with many points still bleeding, cannot doubt that this parasite is capable of inducing a pernicious effect by acting as a blood consumer. Yet the part played by catarrh cannot be undervalued; the symptoms of the disease point often to this condition, and in the fæces of patients the remains of undigested food are abundant, and indicative of impaired digestion, the result of this catarrh. Moreover, the mucous membrane of the duodenum, and part of the jejunum where the parasites are met with, is covered with a tough mucous secretion; in addition to which there are greyish points, the remains of hæmorrhages, in the mucosa of those who have suffered from long-standing anchylostomiasis.

As for the hypothesis of the origin of the constitutional symptoms of anchylostomiasis from blood poisoning, it seems to us that fresh researches are required to put the evidence on this point in a clearer light; and, even admitting that blood poisoning exists, it would still remain doubtful whether its origin is to be found in the parasite or in the organism of the bearer.

We may thus conclude that *anchylostoma*, when numerous, have a pernicious effect upon the system, and that probably their pernicious influence is not simply limited to the loss of blood they occasion, as was at first believed, but depends also on the intestinal catarrh to which they give rise, and perhaps on the action of toxic materials

[1] See Lussana's "Contributo alla patogenesi dell' anemia da anchilostomiasi," in *Archivio italiano di clinica medica*, 1890, p. 759.—EDITOR.

which they elaborate. The relative importance of each of these causal elements remains to be determined by future observations.

No doubt this disease, so well indicated at the present day by the term *anchylostomiasis*, has been confounded before the time of Griesinger's discovery under other names, as those of African or negro cachexy, and their synonyms hypohæmia, mal d'estomac, oppilaçao, or under the more general term of tropical anæmia.. In this manner Hirsch, in his *Handbook of Geographical and Historical Pathology*, under the same heading, speaks of the African cachexy and of anchylostomiasis as identical diseases. Yet the African or negro cachexy has been often a more complex complaint, in which anæmia from *anchylostoma* represented only one element. In fact, the descriptions given of its symptoms and its anatomico-pathological changes [1] point to morbid conditions not always identical. And in its etiology we find playing in different degrees, poor diet, excessive and enforced labour, want of sufficient clothing, exposure to damp and great heat, and the depressing moral influences always acting with more or less intensity in the state of slavery, independently of or together with the presence of *anchylostoma*. The same thing may be said, according to Bozzolo and Pagliani,[2] of the St. Gothard tunnel epidemic of 1880, in which we cannot recognise in every case a simple anchylostomiasis, many cases of anæmia and cachexy having been observed in which *anchylostoma* was present only in small numbers. Indeed, the anæmia of miners is the result of a number of causes which act quite independently of *anchylostoma;* such as excessive heat, defective ventilation, air vitiated from excess of carbonic acid, and the presence in it of other deleterious gases developed from dynamite, or of metallic emanations and atmospheric dust.

In the chapter on beriberi it is pointed out that this disease is now established as an entity which has nothing to do with anchylostomiasis. We will add only here that the researches of Kynsey and Macdonald in Ceylon, and Giles in Assam, show clearly that before anchylostomiasis was recognised in these countries, it was confounded with true beriberi or other diseases under the colloquial names of *beriberi* and *kála-ázar*. But this does not mean that true beriberi is caused by *anchylostoma*.[3]

Finally, I think proper to note that though, with the discovery

[1] See Copland's Dictionary of Practical Medicine, under the term "Cachexy, African."

[2] See L'anemia al traforo del Gottardo dal punto di vista igienico e clinico, Milano, 1880. (Reprint from the *Giornale della Societa italiana d'igiene*, anno ii. Nos. 3 and 4.)

[3] See Sonsino, "Del clima e delle malattie di Singapore, Comunicazione," etc. (in the *Giornale di idrologia e climatologia medica*, Firenze, anno viii. 1886, Nos. 7 and 8); and "Anemia perniciosa, beriberi e anchilostoma" (in *Rivista italiana di clinica medica*, Pisa, 1890, p. 191). See also Beriberi and Anchylostomiasis, editorial article in *Brit. Med. Journ.* July 2, 1892.

of *anchylostoma* and of the disease arising from this parasite, we have the explanation of a great number of cases that physicians, till those later days, used to refer to tropical anæmia, and though this latter in many other cases may be identified with malarial cachexy, there are yet cases in which the term "tropical anæmia" must retain its special signification, being really an anæmia that arises from the influence exclusively of heat, especially in newcomers from more temperate climates. There is no doubt, indeed, that Europeans who go to tropical countries generally sooner or later fall into a state of anæmia, which is solely the effect of the action of the heat upon the organism. This anæmia, arising from the direct action of a warm climate on newcomers, is no other than an exaggeration of the physiological anæmia that seems to affect generally the inhabitants of warm countries, and which is more pronounced in the female sex and in children. It is probable that heat concurs with other causes in lowering the quantity and quality of the blood, and we may assume that one of these concurrent elements is the rarefication of the air, which gives rise to a defective hæmatosis.[1]

Diagnosis.—The diagnosis of anchylostomiasis is not difficult, since we possess a pathognomonic sign of the disease in the presence of the eggs in the stools. Unpathognomonic symptoms alone can only exclude or lead to a suspicion of the infection; dependence upon them may also lead to unpleasant and dangerous mistakes. The microscopical examination of the fæces should thus never be neglected in any case of anæmia, before pronouncing that it is or is not due to *anchylostoma*. Without the examination of the fæces, anchylostomiasis may be confounded especially with chlorosis, pernicious progressive anæmia (essential anæmia), malarial or cancerous cachexy, cardiac affections, nephritis, as well as with beriberi and rhabdonemiasis. But even when the microscopic examination of the stools gives evidence of the presence of *anchylostoma*, there may be in some cases difficulty in deciding whether the anæmia is entirely due to the worm. In those cases we can clear our position only by procuring the expulsion of the worms by suitable treatment, and await the result. The suspicion of complicated anchylostomiasis arises especially when the eggs in the fæces are present only in small numbers, and when the anæmia is at the same time grave. Yet there are cases of simple anchylostomiasis of very long standing in which eggs appear only in small numbers, because the worms, previously numerous, have gradually been reduced to a small number by natural death. In this case it is the absence of all symptoms of

[1] The reader is referred for a fuller consideration of this subject to the article by Principal Birch, forming the first chapter of this work.—EDITOR.

other disease that will conduct to the diagnosis of simple long-standing anchylostomiasis. In these cases there are always effects upon the heart which render complete recovery difficult, even after the total expulsion of the parasites.

From beriberi the diagnosis is not difficult, because in anchylostomiasis there are anæmia and general weakness, and in advanced cases even serous effusions, but never paralytic symptoms, or other symptoms pointing to nervous functional disorders; whilst beriberi is essentially characterised by the symptoms of multiple peripheral neuritis, and not necessarily by anæmia. We must remember that cases of coexistence of the two diseases seem not to be rare in some Asiatic countries.

To distinguish simple anchylostomiasis from anchylostomiasis complicating malarial cachexy, we will attach importance to a history of previous attacks of fever, and to the characters offered by the conjunctiva which in malarial cachexy is nearly always of a dirty yellow tint, whilst in simple anchylostomiasis it has a white and rather bluish colour, accompanied often by puffiness of the eyelids. Ascites preceding œdema of the lower extremities is indicative of malarial cachexia, or of mechanical obstruction to the portal circulation, whilst in anchylostomiasis œdema usually precedes the ascites (Giles). Enlargement of the spleen and of the liver also point to malarial cachexy, or certainly to some complication; and when in such circumstances after expulsion of the parasites we fail to obtain the recovery of our patient, a complication with malarial cachexy, when in malarial countries, is indicated.

To exclude cardiac affections and the cancerous cachexy, or nephritis, we must rely on the absence of the objective symptoms that indicate those diseases.

As for chlorosis, we will suspect this complication when together with amenorrhœa the disorder arises just at the age of puberty, whilst anæmia dating from an earlier age points especially to simple anchylostomiasis.

The complication of true pernicious progressive anæmia is not common in the tropics; but if it should happen to complicate anchylostomiasis, it would be very difficult to diagnose, and in the greater number of cases would be suspected only by the continued persistence of anæmia after complete expulsion of the worms. It would obviously be easier to recognise leucocythæmia and Hodgkin's disease as complications of anchylostomiasis.

Prognosis.—Anchylostomiasis is a grave disease, not only because it may prove fatal when left to itself, but also because it lowers the strength to such a degree as to render the sufferer unfit for work.

When it is largely prevalent, the entire community may feel the evil effects of many persons being disabled. In such circumstances the endemic disease may acquire the importance of a pestilence, as has been the case in these latter years in Assam, where it passes by the name *kála-ázar*. Yet the disease is not frequently fatal even when left to itself. Exception must be made for the very acute cases in which the simultaneous infection by an extraordinarily large number of worms occurs, and in which death may follow if a speedy diagnosis does not suggest timely and suitable treatment. But even in the severest acute cases, if treatment is duly applied, recovery is to be looked for. The greater number of cases are chronic, the worms being introduced gradually and in small numbers at a time, the patient lapsing slowly into a debilitated condition, the strength of the heart's action becoming impaired little by little, so that the organism can adapt itself more or less to the abnormal changes which are so slowly produced. Death at last frequently ensues from some supervening acute disease, or from other complicating chronic ailment, rather than from the anchylostomiasis itself.

It is clear that anchylostomiasis, being a disease of long duration, runs the chance of being complicated by other diseases even more fatal. Moreover, the weakness into which the patient suffering from anchylostomiasis falls, and, above all, the cardiac degeneration that results from it and the impaired power of digestion, facilitate a fatal issue from complicating diseases which otherwise could be recovered from. In this manner the great liability of individuals infected by *anchylostoma* to become the victims of complicating diseases, constitutes in itself an additional element in the gravity of the affection. Even simple anchylostomiasis left to itself, by long standing may at last conduct to such alterations of the heart, and to such lowering of the organism, as eventually to cause death without the co-operation of any complication.

In mild cases, viz. those in which the number of worms is few, or when the disease is only beginning, we easily obtain a cure by proper treatment; and thus in no disease is it more necessary than in anchylostomiasis to recognise the condition in its early stage, and to adopt immediate treatment. To ensure complete recovery it is not necessary that patients be removed from the place where they contracted the disease, provided they are duly warned of the conditions they must observe to avoid reinfection.

The younger the patient the more certain and steady will be the recovery, not only because the blood restoring powers are more effective in young subjects, but also because the complicating diseases so frequent in old age are seldom met with in youth.

Prognosis must be reserved, above all, in cases of old standing, in which the digestive powers may be destroyed by atrophy of the digestive glands, or the heart muscle may have suffered in its structure, in which cases, even after the expulsion of the parasites, we may expect little or no improvement. When insufficiency of the heart causes œdema in important organs, the case is hopeless.

Probably the disease is more dangerous, and runs a more rapid course, in warm climates, not because the worms there act upon the human organism differently than in temperate countries, as has been erroneously supposed by some, but simply because in warm countries there is a greater tendency to fall into a state of anæmia from other causes, and because all lowering influences are less tolerated in warm than in bracing cold climates. Add to this that the infection by a very large number of worms occurs more frequently in warm countries, since the development of the larvæ is accomplished in them, and with great rapidity, all the year round.

Treatment.—The treatment of anchylostomiasis consists—1st, in procuring the expulsion of the worms; 2nd, in repairing the effects of their presence upon the organism.

Anchylostomiasis is never cured spontaneously, except by the natural death of the parasite. Living anchylostomes are never, or very rarely, expelled independently of a treatment that enfeebles if it does not kill them.

For a long time a remedy for the expulsion of this worm was a desideratum. Calomel with jalap and santonine effected the expulsion only of a small proportion of the worms, and this not always. The juice of *Ficus dolearia*, and a preparation of it called *dolcarina*, seems to have succeeded to a certain degree in Brazil. But no remedies have been so successful as *extractum filicis liquidum* and *thymol* have proved to be within the last dozen years. The latter is generally preferred. Ext. filicis maris of good quality is difficult to procure, and if not properly prepared often does not succeed at all. Moreover, it is an irritant poison, and in the doses in which it was used—20 and 30 grammes per diem—for obtaining its full effects, it sometimes produced dangerous and toxic effects, such as convulsions and albuminuria. For this reason it has been suggested recently by Eich of Köln that the dose of 10 grammes should never be exceeded even in the adult. Thymol being a definite chemical substance, the dose can be better regulated, and it is of more certain action. It seems that it gives trouble only when it gets absorbed; but being insoluble in water, it is not readily absorbed, or only in very small quantity. It is necessary therefore to administer it in such a way as to avoid absorption,

and for this purpose alcohols of any kind should be interdicted during its administration and action. The best manner of administering thymol is in powder in wafers (or *cachets*), as it is important to avoid direct contact of thymol with the mouth and pharynx.

The dose of thymol in adults may generally reach 6 or 8 grammes (say 90 to 120 grains) without inconvenience; and this dose often suffices when the number of worms is not enormous. With 8 grammes I obtained the expulsion of 383 *anchylostoma*. The best method of administration is the following:—The previous day give a purgative of calomel, and then, if necessary, clear the large intestine completely by the aid of an enema, and keep the patient on a light and liquid diet, composed principally of milk, eggs, and *bouillons*. Early on the following morning administer three or four powders of 2 grammes each of thymol, at intervals of one and a half or two hours. Keep the patient at rest in bed, and allow him between the powders to drink only water, or aromatics, and still restrict him to a light and liquid diet as long as the bowels have not acted. If thymol by itself does not determine some stools, it is better to procure them with an aperient within the first twelve hours of the last dose of thymol, with the view, not only of determining the expulsion of worms, but of clearing the intestines of thymol.

Thymol gives rise generally to a harmless sense of burning at the pit of the stomach. Small pieces of ice, or simply cold water, is the only thing required to meet this. Vomiting occurred in my practice sometimes after a fourth powder was taken. Vertigo, ebriety, as well as raving, are signs that absorption has taken place. Further evidence of absorption is afforded by the urine, which becomes brownish. Delirium was witnessed by me only in one case in which the dose of thymol had been raised to 10 grammes. When unpleasant symptoms arise before the full dose has been administered, the drug must be discontinued.

In certain cases of great weakness or of complicating diseases, it may not be prudent to administer the full dose of thymol in one day; in that case the remedy may be tried in smaller doses for several days, which is a less efficacious mode of administration. Fenoglio combines thymol and ext. filicis liq. in smaller doses; but this latter should be used only if it is certainly known to be of good quality.

If, after eight or ten days, the expulsion of the worms is not complete, which has to be ascertained by microscopical examination of the stools, it is necessary to have recourse to a second or third administration of the remedy.

Until lately we have relied solely on the total absence of eggs in the stools to establish the fact of complete expulsion of the worms. Absence of eggs, however, only indicates with certainty absence of mature females, but not absence of males and immature females. It has been remarked that generally males are expelled with more difficulty than females, and that they often persist in great numbers after the first expulsion. Recently Leichtenstern gives an additional indication of the persistence of worms, viz. the continued presence of Charcot's octahedral crystals in the stools. Should this fact be confirmed, then we must continue to give the anthelmintic so long as these crystals persist, unless they can be referred to the presence of other intestinal worms.

After the first expulsion of worms, we must act with the view of repairing the effects left by them on the organism. Good nourishment, when procurable, will act as the best blood restorer. To this it is generally convenient to add some preparation of iron, such as the sulphate, or the liq. ferri perchl., which last may contribute to heal the wounds left in the lining of the small intestine. To the liq. ferri perchl. may be added, with advantage in some cases, sodium bicarb., or the tinct. ferri perchlor. may be substituted. I use generally with great benefit small doses of a salt of iron with some quinine, and sometimes also the ethereal tincture of the perchloride of iron. In severe cases the cardiac disorders, and especially cardiac failure with dropsy, must not be overlooked; and very often it is necessary to have recourse to digitalis, tincture of strophanthus, or other cardiac tonics, which must not be used empirically, but in relation to the precise condition of the heart. The state of the bowels must also be attended to and suitably treated.

When anchylostomiasis is not complicated with other ailments, recovery is complete, and is obtained often in a few weeks after expulsion of the worms. This happens especially in the young. In the old, complete recovery is more uncertain. Cases of very long standing, and complicated cases, are more difficult to deal with, and demand treatment in hospital, or under the personal supervision of the physician.

There is no doubt that in some cases recovery is not permanent, and after some time signs of anæmia reappear, and this without the patient having been again subjected to a fresh infection. It seems that the remedy does not expel the very young worms, and when these become adult they give rise to anæmia again. Leichtenstern gives another plausible explanation of the reappearing of the eggs some days after the apparent total expulsion of the worms. According to him, the remedy does not effect in these

cases the expulsion of all the adult worms. The females left behind sicken under the action of the remedy, and the function of ovulation is suspended, hence the temporary disappearance of the eggs from the stools. It is thus necessary that an examination of the fæces be made after some weeks to assure us that all is right. Before dismissing the patient it is the duty of the physician to instruct him as to the manner in which infection takes place, and to give him directions how to prevent further introduction of the parasites.

Prevention.—Prevention demands attention to rules applying both to the individual and to the community. As we have seen, *anchylostoma* is generally conveyed by the hands soiled with earth containing larvæ at a stage of development suitable for commencing a parasitic life. It is possible, too, that the worm be taken in with drinking water which has been polluted with mud containing larvæ. There are some observers (Schopf) who think that larvæ may even be introduced with the air.

The principal personal rules are—1st, hands and nails to be thoroughly cleansed before eating, whenever a person, from profession or pleasure, has been handling mud; 2nd, drink filtered or boiled water only.

But in view of rooting out the disease where it is endemic, it is the duty of the State to provide against the spread of the infection wherever there is a gathering of people engaged in any work that necessitates the handling of mud or earth, especially in coffee, tea, and rice plantations, or brickworks, mines, tunnels, or excavation works. This requires the installation of latrines in those places, and the absolute prohibition of depositing the stools elsewhere. The system of latrines naturally should be adapted to the circumstances and habits of the country and people. But even the very simple system of trench latrines suggested by Giles for the Assamese villages, will certainly suffice for every other country and place where a more refined system cannot be obtained. In any place, and without great labour and expense, a trench several feet deep may be dug in the ground for the purpose of a latrine. A little earth should be daily thrown into it to cover the excreta, and when it is filled to about a foot from the surface it should be entirely closed and covered, and a fresh trench dug.

The general providing of latrines, not only in towns, but also throughout the country along the roads, even far from dwelling-places, is a measure of great importance in preventing contamination of the soil with fresh fæcal matters.

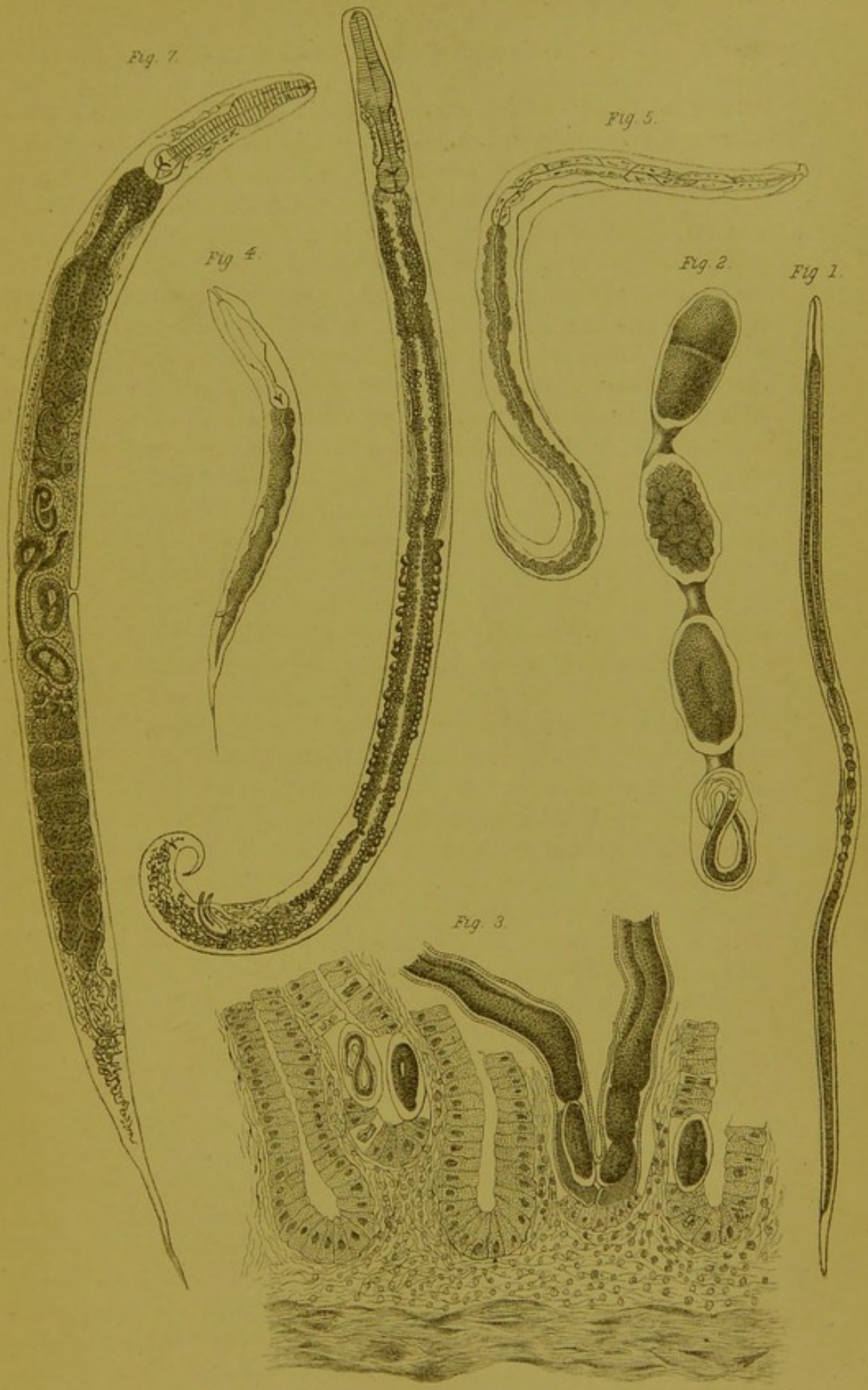
With the general adoption of any system of latrines the future spread of the disease can be certainly prevented. But, according to

Giles, a soil that has been once polluted preserves for a long time the power of infection, as there is an indefinite reproduction of new generations of embryos so long as the free worms find nourishment, viz. fæcal matter in the soil; and even when this is exhausted, the larvæ, although no longer advancing in their development, can nevertheless withstand the want of nourishment, and remain in the undifferentiated non-sexual stage for a very long time without perishing, certainly for not less than six months. The deprivation of a free supply of nourishment, while arresting for the time the development of the larvæ, appears greatly to prolong their lives instead of shortening them, and as soon as new fæcal matter is supplied, they are capable of starting afresh. It is thus important to have recourse in some cases to measures capable of killing the free worms and disinfecting the ground. These measures, according to Giles, should be the following: —1st, The prolonged exposure to the direct rays of the sun of the infected soil; 2nd, the exposure of the infected soil to a temperature exceeding 140° F., which may be obtained by burning on it dry grass and other vegetable refuse; 3rd, the turning of the surface layers of the soil under by ploughing, as the larvæ are thus buried and then killed for want of oxygen. According to the circumstances of the country and the purposes for which the ground is used, recourse may be had to one or other of these processes, or successively to two or to all of them.

Though Giles has not tested the efficacy of these measures, yet they appear likely to be of use, and they deserve certainly to be tried, whatever the true life history of *anchylostoma* may in the future prove to be.

Wherever there is a probability of infection from the eggs of *anchylostoma*, as in hospitals, asylums, barracks, etc., the stools should be disinfected. This disinfection may be effected by means of a 10 per cent. solution of sulphuric acid, or of a 2 per cent. solution of bichloride of mercury to equivalent volumes of the fæces, whenever the baking or boiling of the fæces, which is the surest measure, is not applicable.

To contribute to root out the disease in those tropical countries in which anchylostomiasis is endemic, the institution of special itinerating medical officers, trained in the diagnosis and treatment of the disease, has been suggested by Dr. Macdonald of Colombo, following the system adopted in Brazil. These should visit every station, and inquire into cases of anchylostomiasis requiring treatment. Giles urges strongly the adoption of systematic periodical medical inspection of all labourers employed upon tea estates, combined with the prompt



RHABDONEMA INTESTINALE

treatment of all cases that may be detected; and, in addition to this, he recommends the inspection of all newly-arrived hands, so that they may, if necessary, be treated before being allowed to mix with the others.

We would also advise that the precepts of prevention should be formulated like a catechism, suited to the understanding of the people, and that these instructions should be diffused by means of the medical officers among every class of workers of the soil.

By the rigorous application of the measures indicated, and, above all, by the full observance of a general system of conservancy, there is no doubt that anchylostomiasis could be rooted out, being, among preventible diseases, one of the easiest to prevent, and that with very simple means.

Rhabdonemiasis.

Anguillula intestinalis, now more generally called *Rhabdonema intestinale* (or *R. strongyloides*, Leuckart), was first discovered in soldiers coming from Cochin-China by Normand, and was considered by him to be the cause of a special form of diarrhœa endemic in that country. It has since been found in many other warm countries—the West Indies, Brazil, Egypt, and lately in Ceylon. It is known even in temperate climates, as in some parts of Europe (Italy, Germany, etc.); and everywhere it is often met with associated with *anchylostoma*. Respecting Brazil, Lutz observed *rhabdonema* in half the cases of anchylostomiasis. We may assume that the geographical distribution of the two parasites is almost the same, so that where the one is found we may infer the presence of the other. On the first discovery of *anguillula* in fæcal matters, it was thought that there were two different worms, which were distinguished under the names of *Anguillula stercoralis*, and *A. intestinalis*. But now, following Leuckart, Grassi, and Golgi, it is admitted generally that the two forms belong to only one species of worm, of which *A. stercoralis* is the free form or stage of the other, which is the only one that is parasitic in man,—an alternation of generations usually, *though not always or as a necessary feature in the life history of the worm*, taking place. The so-called *A. stercoralis* has indeed never been found within the human body; it develops only in the stools.

The adult parasitic form, which is only known under female features (Plate VI. Fig. 1), is about 2 mm. long, the breadth being about the 50th or 55th part of its length; it has thus a very slender body. Its oviduct contains a small number of eggs, five or six, ellipsoidal in shape, and measuring about 60 μ in length, and 30 to 34 μ in breadth (Plate VI. Fig. 2). The parasitic adult form has been seen only in dead bodies, upon the surface of the first

part of the small intestine (duodenum and jejunum), mixed with the mucus which smears the lining membrane. Golgi has found them even within the cavity of Lieberkühn's glands (Plate VI. Fig. 3), and I have seen also some of them embedded in the tissues of the walls of the intestine.

The embryo, which is already formed within the egg when expelled by the worm, hatches out very soon in the intestine; and in this manner the fæcal matters generally present only free embryos. These (Plate VI. Fig. 4) are between 0·210 and 0·300 mm. long, with a thickness of about 15 μ. Possessing a sharp-pointed tail, and being rhabditiform, viz. having a short œsophagus with two dilatations, the inferior of which possesses three chitinous processes, they resemble very closely the embryo of *anchylostoma*. There is, indeed, some difficulty in distinguishing them from the latter in the examination of the stools. According to Golgi, in the *rhabdonema* embryo the rudiment of the reproductive organs is more developed than it is in the embryo of *anchylostoma*. The diagnosis, however, between *anchylostoma* and *rhabdonema* is generally easy, because in the microscopical examination of stools when fresh, we find in the case of *anchylostoma* only eggs of the worm, and in the case of *rhabdonema* only embryos. And even if, in the latter case, there are some eggs along with the embryos, these, though resembling the eggs of *anchylostoma*, are seen to be a little longer and proportionally less broad; moreover, they are often united in chains of three or four, being included in a kind of tube (Plate VI. Fig. 2), whilst those of *anchylostoma* are always isolated. According to Golgi and Monti,[1] the eggs of *rhabdonema* are found in the stools only after the action of a powerful drastic. Riva,[2] however, says that he has seen a case in which the eggs were met with in stools passed without a purgative.

The rhabditiform embryos discharged with the fæces are condemned to die when the latter undergo putrefaction. Their development takes place only when there is no putrefaction, and the more liquid the medium is the better. A favourable condition for their development is the admixture of the fæcal matter with water. The embryos may assume two different forms of development, according to Grassi.[3] When the temperature is low (under

[1] "Sulla storia naturale e sul significato clinico-patologico delle cosi dette anguillule stercorali e intestinali." Osservazioni di C. Golgi e Achille Monti, in *Archivio per le scienze mediche*, vol. x. No. 3. Torino, 1886.

[2] "Sopra un caso di anguillulosi intestinale," in *Sperimentale*. Mem. orig. Fasc. 1°. Firenze, 1892.

[3] Grassi e Segré, "Nuove osservazioni sull' eterogonia del Rhabdonema (Anguillula) intestinale," in *Rendiconti dell' Accademia dei Lincei*. Seduta del 16 genn. 1887.

20° C.), they become more frequently filariform larvæ (with long œsophagus), capable, if directly reintroduced into the human organism, of growing into the adult parasitic form without alternation into the adult free form. They resemble in this respect the larva that arises from the embryo of the adult free form as represented in Plate VI. Fig. 5.[1] When the temperature is high (between 30° and 35° C.), more generally the embryo develops into the adult free form. This (called formerly *A. stercoralis*), consisting of males and females (Plate VI. Figs. 6 and 7), is shorter than the parasitic form, the female scarcely exceeding 1 mm., and the male being no more than 0·70 mm. They breed rhabditiform embryos like the parasitic form, and these embryos then grow into filariform larvæ (Plate VI. Fig. 5), which, if introduced into the human organism, develop into the adult parasitic form.

Rhabdonema is seen especially in those persons, as I have myself observed, who are accustomed to drink the foul water of tanks and canals polluted with fæcal matters. Yet, according to some French authors, in Cochin-China persons who have not used at all the water of the country have been equally subject to the infection; in this case it is supposed that the parasite has been taken in with vegetables, which fact may be in relation, they say, to the custom in eastern Asiatic countries of manuring vegetables with fæcal matters.

There is no unanimity of opinion respecting the pathogenetic importance of this parasite in regard to the diarrhœa of Cochin-China. It seems that *rhabdonema* is not found in every case of the endemic Cochin-China diarrhœa, and that it is never seen at the beginning of the disease.[2] For this reason Chastang and others who have practised in Cochin-China, came to the conclusion that the parasite is only a simple coincidence of the disease; or that its development in the intestine, instead of being the cause of the diarrhœa, is merely favoured by the abnormal condition of the intestine. It should then be regarded as a result of an abnormal state of the intestine, as *sarcina ventriculi* is of some abnormal conditions of the stomach.

In other countries there are some pathologists who even incline to regard the parasite as harmless. Golgi and Grassi, who made interesting observations about the life history of *rhabdonema* in Italy, entertain some doubts respecting its pathogenetic qualities, and are rather inclined to regard it as innocuous. Yet Golgi, having

[1] In my cultures of the embryos found in the stools of three cases of rhabdonemiasis observed in Pisa, I obtained always only the filariform larvæ, and never the adult free form. See "Tre casi di malattia da Rhabdonema o Rhabdonemiasi," in *Rivista gen. italiana di clinica medica*. Pisa, 1891.

[2] See de Santi, *loc. cit.* p. 13.

found in the dead body some alterations of the intestinal epithelium corresponding with the seat of the parasites, comes to the conclusion that he cannot exclude some morbid influence of the parasite on the epithelium. Lately Riva expressed the opinion that *rhabdonema* is injurious only by causing detachment of the intestinal epithelium, and in this way causing diarrhœa,—a pathological explanation that was once offered by the late illustrious anatomist Pacini to account for the symptoms of cholera. Yet it is more likely that *R. intestinale* acts upon our organism in a multiform manner, similar to that of *anchylostoma;* and that it probably becomes a serious matter only when a great number of individuals infest the intestine. The disease arising from *rhabdonema*, I proposed lately to call *Rhabdonemiasis*, following the rule adopted for the naming of other entozoal diseases.

As for treatment, we must acknowledge that our means are much less efficacious than for *anchylostoma;* perhaps the difficulty of getting rid of *rhabdonema* is in part due to its being often hidden in the walls of the intestine. Indeed, Golgi avows that filix mas as well as thymol, even in large and repeated doses, does not succeed at all. It seems that Seifert had the same negative results. In one case in which there was only anæmia without diarrhœa, and in which the presence of *anchylostoma* was positively excluded, I obtained complete recovery by persevering with small doses of thymol aided by liq. ferri perchl. Riva, relying upon experiments made on larvæ, recommends chloroform water with creosote.

Respecting prevention, it is necessary to avoid the use of foul drinking water, and in case of doubt to filter it. The relatively large dimensions of the larvæ of *rhabdonema* hinders their passage with water through a good filter; therefore proper filtration of drinking-water suffices. Care must also be taken not to eat fresh vegetables unless they are well cleaned or cooked. To hinder the spread of the larvæ, it is necessary to enforce a general system of conservancy, such as we urged for the prevention of *anchylostoma*.

Liver Parasites.

Liver entozoa may be found in the gall ducts, or in the parenchyma of the organ. *Bilharzia hæmatobia* may be considered rather as a hæmatozoon, as its abode is not limited to the veins of the liver. We will treat of it after having briefly noticed the true liver entozoa.

Among the parenchyma-entozoa we shall not discuss echinococcus, though it is often the cause of severe and dangerous complaints in

man, inasmuch as it is dealt with in all pathological works, and also because it is in no way peculiar to warm climates.

Pentastomum constrictum is the larval form of an *acarida*, of which the adult form is still unknown. It has a whitish, anellated, cylindrical body, with roundish anterior end, and blunted-conical posterior end. The ventral surface is flattened, the entire parasite measuring about 15 mm. in length by less than 3 mm. in breadth. It has four foot-claws near the mouth. The elongated abdomen displays twenty-three rings placed at tolerably regular intervals. It differs from the larval form of *P. tænioides* (*P. denticulatum*), found especially in Europe, in not possessing integumentary spines, and in being a much larger parasite. It is found coiled upon itself in a cyst, situated generally near the surface of the liver, in such a way as to be perceived through the fibrous capsule of the organ.

This entozoon was found at Cairo first by Pruner and afterwards by Bilharz, but only in the dead bodies of negroes. Thus it may be indigenous rather to the Soudan than to Egypt. Pruner also found it in the giraffe. It has been found too, as related by Aitken,[1] in a soldier who died at Bathurst (Gambia), and in an African slave coming from St. Helena. In one of the last two cases it was found encysted, not only in the liver, but also in the lungs, and was associated with signs of pneumonia and peritonitis. Bilharz found also cysts of this entozoon in the walls of the small intestine.

The clinical importance of this *pentastomum*, as that of its European congener, is lessened by the difficulty of diagnosing its presence during the life of the host and the still greater difficulty of treating it. We should rather aim to know the manner of its introduction into the organism of man, in the same way as the origin of *P. denticulatum* from *P. tænioides* is known, in order to give rules for its prevention.

Fasciola hepatica **and** ***Distomum lanceolatum.***—Among the entozoa of the gall ducts and gall bladder we should include both *F. hepatica* and *D. lanceolatum*, the two common flukes of sheep and other ruminants, though they are not frequent in man, and not specially confined to any warm country.

Of the small number of cases recorded in medical literature of infection in man by the common large liver fluke (*F. hepatica*), only one, so far as I know, refers to a warm country. It is a case relating to an individual who had lived three years at Java, and coming to Europe sick with jaundice and fever, after a long illness died at

[1] "On the occurrence of *Pentastomum constrictum* in the human body as a cause of painful disease and death," *Science and Practice of Medicine*, 4th ed. 1865.

Zurich, under Biermer, with signs of liver complaint. The necropsy revealed the presence of one specimen of the liver fluke with obliteration of the hepatic duct. Although *F. hepatica* has hitherto been found only in small numbers in man, it seems that, in some cases, it has been the cause of severe disorders, though not always mortal.

F. hepatica is ingested in eating certain vegetables, as water cresses, uncooked and not well washed, its larva being generally encysted upon grass. The diagnosis of its presence is made by discovering its eggs in the stools.

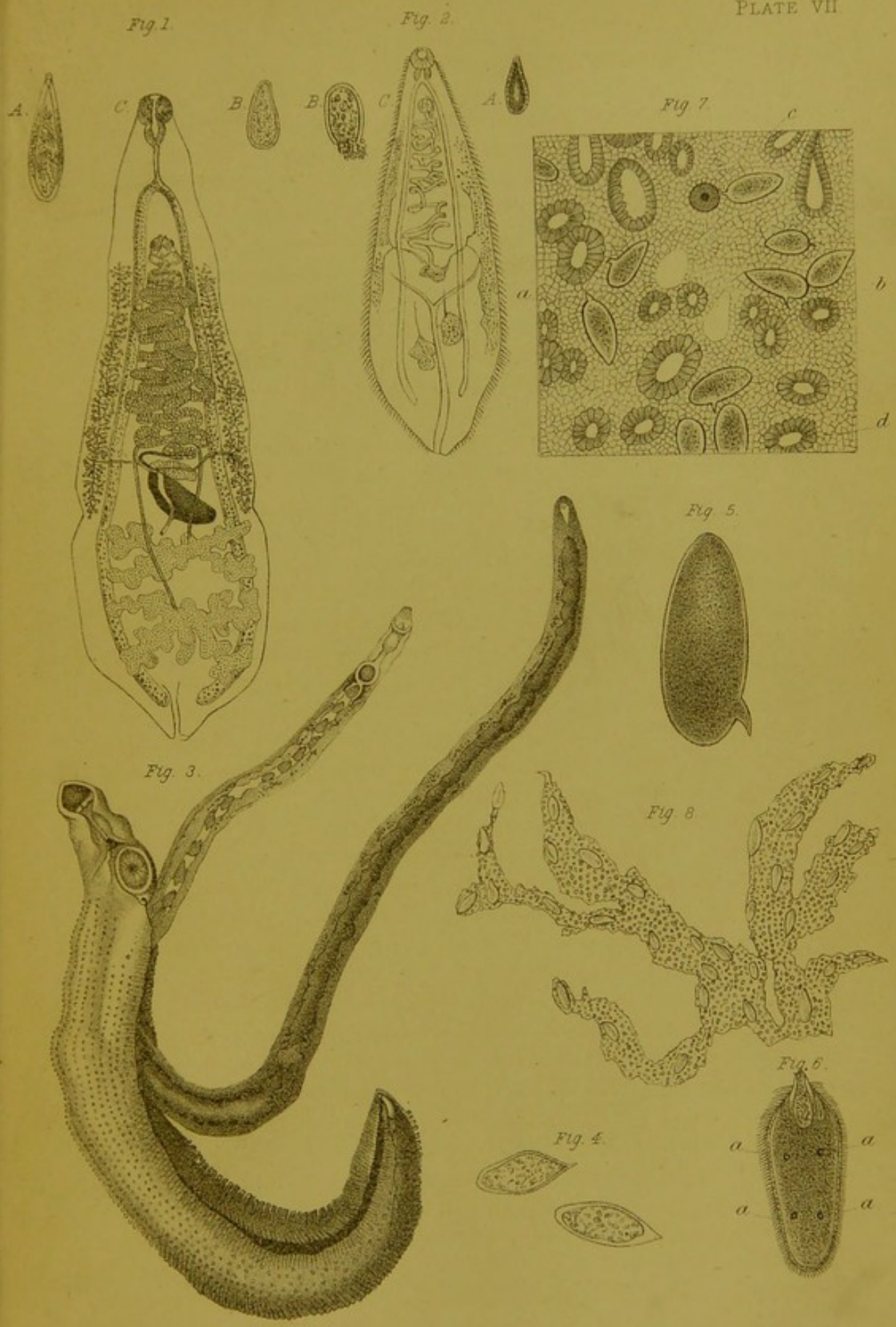
As for treatment, we may only suggest as deserving of trial the purgatives, with ext. filicis maris, which last is said to have been used with advantage against *F. hepatica* in sheep (Grassi).

D. lanceolatum, the smallest European common fluke, has been found in man in one instance associated with the preceding species. But of all the cases, only five in number, hitherto observed in man, not one has been met with in a warm country. It is acquired probably in the same manner as *F. hepatica*, viz. in eating unwashed vegetables. We have given the characteristics of its eggs in treating of diagnosis founded on fæcal examination.

About these two entozoa we have only to add, that it is very probable that future accurate researches, both in the dead body and in microscopical examinations of the stools of persons affected with liver complaints, will often lead to the discovery of these worms among peoples backward in civilisation, wherever these entozoa are found common parasites in their domestic animals.

***Distomum sinense* and *D. conjunctum*.**—There is another fluke inhabiting the gall ducts that is frequently found in man, especially in some parts of Japan, where it often causes a severe hepatitis. This is *D. sinense* (Cobbold), or *D. spathulatum* (Leuckart), discovered first by MacConnell at Calcutta at the necropsy of a Chinese, and, later, in Mauritius by MacGregor, also in a Chinese. In Japan this worm has been described by Baelz as two new distomes, under the double name of *D. hepatis perniciosum* and *D. hepatis innocuum;* but these two forms have been identified with *D. sinense* (Ijima). It seems that its geographical area, so far as is at present known, is confined to the far eastern countries, having been observed only in Corea, China, Japan, and Tonquin, or in individuals coming from those countries. In Japan it is particularly frequent in certain humid, low districts near the sea, especially in the provinces of Okayama and Katayama, where the people drink very foul water; there the worm is found perhaps in 20 per cent. of the population.

The presence of this parasite may probably be diagnosed by discovering its eggs in the fæcal matters. These eggs are brownish and



LIVER FLUKES AND BILHARZIA.

oval-shaped, with a double contour and an operculum. Their average length is $\frac{1}{833}''$ (MacConnell), or 30 μ, their length, roughly speaking, being twice their breadth. Their length is about a quarter less than that of *D. lanceolatum* (Plate VII. Fig. 1, *b*).

D. sinense has been seen to give rise to liver complaints, followed by fatal diarrhœa and marasmus. The necropsy reveals enlargement of the liver with diverticula of the gall ducts; the diverticula are as large as a filbert or nut, and contain the worm. The parasite measures 20 mm. in length (Plate VII. Fig. 1, *a*), and resembles in form *D. lanceolatum*, but its structure is different. In fact, instead of having the two testicles in the middle third of the body, and anteriorly to the ovary, as in *D. lanceolatum*, *D. sinense* has two ramified testicles at the posterior end of the body (Plate VII. Fig. 1, *c*).

Whether diarrhœa be a consequence of the liver complaints produced by the worm is not quite clear. The entire clinical characters of this parasitic disease are still obscure. *D. sinense* is likewise found in cats.

Another worm akin to *D. sinense*, but smaller, is *D. conjunctum* (Plate VII. Fig. 2, *a*), which has been found in Calcutta in natives by MacConnell, and in dogs by Lewis and Cunningham.

According to MacConnell, the eggs are oval; the shell has a double contour and is operculated, and has granular contents. Their average length is $\frac{1}{750}'' = 33$ μ, being a little larger than those of *D. sinense* (Plate VII. Fig. 2, *b*). *D. conjunctum*, as *D. sinense*, has the testicles posterior; but they are not ramified, and this fluke is further distinguished by having its integument covered with small spines (Plate VII. Fig. 2, *c*).

Both *D. conjunctum* and *D. sinense* should receive the attention of physicians practising in warm countries, and especially in Asia, where very probably these worms are more diffused than is at present generally supposed, and play perhaps an important part in the liver complaints of those countries. Up to the present we know too little of their clinical importance, and nothing whatever about the way in which they are introduced into man, which might enable us to give useful rules for their prevention. It is probable that they are introduced with some food peculiar to those eastern countries.

Bilharzia hæmatobia and Bilharzia Disease.

History and Geographical Distribution.—The knowledge of *Bilharzia hæmatobia* goes back only to 1851, in which year it was discovered by Bilharz in Cairo. The morbid disorders

caused by it in man were previously confounded with other diseases, and were described as symptoms of them. Prospero Alpino (died 1617), who wrote about medical subjects concerning Egypt,[1] had, however, remarked the great frequency of urinary calculi in Egypt. Renault,[2] a French military surgeon in Egypt at the end of last century, had noted that many soldiers were subject to hæmaturia. The discovery of Bilharzia gives the key to the parasitical origin of those pathological facts.

John Harley[3] in 1864 was the first to observe cases of Bilharzia disease in persons coming from the Cape; and thus it was shown that the endemic hæmaturia of the Cape was, as that of Egypt, the result of Bilharzia. At present, the existence of Bilharzia disease has been ascertained in many parts of South Africa, especially along the east coast watershed, as at Uitenhage and Port Elizabeth, Fort Beaufort, Alice, Graham's Town, King William's Town, and more to the north, at Durban and Pietermaritzburg in Natal. The existence of the parasite has not been demonstrated in the corresponding inland countries, as the Orange Free State and the Transvaal; but it is improbable that the range of the parasite is so limited in South-Eastern Africa as to be confined to the coast merely.

Eyles and Eden found Bilharzia in the Gold Coast.[4] More recently it has been found in individuals coming from South Tunisia (Villeneuve, Brault, and Cahier).[5]

As for other parts of Africa, I can say that the late Dr. Nachtigal assured me, by verbal communication made in 1875, that in his travels from Tripoli to Egypt through the countries of the Tibbos, Lake Chad, Darfoor, and Kordofan, he found endemic hæmaturia very prevalent there, though he could not verify by positive observation its connection with Bilharzia. Endemic hæmaturia is found also in the islands on the eastern coast of Africa, as Bourbon and Zanzibar;[6] and in Mauritius quite recently the hæmaturia there has been found by Chevreau to be caused by Bilharzia.[7]

[1] De Medicina Ægyptiorum. Libri quatuor. Venet. 1591.

[2] See Copland's *Medical Dictionary*, in the article "Hæmorrhage from the Urinary Organs."

[3] On the "Endemic Hæmaturia of the Cape of Good Hope," by John Harley, in the *Med. Chir. Trans.*, published by the Royal Medical Chirurgical Society of London, 2nd series, 29th vol. London, 1864.

[4] See "*Bilharzia hæmatobia* in West Africa," in *Lancet*, 1887, ii. p. 460.

[5] See Rathelot, "Contribution a l'étude de la Bilharzia hæmatobia," *Thèse de Paris*, 1892. Cahier, "La Bilharzia hæmatobia en Tunisie," *Archiv. de Méd. Milit.*, Février, 1893. I have myself lately met with some cases of Bilharzial hæmaturia in natives who have never left South Tunisia; but it is probable that Gafsa is the place most infected at present.

[6] See Castle, in *Lancet*, 1891, i. p. 931.

[7] Chevreau et de Chazal, Étude sur le Bilharzia hæmatobia de l'ile Maurice (extract from the *Bulletin de la Societé médicale de l'île Maurice*, 4 Juin 1890). Maurice, 1890.

The existence of hæmaturia is also indicated by Felkin[1] at various places on the White Nile between 6° N. and the Albert Nyanza. In this manner we have sufficient data for concluding that Bilharzia is spread all over the African continent. Very probably, too, it is now indigenous on the opposite Arabian shore of the Red Sea.

B. crassa, which in Egypt exists both in cattle and in sheep, has been found also once in India, by Bomford, in cattle, and was found by Grassi to be common in sheep in Catania (Sicily). But as for Bilharzia in man, its endemic prevalence is limited, so far as we at present know, almost to the African continent. Only lately (1888) the occurrence of two cases in England, referred to by the late Berkeley Hill, leads us to suspect that Bilharzia may be contracted even in countries with a temperate climate, such as England.[2]

Characters of the Worm and its Life History.—*Bilharzia hæmatobia*, Cobbold, or *Distomum hæmatobium*, Bilharz, is a trematode worm, in which the male and female reproductive organs occur in separate individuals. Both male and female possess two suckers and a digestive apparatus, terminating very near the hinder end of the body in a single blind intestine. The intestine is often found filled with blood globules, and thus we have evidence of the worm being hæmatophagus. The size and appearance of the male and of the female are quite different. The male is opal white, and measures not more than 15 mm. in length by 1 mm. or a little more in breadth. Its body assumes a cylindrical form, which is due to the lateral borders being bent inwards, constituting in this manner a true gynæcophoric canal, so called because it receives part of the body of the female as shown in Plate VII. Fig. 3. The male has no copulatory organ; the spermatic fluid, being poured into the gynæcophoric canal, is probably taken up by imbibition by the vagina of the female.

The female, measuring 20 mm., is thinner, and generally appears grey or brownish from the colour of the contents of the intestine. She lies in the gynæcophoric canal of the male, with her ends projecting.

The eggs, which can be distinguished even within the uterus, shining through its walls, appear in the urine (Plate VII. Fig. 4), as bright and translucent oval bodies, with a smooth surface and a thin non-operculated shell, possessing a spine situated ordinarily at one polar end, but sometimes laterally. They have a length of

[1] Felkin, On the Geographical Distribution of some Tropical Diseases, etc. Edinburgh, 1889.

[2] See "Clinical Lecture on Hæmaturia," in *Brit. Med. Journ.* 26th May 1888.

0·160 mm., and a breadth of 0·060. Their segmentation begins and may be completed in the uterus, and when discharged with the urine they often contain a well-formed embryo. The position of the spine of the egg, whether polar (Plate VII. Fig. 4), or lateral (Plate VII. Fig. 5), is due, according to Prof. G. Fritsch, of Berlin,[1] to the different situations which the posterior oviduct may occupy in respect to the shell gland, the former opening generally in the axis of the latter, but sometimes laterally to it. Cobbold[2] regards the spine as a species of holdfast, which is found more highly developed in the ova of certain other trematodes.

The embryo is ciliated (Plate VII. Fig. 6). When it hatches out from the egg in the urine it soon acquires a very rapid movement, often changing its form. Generally when in motion it presents a form between a cylinder and a cone. The anterior part ends in a kind of proboscis provided with a central opening, and presents a median granulated sac which communicates with the opening. This median sac seems to be the rudiment of a digestive organ, whilst one or two other sacs seen in certain positions at the side of the median one, according to Railliet,[3] represent two independent unicellular glands. In the rest of the body are seen Cobbold's sarcode globules, which were thought to be the beginning of a new stage in the life history of the parasite. Cobbold could also distinguish—and I also have seen this in the embryo—a distinct water vascular system, consisting of two main stems which, in the course of their windings, give off several anastomosing branches. Lately Railliet and Cahier have observed at two points of the posterior part of the body two ciliated infundibuli, resembling those described by Thomas in the embryo of *Fasciola hepatica*, and which belong to the origin of the excretory system. These two organs are best made out, according to my own observations, by slowly lowering the focus of the microscope.

The embryo hatched in the urine does not retain its vigour there, and often dies within a short time. Yet I once saw a living embryo in urine that had been passed for twenty-four hours. Eggs removed from the urine and placed in pure water hatch very soon, the embryo appearing more agile and vigorous than in the urine. Nevertheless even in pure water the latter die after a time, and up to the present it has been found impossible to discover its ulterior transformations.

[1] "Zur Anatomie der Bilharzia hæmatobia," in *Arch. für mikrosk. Anatomie*, Band xxxi.

[2] "On the Development of *Bilharzia hæmatobia*," etc., in *Brit. Med. Journ.*, 1872.

[3] Railliet's "Observations sur l'embryon du Gynæcophorus hæmatobius," in *Bulletin de la Société Zoologique*, 1892, tome xvii. p. 161.

It is known that the trematode embryo generally penetrates the body of some species of mollusc in order to be transformed into a nurse (*redia*), which produces in its interior a generation of larvæ (*cercariæ*). These, after escape from their intermediary host and subsequent encystment, pass in their turn into the body of the definitive host to become mature flukes. Up till now these different phases of development have not been discovered for Bilharzia, although both Cobbold and I have diligently searched for them.[1]

Although we know nothing of the transformation of the embryo after it has passed with the urine or fæcal matter into water, we can suppose that its reintroduction into the human organism is effected by means of drinking water, either as a free larval form, or included within the body of some small animal serving as intermediary host. This intermediary host instead of being a mollusc, may be a species of *Nais*, or similar animal, which from its transparency might pass unobserved in the drinking water.[2] The view that the reintroduction of the parasite may be made through the skin, urethra, or anus, whilst bathing, instead of by the way of the digestive organs, is hardly admissible, if we bear in mind that there is no known example of an entozoon that introduces itself into the human body by any of these ways, instead of passively by the mouth. Moreover, against such a supposition there are cases of persons who have had the worm without having ever bathed in rivers or canals.

Post-mortem Examination and Pathological Anatomy. —What we know about this subject is the fruit of researches made up till now exclusively in Egypt. Bilharz and Griesinger,[3] out of 363 necropsies made at the Kasr-el-ain Hospital in Cairo, found Bilharzia and its resulting lesions in 117. Out of 75 necropsies in indigenous subjects in Nubian, Soudanese, or Abyssinian, which I performed between 1875 and 1880 in the Kasr-el-ain Hospital, or other hospitals of Lower Egypt, I found it in 38. In 16 other necropsies made by myself, together with the late Dr. Gulliver of

[1] See Cobbold, *loc. cit.* in *Brit. Med. Journ.*, 1872; and see also my paper, "Ricerche sullo sviluppo della Bilharzia hæmatobia," Torino, 1884," reprint from *Giornale della R. Accademia di Medicina de Torino*, Fasc. 8, 1884.

[2] Dr. Sonsino, who is now prosecuting his studies of Bilharzia disease in Tunis, has notified to the *Lancet* (August 19, 1893) that the life history of this parasite "resembles that of the holostomes, the intermediary host belonging to the class of arthropoda." Should particulars of his observations reach me in time, they will be given in the appendix.—EDITOR.

[3] Griesinger, "Beobachtungen über die Krankheiten von Ægypten," *Arch. für physiol. Heilkund*, 1854, xiii. p. 561 (Distomum krankeit).—Bilharz, "Distomum hæmatobium und sein Verhältniss zu gevissen pathol. Veränderungen der mensch. Harnorgane," in *Wiener med. Wochenschrift*, 1856, Nos. 4 and 5.

St. Thomas' Hospital, in the year 1883, during a cholera epidemic, we found in no fewer than 4 cases alterations due to Bilharzia. Thus I have a total of 91 necropsies, of which 42 presented Bilharzial infection, a proportion even higher than that obtained by Bilharz and Griesinger.

Griesinger's statement that Bilharzia is found more frequently in necropsies made in June and August than in those made in October and January, is not in accordance with my results, which rather go to establish that the frequency of the occurrence of lesions from Bilharzia, as seen in necropsies, has no relation to season.

The adult worm is generally found in the interior of the portal vein, its branches and its roots, as the mesenteric and splenic veins. It has been found also in the small veins of the urinary bladder, and in the wall of the ureters (once by myself), and recently in the interior of the abdominal vena cava, by Colloridi, in Alexandria.[1] Its abode is not limited to the portal system, and it is probable that with further careful research adult *Bilharziæ* will be found in other veins of the body, and perhaps in every part of the vascular system.

The parasites are often found embedded in clots, and most frequently in those occurring at the point of division of the portal vein into its two hepatic branches. I once found in that situation more than forty specimens, and Kartulis,[2] in one case, counted 300 of them collected from different parts of the portal vein and its hepatic branches.

To insure finding the worms in the portal vein it is well, as I suggested so far back as 1875, to tie the portal vein with a double thread before cutting it, so as to prevent the escape of the blood. The search for the parasite is more difficult and tedious when it is practised in the small veins of the urinary bladder, or of the other organs.

The morbid alterations due to Bilharzia have a different aspect according to the number of parasites which have contributed to produce them, and the time the infection has lasted, or even according to the organ or tissue in which they have been set up. But whatever be the affected organ, be it the urinary bladder, the ureters, the seminal vesicles, the prostate, or the intestine, in any case the specific character of the alteration is revealed by

[1] See "La Bilharzia hæmatobia dell' uomo ed i fenomeni morbosi cagionati da essa," in *Giorn. internazionale delle scienze mediche.* Napoli, Novembre, 1891.

[2] "Ueber das Vorkommen der Eier des Distomum hæmatobium Bilharz in den Unterleibsorganen," reprint from Virchow's *Archiv für pathol. Anatomie und Physiologie*, etc., 1885, Band xcix.

the presence in the tissues of the eggs of the worm; and it is to the presence of these eggs that the morbid processes, of a more or less intense inflammatory character, are due. To these alterations, produced by the deposition of *Bilharzia* eggs in the tissues, I gave the Italian denomination of *infarcimento Bilharzico* (from *infarcire*, to stuff), which, I think, we may conveniently translate into English by the expression *Bilharzial infarction.*

The urinary bladder is the organ which is most frequently and most extensively affected with Bilharzial infarction. I have seen only a few cases in which the urinary bladder was apparently unaffected, the ureters and seminal vesicles alone revealing important alterations.

At the beginning of the infarction, we generally find in the urinary bladder only a certain degree of dilatation and fulness of the small blood vessels; but even at this stage a histological examination of the affected tissues will discover some isolated eggs of the *Bilharzia.* These are found sometimes even in the small clots taken from the dilated vessels, and in this case they are still fresh and with the vitellus in a state of segmentation, or the embryo formed. The first deposit of eggs in the mucosa of the urinary bladder is often revealed by the presence of some small, pellucid, or opaline eminences not exceeding the size of a millet seed, consisting of vesicles or papules, which, under the microscope, are found to contain clusters of eggs. This miliary appearance is very like that which is sometimes met with in the intestinal mucous membrane of persons dead from infectious diseases, such as acute intestinal catarrh and cholera, and is even like the *sudamina* of the skin, which is now also sometimes regarded as an expression of an infective process. So far as I know, this appearance passed unobserved by others until it was pointed out by the writer in 1875. In the more advanced cases we find nodules, or patches of variable dimensions—perhaps several centimetres in diameter. The more common seat of these is the inferior part of the wall of the bladder, comprising the vesical trigone; but they may be found in any part of the bladder, and even may sometimes occupy the greater part of its surface.

The patches, often roundish, project, being rendered more evident still by their abnormal yellow, grey, or brown tinge. They present a rough and granulated surface, and are of a leathery and more or less hard consistency, creaking under the knife as if filled with grains of sand.

These patches constitute the most characteristic and most frequent appearance of Bilharzial infarction of the bladder and

ureters. They are the expression of a sclerosis resulting from an inflammatory process accompanied by hyperplasia, which has taken place in the mucosa and submucosa, and are due to a deposit of eggs which, in the course of time, have undergone calcification. The induration is often increased by the deposit on its surface of eggs and saline matters derived from the urine. According both to Belleli and Kartulis,[1] the deposit of the eggs is generally more abundant in the submucous tissue than in the mucosa itself; and these authorities state that eggs are but seldom found in the muscular coat, especially at the point of union with the serous surface of the bladder. Leuckart[2] refers to a case observed by Griesinger in which even the serous membrane of the bladder, and the corresponding parietal layer of the peritoneum, showed pigmented excrescences of cockscomb form.

In the histological examination of the tissues the eggs sometimes appear as if they were circumscribed by a capsule of newly formed connective tissue, which seems to be the result of a healing hyperplastic process, and tends to isolate the clusters of eggs, as happens in the case of foreign bodies embedded in animal tissues.

The tissues of the mucous and submucous layers present also an infiltration of leucocytes, sometimes collected in masses so as to form small abscesses. In the same tissues, vessels obliterated by the eggs of the parasite are sometimes seen.

Besides the sclerotic patches, which sometimes exhibit superficial sloughs, the consequence of ischæmia from occlusion of the corresponding blood vessels, we find true new growths in the urinary bladder. These present themselves in different forms, as villosities, polypiform growths, condylomata or cockscomb like tumours. They also differ in consistency, presenting sometimes a smooth surface, more frequently a rough one, having the aspect of an arbutus-berry or of a strawberry. Sometimes they are abraded or ulcerated. These new growths generally have the histological characters of simple fibromata, or of papillomata. Carcinoma, even, is not infrequently found associated with Bilharzia disease, so that it has been maintained that Bilharzial infarction may be a stimulus to the development of malignant new growths (Harrison and Colloridi).[3] The patches and new growths, when invading the tissues near the entrance of the ureters into the bladder, may and do contribute to hinder the escape of the urine into the bladder, and thus by

[1] "La Bilharzia hæmatobia — Osservazioni anatomo-patologiche e cliniche del Dott T. Belleli," *Estr. dalla Gazzetta degli Ospitali*, 1886; and for Kartulis, *loc. cit.*

[2] See Die Menschlichen Parasiten, etc., Leipzig, 1863, Erster Band, p. 629.

[3] See *Lancet*, 1889, ii. p. 163; and Colloridi, *loc. cit.*

themselves, or together with embedded stones, may be the cause of even grave renal mischief.

Dilatations of small veins or capillaries containing adult worms and fresh eggs are often found in the new growths.

The urinary bladder, when the disease has proceeded farther, has often its walls much thickened from hypertrophy of the muscular coat and hyperplasia of the connective tissue, and its cavity is frequently rather smaller than normal, and sometimes almost entirely occupied by the new growths.

The interior of the bladder is found coated by bloody mucus and flocculi such as are found in the urine. Sand and small stones even are found; the latter may be free or embedded in the opening of the ureters, or in accidental diverticula of the vesical walls. Eggs of *Bilharazia* are found in the bloody stained mucus and flocculi.

The ureters are often affected, but perhaps less frequently so than the bladder. The Bilharzial infarction is more frequent in the lower part of their course; I have found it, however, in the median and even in the superior portions, not far from the renal pelvis. Griesinger found, but very rarely, similar alterations in the pelvis of the kidney. I have sometimes seen, even in the ureters, the commencement of these alterations as manifested by small miliary vesicles, such as I have described in connection with the bladder. The Bilharzial infarction often appears in the ureters as small, hard, and rough patches, disposed as a ring occupying the periphery of the canal. These patches are often smeared by viscid mucus, and sometimes present, included in their substance, small stones, usually of a black colour, which render the passage of the urine difficult, and are in this manner the point of departure of grave renal alterations, especially of small renal dilatations, and eventually of hydronephrosis. Bilharz speaks of a case in which complete occlusion of a ureter was found, and which had given rise to destruction of the renal tissue, the kidney being transformed into a cyst, having the appearance of a lady's fan.[1] I recollect a case in which the necropsy showed a double hydronephrosis, in which one of the kidneys, with the corresponding ureter, was of such a size, and occupied so much space in the abdominal cavity, that when this was opened the enlarged kidney appeared as if it were the stomach enormously distended. The two cysts were filled with bloody and purulent urine. This double hydronephrosis had been determined by the occlusion of both ureters by small stones impacted at their opening into the bladder; and these

[1] See *loc. cit.* in *Wien. med. Wochenschrift.*

stones, in their turn, were formed in consequence of Bilharzial infarction.

Even in the tissue of *the kidney*, eggs of *Bilharzia* have been found sometimes by Kartulis, Mackie, and Bowlby,[1] and not in small numbers. In long-standing cases the kidneys often exhibit alterations, due rather to secondary diseases of other parts than to the deposit of eggs in their tissues, which, when it does occur, is generally only to a very limited extent. Besides pyelitis and kidney abscesses—the consequence of cystitis, and hydronephrosis —the result of an obstacle to the passage of the urine, the kidneys may present alterations referable to any other form of nephritis.

Next to the bladder, *the seminal vesicles* are the organs most frequently and most severely affected. The alterations in them resemble closely those in the bladder, their walls being thickened and indurated, and filled with eggs more or less calcified. A fact noted by Mohammed Chaker[2] is, that the eggs abound even in the muscular coat. Eggs in great quantity are found also upon the inner surface of the organ, and thus is explained their presence in the semen.

Even *the female genital organs* may be and are subject to the Bilharzial infarction. This has been verified by recent observations in living subjects made by Chevreau and de Chazal at Pamplemousses (Mauritius). They observed several instances of new growths, not only in the vaginal wall, but even in the interior of the cervix uteri; these were found to contain, not only eggs, but even adult *Bilharziæ*. In some cases the infarction of the vaginal wall had rather the character of patches with incrustations, and in every instance there were signs of vaginitis, more or less discharge, often bloody, and containing the characteristic eggs.

Hæmorrhagic infarctions are frequently found under the mucous membrane of *the large intestine*, and often assume very large dimensions; this is due probably to the great vascularity of the organ. Polypiform growths are also common; sometimes, owing to their situation, they may be mistaken for piles; but they are generally more deeply seated, and are usually harder than piles, and have the histological characters rather of adenomata than of papillomata. According to my observations,[3] confirmed by those of Mackie, the eggs in the tissues of the intestine often present a

[1] For a case examined by Bowlby, see *Lancet*, 1889, i. p. 786.

[2] "Etude sur l'hematurie d'Egypte causeé par la Bilharzia hæmatobia," *Thèse de Paris*, Paris, 1890.

[3] See "Della Bilharzia hæmatobia e delle alteranzioni anatomo patologiche che induce nell' organismo umano," etc., Firenze, 1876. Reprinted from the medical journal, *L'Imparziale*, p. 19.

lateral (Plate VII. Fig. 7), instead of the ordinary terminal spine; whilst in the case of the eggs discharged with the urine, the spine appears always to be terminal. The lateral situation of the spine in that case is usually to be regarded as the result of a deformity produced in the eggs by compression in the tissues of the intestine, instead of as a primitive malformation produced while the eggs were passing through the shell gland of the worm (Fritsch).

According to Belleli and Kartulis, eggs are more abundant in the submucous than in the mucous layer of the intestine; they are very rare in the muscular coat. The latter is, however, hypertrophied in the places corresponding to the infarctions. I once observed a case in which eggs were abundant even in the muscular coat.

As for *the liver*, the deposit of eggs therein, as seen by Bilharz himself, and confirmed by Kartulis and Schiess Bey, is generally very scanty. In this organ the eggs produce a sclerosis of the tissues, and thus the clinical form that may arise is a kind of cirrhosis; but this is rarely sufficiently pronounced to be capable of being diagnosed during life. No relation has hitherto been established between Bilharzial infarction and liver abscess, a condition very frequent in Egypt, but not more so than in many warm countries of Asia and America where *Bilharzia* has not been found.

Even the presence of eggs in *the lungs*, noted first by Mackie,[1] seems to cause a certain degree of hyperplasia of the connective tissue, but till now there are no observations in which the pulmonary alterations had reached such a degree of severity as to have given rise during life to special morbid disorders.

Symptoms, Course, and Issue of Bilharzia Disease.—The symptoms of Bilharzia disease refer especially to the urino-genital system and to the intestines. The more important are those referable to the urinary organs. They begin with a peculiar form of hæmaturia, which has long been known to be endemic in the countries where the existence of the worm has now been established. The characters of this hæmaturia are generally very peculiar. In recent and uncomplicated cases, it is only the last few drops of urine that are blood tinged, or there may be some drops of pure blood passed at the end of and after each act of micturition. This is the reason that in the young it is very frequent to find the shirt stained by blood. Often hæmaturia begins without any subjective symptom, and for this reason it may exist for some time unobserved. Sometimes there is some pain at the meatus, or a sense of weight or pricking in the perineum, especially during the act of micturition.

[1] See *Lancet*, 1885, ii. p. 168.

At other times pain is severe from the beginning of the hæmaturia, and may extend to the hypogastric and lumbar regions; and this pain may even, in some cases, precede the onset of hæmaturia.

The blood passed after the act of micturition sometimes becomes more abundant in consequence of unusual bodily exertion, or of excesses in eating and drinking, or even without any apparent cause. Blood may also be more extensively mingled with the urine, giving it a bloody tinge more or less pronounced, and forming clots that may be the cause of dysuria and even of ischuria. This increase of blood is generally transitory, and lasts only for some days or weeks, after which it is again passed only in small quantities.

The appearance and characters of the urine of persons affected with Bilharzia disease are after all very variable, not only in different individuals, but even at different times in the same individual. Nothing particular is to be noted concerning the specific gravity. The reaction generally is acid, even when the urine contains a considerable quantity of blood. A neutral or alkaline reaction is a consequence rather of a certain degree of vesical catarrh. The urine in ordinary cases may appear at first sight of an amber colour, and limpid, like normal urine; but viewed in bulk through the walls of a glass, it is easy to distinguish in it some minute dirty-white or yellowish-grey, or even red flocculi or specks, or thin filaments much coiled upon themselves. Being heavy, the flocculi, specks, or filaments after a time fall to the bottom of the glass. These particles are the characteristic product of the specific catarrh due to Bilharzia disease in the bladder, or other parts of the urinary apparatus. As seen by the microscope (Plate VII. Fig. 8) they consist in different proportions of red corpuscles, leucocytes, epithelial cells, and *Bilharzia* eggs connected together by mucus, and are often mixed with crystals of uric acid, oxalate of lime, or triple phosphate.

When there is an increase of hæmaturia the urine is of a red or brown colour, depositing a sediment of blood more or less abundant, and which is often composed of clot. Between the urines of a limpid or amber colour and those of a brown colour there are numberless gradations, to which correspond different proportions of sediment.

The hæmaturia with its alternations lasts for months and years, and then in many cases begins to subside little by little till it disappears altogether, so as to give rise to the hope that recovery is complete. In some cases it really seems that this complete recovery does take place; when this happens it is in consequence of the death or discharge of the worms, without a fresh infection occurring. But

in many other cases after the hæmaturia has altogether subsided, if we repeat our examination of the urine, collecting directly upon a slide the last drops of urine passed in the act of micturition, we find under the microscope that they still contain fresh eggs of *Bilharzia*, showing that the infection still persists. Even after the complete cessation of the hæmaturia, it often happens that other disorders, as gravel or stone, supervene in consequence of the Bilharzia infection. The presence of eggs in the last drops of urine could probably be made out in some cases at the beginning of Bilharzia infection, even before the onset of hæmaturia.

In other cases when the infection is the result of numerous worms, and fresh infections succeed one after another, the hæmaturia instead of subsiding is attended with even more severe symptoms of catarrh, both the hæmaturia and the catarrh being aggravated and maintained by the supervention of gravel, or of stone in the urinary tract. Thus the pain in such cases becomes more severe, the urine more purulent, and even vesical tenesmus, dysuria, ischuria, appear and aggravate the state of the patient. The gravel and stone must be regarded only as effects of the Bilharzia disease. In fact, Meckel, Hartmann, and others (among them myself) proved that many urinary calculi in Egypt contain within their nuclei clusters of the eggs of *Bilharzia*. I found eggs of *Bilharzia* only in the small and multiple stones composed of uric acid.

It is necessary to remember that the symptoms of stone in the bladder may be simulated by the hardened and rough patches produced by the deposit of eggs, and by the incrustation derived from the urine which forms on them. It is necessary that a careful examination by the sound be made so as not to confound these two widely different conditions.

When Bilharzia disease is so far advanced, it is indeed necessary to have recourse to every kind of exploration, as catheterism, sounding, examination by the finger in the rectum, etc., made with all precautions, to ascertain the true state of the bladder and adjoining organs. Indeed, the bladder, as we have seen when treating of the morbid anatomy, may present not only patches with incrustations, but even new growths of different forms and sizes, and its walls may be hypertrophied, reducing its cavity, whilst at other times there may be dilatation of the bladder. The presence of new growths may become the cause of a fresh onset of hæmaturia, or of its increase; and in these cases the hæmaturia is often abundant, and the blood may be passed at every act of micturition. We have mentioned that even carcinoma of the bladder, appearing

in old standing cases of Bilharzia disease, is thought by some observers (Harrison, Colloridi) to be the consequence of Bilharzia disease, as are also urethro-perineal and vesico-perineal fistulæ (Mackie). In such cases we have all the symptoms that ordinarily accompany these affections, with the addition that eggs of *Bilharzia* are discoverable in the altered tissues, or in the discharges.

Other symptoms may appear, pointing specially to affection of the seminal vesicles, or of the prostate. The finger introduced into the rectum may aid in the detection of these latter affections. In the case of lesion of the seminal vesicles there are often abnormal ejaculations or spermatorrhœa, and the semen may be mixed with blood and contain eggs of *Bilharzia*. Prostatic implication is a cause of irritable bladder, vesical tenesmus, and dysuria, and is often evidenced by enlargement of the organ. In other cases the pains and disorders point to an affection of the ureters and kidneys, which may end in uræmia.

When the infection is recent and mild, being produced only by a small number of worms, and the consequent hæmaturia is inconsiderable, the complaint causes but little annoyance, and the general health is not compromised. But when hæmaturia lasts for a long time, and catarrh is severe, the patients come to suffer in health; and this is shown by a certain degree of pallor, anæmia, and weakness. In older and complicated cases, not only are the anæmia and weakness more pronounced, but emaciation also supervenes, with all the tortures and distresses which accompany the severer forms of urinary affections.

As for the symptoms due to Bilharzia disease of the intestine, they have not yet been well determined. Often they consist of a state of irritation due to catarrh of the large intestine, and especially of the rectum. This catarrh must not be confounded with the true dysentery of hot climates, which is present as an endemic disease in countries where *Bilharzia* has not been found, and which even in Africa often attacks persons not infected by this worm. Even Griesinger, who was prone to refer the origin of dysentery in Egypt to Bilharzia infection, declared that he had met with cases of the more severe forms of dysentery, in which the presence of the eggs of *Bilharzia* was negatived even in the necropsy.

Yet the symptoms of the localisation of Bilharzia disease in the intestine resemble much those of ordinary dysentery. Indeed, we often find in the former as in the latter a pain with heaviness in the lower end of the rectum, with frequent desire to go to stool, and with evacuation only of bloody mucus,—symptoms that present often a chronic course, and that are often accompanied by exhaus-

tion. With the finger introduced through the anus, small soft growths are made out; and if one of these is removed and examined with the microscope, *Bilharzia* eggs are discovered; eggs which often possess a lateral instead of a terminal spine (Sonsino, Mackie). According to Mackie, there are cases of infection of the intestine without any evidence of it in the genito-urinary apparatus. Besides the symptoms of irritation already mentioned, the infection of the intestine may give rise even to recurrent hæmorrhage—generally, not abundant. In a case related by Arthur Davies,[1] in which was noted the passing of pure blood with mucoid clots *per anum*, numerous eggs—all of them having terminal spines, however—were found containing living embryos.

As for the localisation of Bilharzia disease in the female genital organs, we have already noted that this may give rise to symptoms of vaginitis and bloody discharges; and it is probable that in some cases the presence of Bilharzia disease in these organs may even interfere with the normal accomplishment of the reproductive functions.

As we have said, the eggs of *Bilharzia* have been found deposited in the tissues both of the liver and lungs. Until now we are ignorant of the clinical phenomena produced by those deposits. We know nothing about the disorders that may arise from embolisms and septicæmia consequent on the death and decomposition of worms not expelled by the natural passages from the organism.

We may also mention that Griesinger once found the shell of an egg in the blood of the left side of the heart. We must also mention that Gautrelet[2] found eggs of *Bilharzia* in a gall stone passed by stool by a woman who twenty years before had lived in Egypt. These observations would extend even further the influence of *Bilharzia* as a cause of disease.

Nor can we neglect certain observations due to Griesinger, which give rise to the suspicion that *Bilharzia* may extend so much its pathogenetic action as to determine sometimes acute disorders ending in death. He speaks of the necropsy of two cases, terminating after an obscure illness of brief duration, in which the only morbid lesions discovered were recent Bilharzial infarctions accompanied by evidences of recent vesical and pelvic catarrh, together with a state of red-brown diffused hyperæmia of the kidneys, unaccompanied by signs of ancient Bilharzial infarction, and without independent

[1] A case of endemic hæmaturia from the Cape of Good Hope. Reprinted from *St. Bartholomew's Hospital Reports*, vol. xx.

[2] See *Union Médicale*, 1885, quoted by Mohammed Chaker's thesis above cited.

lesions of other kinds which could explain the death. Thus Bilharzial infection can occasion a grave complaint, ending in death within a brief time, from acute lesions, referable especially to the kidneys. This must be kept in mind by practitioners in Egypt and elsewhere throughout Africa, where *Bilharzia hæmatobia* may be found, and figure as an element in the morbidity and mortality of mankind.

Diagnosis.—Although the diagnosis of Bilharzia disease is easy, it cannot be effected with certainty without the objective evidence of the presence of the worm, which is to be obtained by discovering its eggs with the microscope in the urine or fæces, or in diseased tissues, such as rectal growths removed from the body. As for the urinary organs, the characters of the hæmaturia are generally so peculiar as to render this alone sufficient for suspecting its parasitic origin, whenever the patient belongs to or has lived in a country where Bilharzia disease is endemic. Blood at the end of micturition may be met with also in some urethral diseases, or in stone, especially in children; but in these latter cases there are other symptoms of those diseases, and the onset of the hæmaturia is preceded and accompanied by other troubles.

I have seen in Egypt cases of simple hæmaturia instead of chyluria produced by *Filaria sanguinis;* but the characters of the hæmaturia in this case are very different from those of Bilharzial hæmaturia, as it is generally abundant, and presents itself in rare intermittent attacks.[1] The two parasites may coexist, and then we ought to arrive at the diagnosis of each by the detection of their products—eggs in one case, and embryos in the other.

It is necessary to bear in mind that in some cases we can have Bilharzia disease without evident hæmaturia, and that in such a case eggs may also be found in the last drops of a bloodless urine.

As diagnosis must also include the alterations left after the natural death of the worms, it is sometimes necessary with a sound to scratch gently the surface of the bladder with a view of obtaining some particles of mucus, or encrustations in which the microscope may detect the presence of old eggs.

When the symptoms point to the intestine the microscopical examination of the fæces, or of the mucosites that may be picked up by the introduction of the finger, is necessary; and when there is any polypiform growth, the excision of a bit of this followed by its histological examination, may assist the diagnosis. It ought not to be forgotten that in African countries, especially in Egypt,

[1] See "A new series of cases of *Filaria sanguinis* parasitism observed in Egypt," etc., in *Med. Times and Gaz.* Sept. 22, 1883.

there is no disorder or symptom referable to the urinary tract that may not have its cause in *Bilharzia;* it is therefore only the absence of the eggs that can assure us of the non-existence of this cause.

Finally, in any obscure and doubtful case of disease in the above-named countries, in order to arrive at a diagnosis we ought to have recourse to further microscopical examinations, as of the semen, of the sputa, or of pus from any source, to ascertain the presence or absence of eggs.

Prognosis.—Bilharzia disease, though not of itself, may often prove fatal through its consequences; and on this account it must be reckoned as a dangerous disease. Moreover, it generally runs a long course; and even after the death of the worms, alterations are left that give more or less trouble for the rest of life.

When the infection is of long standing, and the number of the worms is great, there are always signs of anæmia and feebleness. The patient, when the disease becomes complicated, loses much of his physical and mental energies, owing to the sufferings which are inherent to all urinary complaints. Hence Bilharzia disease acquires a gravity not only as regards the individual patient, but even as regards the welfare of an entire population wherever it is common and extensively diffused, as it is in some African countries. Moreover, the patches found in the urinary bladder are incapable of healing; and if a stone be formed in the bladder, or in some other part of the urinary system, though it may be removed, it is always a very grave complication. Yet, the gravity of Bilharzia disease is certainly in relation to the number of the worms, which we can estimate only in a rough way by the number of the eggs discharged in the excreta (urine and fæces). A small number of worms probably does not produce alterations of great importance. This, perhaps, explains why the disease appears more grave or less grave in different countries. The descriptions of the disease as observed in Egypt give to it a gravity that does not appear from the descriptions of the disease as it is observed at the Cape. Probably at the Cape there is less chance of being infected with a great number of worms, and these in the majority of cases remain small in number.

The statement that Bilharzia disease must be regarded rather as one of the curiosities of pathology than as an affection of much gravity or importance, is at variance with what has been observed, not only in Egypt, but even in some cases in European patients who have contracted the disease in parts of Africa other than Egypt. We cannot comprehend in what way the parasite could act differently on its host in different countries.

The duration of the life of the worm appears to be very long, if I may judge from a case of whose authenticity I cannot entertain a doubt. It is the case of a native gentleman, who, having left Egypt when a boy while suffering from Bilharzial hæmaturia, lived uninterruptedly nine years in France for the purpose of pursuing his university studies. When, after this long absence, he returned to Egypt, though he was apparently free from hæmaturia, it was ascertained that he still passed living eggs of *Bilharzia* in the last drops of his urine. Unless we admit that this gentleman was the subject of a reinfection while in France, which does not appear at all likely, we must infer that in this case one at least of the worms lived not less than nine years. This long duration of the life of *Bilharzia* is a circumstance that renders the prognosis of the disease even graver. Finally, the gravity of the prognosis is further aggravated by the facts that as yet we have no means of eradicating the disease, and that we can only oppose to it a palliative treatment, which cannot prevent its dangerous sequelæ.

Treatment.—The treatment of Bilharzia disease should aim at fulfilling two indications — 1st, to procure the expulsion of the worms; 2nd, to repair the lesions produced by the worms in the organs. Unfortunately we have no means at our disposal for procuring either the expulsion or the death of this parasite, and therefore we must limit the treatment to the second indication, that is, of rendering its presence less dangerous by combating its effects. From our acquaintance with the precise seat of the parasite, it is clear that the application of a local treatment, such as injections into the bladder, cannot reach the bulk of the adult worms so as to kill them. To profess, therefore, to cure radically hæmaturia arising from *Bilharzia* with medicated injections (perchloride of mercury or other substances), as has been advanced by some practitioners, is a mere delusion, because it is not possible to reach and kill with these injections the worms, which live principally in the various branches of the portal system. The cessation of hæmaturia during any treatment is, in most of the cases, only accidental, and due to the natural course of this hæmaturia, which is not always continuous. But temporary cessation of hæmaturia is not recovery, as it is not possible to obtain a radical cure without getting rid of the worms. I certainly do not assume that it is impossible to discover a substance that, introduced into the blood, would kill the worm without endangering the life of the host; but this remedial agent has yet to be found. Meanwhile, we may be allowed to entertain the suspicion expressed long ago, that dead worms in the circulation may prove more dangerous than

living ones, as they may then become more readily the starting point for the formation of clots and embolisms. But, apart from this, no substance can be effectual if it be not absorbed and diffused through the blood; and this very probably is not the case with substances introduced into the urinary bladder, and in any case we would attain our end better in other ways.

Whoever has seen the morbid alterations so often produced in the walls of the urinary bladder by the deposition of ova in the coats, viz. the patches we have described, cannot believe in the power of medicated injections to cure them. Medicated injections, it is true, may kill the embryos in the ova poured into the cavity of the bladder, but that is not a result of any real importance, either for the individual or for the protection of others, seeing that the embryos do not grow to adult life in their original host, and if discharged and allowed to remain in the urine, especially if it becomes stale, soon die. Medicated injections, and, above all, washing out the bladder with mild antiseptic and astringent fluids, can only be useful in alleviating the complicating catarrh of the bladder and prostate, fulfilling in this way the second indication of treatment. We must be cautious, however, in the choice of substances for injection, as the urinary tract when affected by Bilharzia disease reacts with much intensity to any stimulus; and I have seen, and others have seen, intense cystitis arise from carbolic acid, or potassium iodide solutions (Guillemard [1]) used as injections, with the hope of curing the hæmaturia.

The catarrh of the urinary tract is the condition we ought principally to keep in view in treating Bilharzial patients. And we should have recourse not only to the washing out of the bladder, but also to the constant administration of internal remedies. In choosing these we must follow such special indications as may be gathered from treatises on diseases of the urinary organs. Thus, gallic acid or its substitutes, uva ursi and buchu, will answer in certain cases, whilst in others the use of anodynes and bland diluents will be more appropriate, whilst at other times these may be alternated with astringents or balsamics.

Gravel and stone will demand special treatment, especially surgical measures, for which we also refer the reader to special treatises. The same remark applies for new growths in the bladder. We will only remark about surgical measures, that in these later years Mackie in Alexandria seems to have had very good results from cystotomy, not only in cases complicated with stone or new

[1] On the Endemic Hæmaturia of Hot Climates caused by the presence of Bilharzia, etc., London, 1882, pp. 3 and 53.

growths, but even in cases of simple but long standing Bilharzia disease; such a proceeding relieves the pains and the dangers of grave catarrh of the bladder.

As for the condition of the intestine, it is clear that whenever new growths in the rectum are within reach, they ought to be extirpated; for the rest we must be content to make use of those remedies which best serve to allay the complicating intestinal catarrh, or to stop hæmorrhage.

Surgical means, as the extirpation of new growths or the scraping of patches, are applicable even to the Bilharzial infarction of the vagina and cervix uteri. They have been had recourse to by Chevreau and de Chazal with good results, so far as concerns the local lesions.

In every case we must keep in view the condition of the patient, and try to maintain the general health by tonics, when necessary, and by a suitable regimen. We must rest content with alleviating the disease, as we must admit that it is curable by nature only and not by art. Nature certainly does effect a cure in many cases; this is evident if we bear in mind, that of the great number of persons who suffer from hæmaturia in Egypt, only a limited number die from Bilharzia disease, or become eventually the subjects of grave troubles arising from it. Whether nature rids the organism of the worms only by their death, or whether on attaining a certain age the worm itself spontaneously quits the host, we do not know, as there are no observations of adult worms being seen in the urine. Yet a case in which I saw double adult *Bilharziæ* in the wall of one ureter, lying in the tissues outside the blood vessels, led me to suspect that they might have been searching for a change of abode in order to deposit their eggs in the open air.

F. Macready Chute,[1] having seen the spontaneous cessation of Bilharzial hæmaturia after a severe attack of continued fever, suggests that the fever poison may have proved fatal to the entozoon. He does not ascribe the supposed death of the parasites in this case simply to the high temperature, as recovery has not ensued in other cases of high temperature—in mumps or measles, for example. Although a single case cannot be used to draw any conclusion from, yet it is certainly suggestive of new inquiries on the subject.

Before concluding the subject of treatment, we must say something about a question that is often put to the medical man, viz.

[1] See Chute's "Bilharzia hæmatobia," etc., in the *South African Medical Journal*, Oct. 19, 1888, p. 90 and following.

whether it is better for a patient suffering from Bilharzia disease to leave the country where he has been infected? There is no doubt that leaving for another country where the parasite does not exist insures the patient against further infection, and consequently against an aggravation of his condition. But change of place cannot be useful to him in any other way; and if the patient observe those measures that preserve from reinfection, he will secure the same advantages, and that without the inconveniences attending change of place. This brings us to speak of prevention.

Prevention.—To establish the rules of prevention from a parasitic disease on solid ground, we must become acquainted, before all, with the life history of the parasite; unfortunately, as we have already said, this is not yet made out for *Bilharzia*. Yet we have sufficient facts to permit us to state, almost with certainty, that the *Bilharzia* is taken in with drinking water. The facts are as follows:—First of all, the eggs of *Bilharzia*, discharged from the human organism either with the urine or with the fæces, develop an embryo which, being ciliated, presents an organisation suited only for aquatic locomotion. Thus we are led to believe that it is only in water that this embryo can accomplish its ulterior transformations—whether these be through an intermediary host or otherwise—by which it can attain that stage of growth in which it is capable of developing into the parasitic stage on being introduced by means of water into the human body. In the second place, we have sufficient evidence to show that the persons most subject to infection by *Bilharzia* are just those who drink water without any regard to its source. Thus in Egypt the disease is generally confined to the natives, and especially to rustics, who disregard the rule of filtering their drinking water. Indeed, they use in their houses a kind of porous earthenware pitcher to store water. These pitchers could serve very well as filters if used in the European manner; but instead of this, the filtered water which percolates through the walls of the vessel is left to run to waste, and that which is left in the interior of the pitcher, in a concentrated state of impurity, alone is drunk, being agreeable on account of its coldness.

People, especially men and boys, often bathe in canals and in the Nile, and often pollute it with their excreta, while at the same time they quench their thirst with the water. Not only so, but they are accustomed also to draw their drinking water from gutters or puddles in the fields, which are even more polluted by excreta.

In Egypt, and generally in all tropical and subtropical countries, even in South Africa, people, especially the male sex, often spend no

inconsiderable portion of the day in the water; and observers in those countries have noted that the infection is more commonly found in those that bathe often in the rivers or streams. Guillemard's observation, that *Bilharzia* occurs more frequently in boys and men than in women, may be explained by the different habits of the sexes as regards bathing. So far back as 1875,[1] I stated that some of my patients afflicted with endemic hæmaturia from *Bilharzia* told me that their complaint had originated after bathing in the Nile. I inferred then, as I do now, that *Bilharzia* may be contracted by swallowing water while bathing.

Some observers in South Africa explain the fact of the contraction of *Bilharzia* while bathing, by suggesting that the parasite is introduced on these occasions by the way of the skin, urethra, or anus;[2] a manner of introduction which I have already rejected as being next door to the impossible.

In Egypt there are other well-known facts in support of the belief that the introduction of the parasite takes place in foul drinking water. In Alexandria and Cairo, where a general system of purification of the water of the Nile is practised, though not a perfect filtration, Bilharzia disease is generally less frequent than in the smaller towns and villages of the rest of the country, and the Europeans, in those cities, who take the precaution of filtering in their own houses the very impure water are never subject to the parasite. If there are examples of Bilharzia disease in Europeans, it is always in persons who had been placed in circumstances in which they had to drink unfiltered water.

I have thus come to the conclusion that whilst it may be held that in Egypt perhaps no one who habitually drinks unfiltered water reaches old age without suffering from Bilharzia disease, on the other hand, there is no person who, having always made it a point to drink filtered water only, has become affected with this disease.

Filtration or boiling is the great and only preventive against Bilharzia. And I believe that if the natives of Egypt would use their porous earthenware pitchers as a filter, as Europeans do, the prevalence of Bilharzia disease would in a short time be much reduced, to the great benefit of the native population.

The prevention of *Bilharzia* may thus be reduced to a few simple rules which I embodied in the following words in a paper read at the last International Congress of Hygiene: "Pure spring water, or

[1] See Della Bilharzia hæmatobia, etc., Firenze, 1876, p. 37.

[2] See Guillemard, *loc. cit.* at p. 32, and Allen's "Parasitic hæmaturia, or bloody urine," in *Practitioner*, April 1888, at p. 310.

else boiled or filtered water alone to be drunk. Drinking water to be preserved in good and well-covered vessels. River or lake water not to be imbibed while bathing."

DESCRIPTION OF PLATES IV. TO VII.

PLATE IV.

Eggs of worms found in the alimentary canal of man :

FIG. 1, *a*, *b*, *Ascaris lumbricoides.*
FIG. 2.—*Oxyuris vermicularis.*
FIG. 3.—*Trichocephalus dispar.*
FIG. 4, *a* and *b*, *Anchylostoma duodenale.*
FIG. 5.—*Distomum lanceolatum.*
FIG. 6.—*Fasciola hepatica.*
FIG. 7.—*Distomum sinense.*
FIG. 8.—*Bilharzia hæmatobia.*
FIG. 9.—*Tænia solium.*
FIG. 10.—*T. mediocanellata.*
FIG. 11.—*T. nana.*
FIG. 12.—*T. leptocephala.*
FIG. 13.—*Bothriocephalus latus.*

FIG. 1, *a* and *b*, $\frac{350}{1}$. FIGS. 6 and 8 $\frac{200}{1}$; all the others $\frac{400}{1}$.

PLATE V.

FIG. 1.—*Distomum heterophyes*, $\frac{35}{1}$. (After Leuckart.)
FIG. 2.—*Distomum Buski.* Nat. size. (After Leuckart.)
FIG. 3.—*Amphistomum hominis*, $\frac{2}{1}$. (After MacConnell and Lewis.) *a*, ventral aspect; *b*, dorsal aspect.
FIG. 4.—*Anchylostoma*: *a*, male ; *b*, female. (After Perroncito.)
FIG. 5.—*Anchylostoma.* Mouth capsule. (After Perroncito.)
FIG. 6.—*Anchylostoma.* Bursa of male.
FIG. 7.—*Anchylostoma.* Male and female coupled. (After Leuckart.)
FIG. 8.—*Anchylostoma.* Egg with embryo, $\frac{350}{1}$.
FIG. 9.—*Anchylostoma.* Embryo hatching. (After Perroncito.)
FIG. 10.—*Anchylostoma.* Larva. (After Perroncito.)

PLATE VI.

FIG. 1.—*Rhabdonema intestinale.* Adult parasitic stage. (After Grassi and Parona.)
FIG. 2.—Chain of eggs of *R. intestinale* seen in the contents of the intestine eight hours after the death of the bearer. Oc. 3, obj. 8 closed, Hartnack. (After Golgi and Monti.)
FIG. 3.—Section of mucous membrane of duodenum, showing the seat of eggs and parasite, with infiltration of leucocytes between muscularis mucosæ and the epithelium of the glands. Oc. 3, obj. 7, Hartnack. (After Golgi and Monti.)
FIG. 4.—Embryo of the parasitic worm taken from the contents of the intestine. Oc. 3, obj. 8, Hartnack. (After Golgi and Monti.)
FIG. 5.—Filariform larva, arising from the embryo of the adult free form (*A. stercoralis*). Oc. 3, obj. 8, Hartnack. (After Golgi and Monti.)
FIG. 6.—Male of the free form (*A. stercoralis*).
FIG. 7.—Female of the free form (*A. stercoralis*).

PLATE VII.

FIG. 1.—*Distomum sinense: a*, nat. size ; *b*, egg $\frac{350}{1}$ (after MacConnell); *c*, $\frac{8}{1}$ (after Leuckart).
FIG. 2.—*Distomum conjunctum : a*, nat. size ; *b*, egg $\frac{350}{1}$; *c*, $\frac{8}{1}$ (after MacConnell).
FIG. 3.—*Bilharzia hæmatobia*, male and female, the latter in the canalis gynœcophorus of the former. (After Fritsch.)
FIG. 4.—Two eggs of *Bilharzia*, with polar spine, as they appear in fresh urine, $\frac{180}{1}$. (After Harley.)
FIG. 5.—Egg of *Bilharzia* with lateral spine, $\frac{350}{1}$.
FIG. 6.—Embryo of *Bilharzia*, $\frac{350}{1}$. *a*, position of ciliated infundibula.
FIG. 7.—Section of polypiform growth of rectum, with eggs of *Bilharzia : a*, egg with polar spine ; *b*, egg with lateral spine ; *c*, transverse section of egg ; *d*, section of gland.
FIG. 8.—Mass of mucus, containing ova, derived from a specimen of urine, $\frac{50}{1}$. (After Harley.)

CHAPTER XXIV.

DISEASES OF THE SKIN IN TROPICAL CLIMATES.

BY PATRICK MANSON, M.D., M.R.C.P., LL.D.

General Remarks.—In tropical and subtropical climates, with the exception of such diseases as depend on low atmospheric temperature, such as chilblains, most if not all of the skin diseases of Europe are to be found, many of them—in consequence of the fostering and stimulating effects of high temperature, abundant moisture, and dirty, unhygienic habits—in an aggravated degree. Thus in those climates all the epiphytic and ectozooic diseases of Europe are very prevalent, and generally in a severe form; ringworm, favus, pityriasis versicolor, scabies, phthiriasis, are only too common, unless in such countries as Japan, where personal cleanliness amounts almost to a religious sentiment. On the other hand, there are one or two communicable skin diseases which seem not to be able to thrive well in very high temperatures; among the exanthemata, scarlet fever is a notable example of this; and among the less grave diseases might be cited molluscum contagiosum, which, according to my experience in China and Chevers' in India, is a very rare disease in hot countries; common warts too, a feature on almost every schoolboy's hands in Europe, are, like molluscum contagiosum, rare in tropical countries, at all events in some of them.

If we would attempt to particularise and classify the causes principally concerned in bringing about the peculiarities of the endemicity and spread of skin disease in the tropics, the following arrangement might be suggested :—

1st. *Direct solar heat and high atmospheric temperature;* giving rise to or aggravating such affections as leucoderma, sun erythema, prickly heat.

2nd. *Heat combined with moisture;* favouring such specially tropical diseases as tinea imbricata, pinta, pemphigus contagiosus,

and aggravating the other epiphytic diseases common to tropical and temperate climates alike.

3rd. *Personal habits;* leading to the spread of the epiphytic diseases, and such contagious skin affections as yaws, syphilis, parangi, leprosy, itch, phthiriasis.

4th. *Racial proclivities;* developing keloid.

5th. *The distribution of intermediary hosts of parasites;* determining the distribution of filaria disease, guinea-worm disease, probably craw-craw.

6th. *The distribution of ectozoa;* such as *Pulex penetrans.*

7th. *Unknown endemic conditions;* producing such affections as verruga, Oriental sore, sloughing phagedæna.

In the following sections no attempt will be made to treat the subject of skin diseases in the tropics exhaustively; to do so would be to write a book on skin disease in general. All that can be done here is to treat rather fully of those which are peculiar, or nearly so, to the tropics, and to indicate, principally from a practical point of view, some peculiarities impressed on certain pandemic skin diseases by tropical conditions, and to suggest appropriate treatment.

Section I.—Epiphytic Skin Diseases.

1. Tropical Ringworm.
   (*a*) *Tinea circinata.*
   (*b*) *Tinea imbricata.*
2. Pinta.

Section II.—Skin Affections caused by Animal Parasites.

1. Craw-Craw.
2. Guinea-Worm.
3. Filaria Loa.
4. Filaria Volvulus.
5. The Chigger or Sand-Flea.
6. Cutaneous Myiasis.
   (*a*) *Lucilia macellaria.*
   (*b*) *Dermatobia noxialis.*
   (*c*) *Ochromyia anthropophaga.*
7. Leeches.

Section III.—Skin Affections probably caused by Micro-Organisms.

1. Boils.
2. Tropical Sloughing Phagedæna.
3. Oriental Sore.
4. Pemphigus Contagiosus.
5. Verruga.

Section IV.—Unclassed Skin Diseases.

1. Prickly Heat.
2. Keloid.
3. Ainhum.

SECTION I.—EPIPHYTIC SKIN DISEASES.

1. Tropical Ringworm.

(*a*) ***Tinea circinata.***—The ordinary form of body ringworm (*Herpes circinatus, Eczema marginatm, Tinea circinata, Tinea corporis*)

as met with in the tropics differs in no essential respect from the body ringworm of more temperate climates. Owing to the fostering influences of heat and moisture the trichophyton grows more luxuriantly in the tropics; hence the disease it gives rise to is more prevalent, more persistent, spreads more rapidly, and over larger areas of skin, is more difficult to cure, and more prone to relapse, than in the temperate zones. Depending on the same parasite,[1] however, the trichophyton, it is essentially the same disease. It is very common in all tropical countries, but especially prevalent in those in which the atmosphere as well as being hot is impregnated with moisture. It is known by a variety of names such as dhobie (washerman) itch, Burmese ringworm, Chinese ringworm, and a number of other local names; under some of these it is familiar to every resident in tropical and subtropical climates.

As its ordinary features are fully described in all text-books on skin disease, it is unnecessary to enter much on the symptomatology, pathology, etc., here. On the bodies of natives it is seen as rings, or parts of rings, enclosing areas of variable extent, the surfaces of which have usually a slightly lighter colour than the surrounding healthy skin. The periphery of the patch is abruptly defined by a red, slightly raised, scurfy, itching rim about a line in breadth, abrupt to the sound skin, shading off more gradually on the diseased side. In the paler area included in the ring there is a certain amount of scurfiness, and here and there, perhaps, spots or short lines of active disease, but seldom a second or third ring of any extent. When the patch is small the ring is usually complete; but when it has attained a diameter of several inches it is invariably interrupted in places, or fragmentary, or horseshoe-shaped. Itching in hot, damp weather is very troublesome; in fact, the subjects of ringworm can usually foretell the approach of rain by the increase of the itching, the moisture in the atmosphere evidently favouring a more rapid growth of the trichophyton.

A usual situation for ringworm in the adult European in hot countries is the crutch; often, though less frequently, the armpits; much more rarely the breast, abdomen, back, or limbs. In the crutch the disease in hot, damp weather causes a very great amount of itching and positive distress, interfering with sleep, and so tormenting the unfortunate victim that he scratches himself till he bleeds, or irritates his skin into an erythema, or a weeping eczema, or a crop of

[1] In view of the recent discoveries by Sabourand (*Bull. et Annal. de dermat.* November 1892 and February 1893; *Annales de l'instit. Pasteur*, June 1893) as to specific differences in the fungus in different types of common ringworm, it may be necessary to modify somewhat this statement.

boils. Many Europeans suffer in this way every hot season until they learn how to treat themselves, and how to prevent the recurrence of the disease. Frequently the patch of ringworm, which can readily be diagnosed by its red, slightly elevated, perhaps vesiculated, festooned margin, and its scurfy, congested area, creeps down the thighs, over the buttocks, and up on to the skin of the pubes or abdomen. When cold, dry weather sets in the disease ceases to be active or troublesome, all that can be seen then being simply a slightly pigmented, scurfy surface with rather a sharply defined border, on the scrotal aspect of the thighs; on the return of the hot season all the old symptoms recur as before.

In scrapings from the elevated ring forming the margin of the patches the trichophyton can usually be found; some time may have to be expended in the hunt, as in many cases, especially where the parts have been much scratched, it is not nearly so abundant as is the fungus in *Pityriasis versicolor* or *Tinea imbricata;* nevertheless, by perseverance and the free use of liquor potassæ to the scales, it can generally be demonstrated and an accurate diagnosis arrived at.

Ringworm of the scalp in European children in China is rare, and, in my experience, when it does occur, is easily cured—so different in these respects to what obtains in England. In native children, however, a circumscribed epiphytic affection of the scalp is very common; but it is by no means the serious and obtrusive disease that ringworm of the scalp is in Europe. I have not studied the subject sufficiently to be able to pronounce as to the exact characters and habits of the fungus.

Ringworm of the body is easily cured. In cases in which the inflammation induced by scratching is severe, it is as well to attempt to soothe the parts by lead lotion, or the application of some astringent, antiseptic dusting powder, before applying active epiphyticides. In milder cases I have found three or four applications of Vleminckx's solution sufficient. It is thus prepared:—1 oz. of quicklime, 2 oz. of sulphur, 16 oz. of water; boil with stirring till reduced to 10 oz.; allow to stand for a short time, and then pour off the clear sherry-coloured solution of sulphuret. It is better, if the parts are much irritated, to dilute the lotion with an equal part of water for the first application. Before using it the affected skin ought to be well washed with soap and warm water, and dried; while the skin is still soft and moist from the washing, the lotion should be dabbed on with a piece of sponge and allowed to dry. It is better to apply it at bedtime, washing it off in the morning. A more certain, though rather more troublesome remedy, is chrysophanic acid in some form. An ointment of 20 grains to the ounce of

vaseline rubbed in twice a day never fails to cure in two or three days. The patient ought to be warned to cease using the drug as soon as any sign of the characteristic erythema shows at the margin of the diseased patch, and he should be informed of the staining effect on the clothes, and of the swelling of the face sometimes following its incautious use. Besides these there are many native remedies for ringworm, some of them of marked efficiency, and probably superior to chrysophanic acid, inasmuch as they do not stain the clothes or give rise to what may be an alarming erythema. A Burmese plant, called in the native tongue "thembean measley," is said to be a very sure remedy, and gives rise neither to pain nor irritation. Possibly this is the *Cassia alata* which has long been used in the Straits of Malacca as a remedy in ringworm. Two or three of the fresh leaves are crushed between the fingers, dipped in spirit and rubbed in firmly; two or three applications suffice. In China a tincture prepared from some plant—possibly this "thembean measley"—is a favourite remedy with foreigners. Recently an ointment prepared from the leaves of the *Cassia alata*—a plant found in most tropical countries—has been much extolled by certain French writers.[1] An extract is prepared by digesting 100 parts of the leaves in 150 parts of glacial acetic acid and 300 parts of water for eight days, then straining and evaporating. One part of this extract to four parts of lanoline rubbed in twice a day usually cures, even obstinate cases, in three or four days. *Cassia alata* contains chrysophanic acid, and it may be that its virtues as an epiphyticide depend on this ingredient.

For the dark and thick skinned natives by far the best and most convenient remedy is the liberal application of a liniment of iodine of about twice the strength of the present pharmacopœial preparation. One application is usually sufficient.

The European can do much to protect himself from troublesome attacks of ringworm by a little attention to his toilet. A powder of equal parts of boracic acid, oxide of zinc, and starch should be applied with a powder puff daily after the bath to the armpits and crutch. If serge or cloth trousers are used, short cotton drawers, like bathing drawers, should always be worn next the skin and changed daily. If these precautions are taken there is little chance of contracting ringworm.

(*b*) *Tinea imbricata.*—In the Eastern Archipelago and in the neighbouring countries there is a peculiar form of ringworm, absolutely distinct from ordinary *Tinea circinata*, which the writer has described under the name of "*Tinea imbricata.*" In many

[1] A. M Porte, *Arch. de méd. nav.* October 1890.

places it is exceedingly prevalent, and sometimes attacks Europeans as well as natives.

Clinical Features.—In this disease a large area of skin

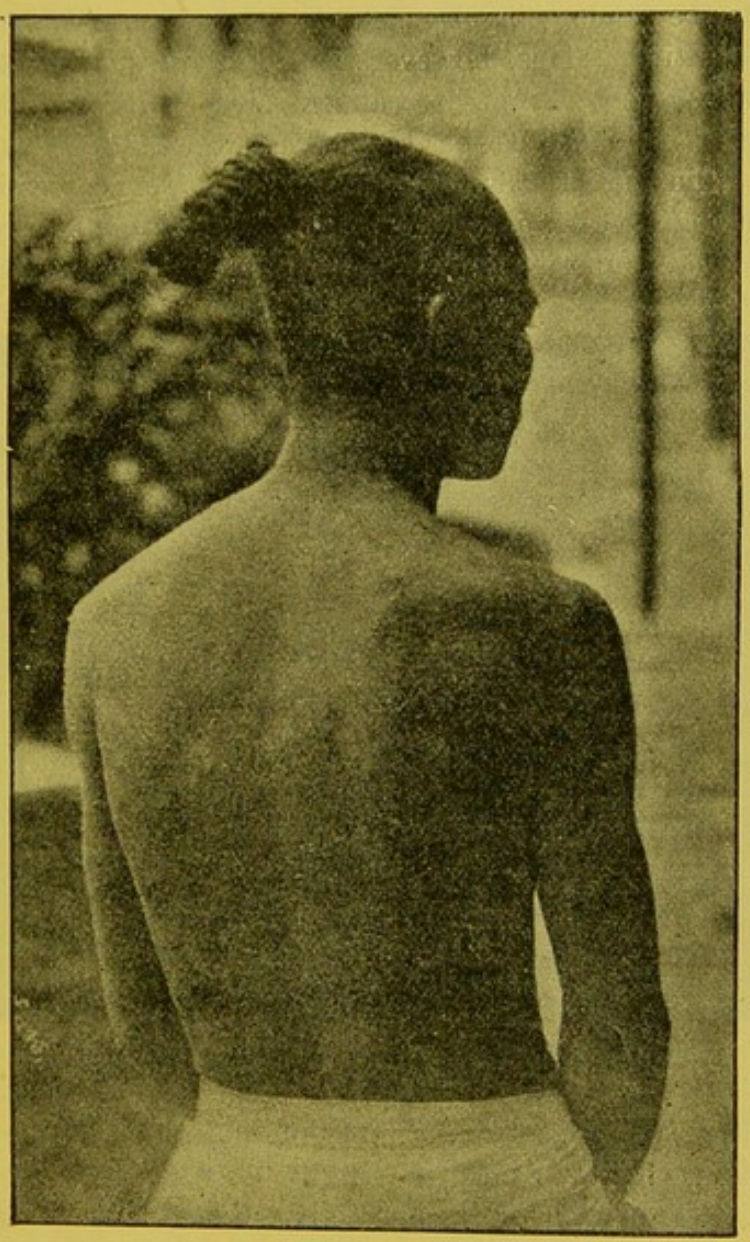


Fig. 80.—*Tinea imbricata.* (From a photograph by Dr. Kerr, Straits Settlements.) The reproduction of the photograph has not been very successful, but if contemplated from a little distance some idea of the pattern of the disease is conveyed by this figure.

(Fig. 80), or even the whole surface of the body, with the exception of the palms of the hands and soles of the feet, and perhaps part of the hairy scalp, is covered with disease. Not a square inch is free from peculiar, tissue-paper-like scales. There is

usually no redness or injection or sign of inflammation of the skin anywhere, nor is there any evidence of irritation—such as marks of scratching—to be detected, although the patient may state that the skin itches sometimes in warm, damp weather. He appears to be undergoing a general, non-inflammatory disquamation or moulting. This, however, is not a passing or temporary phenomenon, such as might happen after some exanthematous fever; for, if interrogated on the subject, the patient will tell us that his skin disease has been in existence and persistent for months, perhaps years, and that it spread over the surface of the body from one or more points.

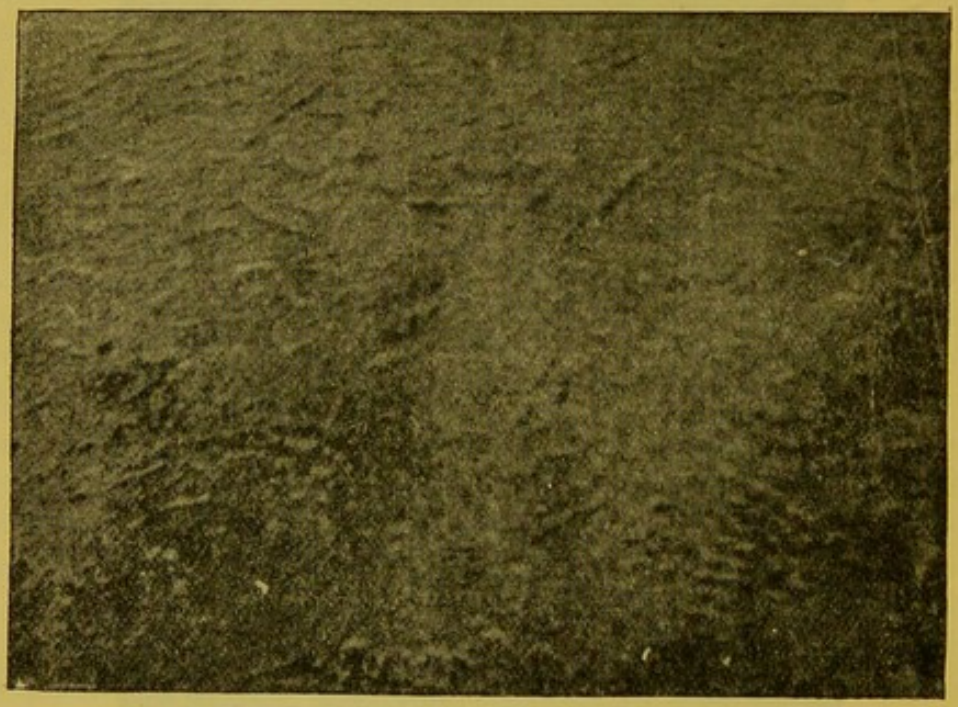


FIG. 81.—*Tinea imbricata.* (From a photograph by Dr. Kerr.)

If we look carefully at one of the numberless scales, we find that it is perhaps half an inch in length by a little more than an eighth of an inch in breadth, that it has a free border, and that it is firmly attached by the opposite edge (Fig. 81). It is therefore in this respect unlike the scales of psoriasis, pityriasis, or ichthyosis, which are generally exfoliated by separation all round their periphery, the last part, as a rule, to lose hold in their case being the centre. These scales, on the contrary, are like surgical flaps—free at one edge and continuous with the body at the other.

If we further analyse the arrangement of the scales we discover a pattern, so to speak. It is as if the disease had advanced in parallel and concentric lines from a great many different centres.

Each line of scales is roughly parallel to the one in front of it and to the one behind it, the lines being from a quarter to half an inch apart. Where one system of concentric circles has infringed on another system the regularity of the pattern is broken and complicated, and it may be difficult or impossible to make out; but the general impression conveyed is such as I describe. The arrangement is something like the rings of light and dark surface on watered silk, or of some kinds of moss on weather beaten rock.

Further, all the scales are arranged so that the free border of each is towards the centre of the circle or system of circles to which it belongs; the attached border is, therefore, towards the periphery.

If we look carefully at the skin underneath the scales, where the ringed appearance is distinct, we observe that it is paler than the general surface; whereas the skin just at the attachment of the scales is rather darker.

Microscopical Examination.—If we detach a scale and place it, after moistening with liquor potassæ, under the microscope, there is no difficulty in seeing a most luxuriant growth of a fungus resembling in many respects the ordinary trichophyton, but differing slightly from it in some details, more especially in its prodigious abundance. The fungus lies layer upon layer, the strings of conidia and mycelial filaments being packed almost as closely as they can lie. One never sees such abundance of epiphytic growth in ordinary trichophyton disease, and I look on this profusion of fungus as one of the distinguishing features of the disease I am describing.

Inoculation Experiments.—We appear then to have to deal with an epiphytic skin disease. If this conjecture be correct, the disease ought to be communicable by inoculation. A good many years ago I made the experiment several times, with the result that, after a sort of incubation stage of ten days, a minute, slightly elevated, brown spot appeared at the site of inoculation. After seven more days this spot had increased in diameter to three-eighths of an inch, and the epidermis had ruptured in the centre and become separated from the underlying skin for some distance outwards. In another day or two the ring thus formed had acquired a greater diameter, and had now a scaling periphery, exactly resembling the disease in the patient from whom the virus, if I may so speak of it, was derived. Furthermore, in the centre of the circle a brown, slightly elevated spot had once more developed. The outer ring continued to expand, and the brown spot in the centre to increase in diameter and its centre to break down; so that in a short time there were two rings of scales, one inside the other. When these two rings had opened somewhat, a third began to form

from another brown spot in the common centre; later on a fourth; and when I thought it prudent, in the interests of the students who kindly lent me their arms for experiment, to kill the fungus, there was generally a series of five concentric circles of scaling epidermis, the outermost of which had a diameter of $1\frac{1}{4}$ inches. This took about thirty or forty days to develop. The rings were, therefore, about an eighth of an inch apart, and appeared to be advancing at the rate of an inch in twenty-five to thirty days. A flake of epidermis, from any part of the rings, placed under the microscope was found to be covered with a fungus similar to that found in the original disease, and it was present in the same luxuriant profusion. It was also evident that the brownish appearance about the skin at the attached edge of the scales was due to the colour of the fungus when seen in dense masses, and not to ordinary skin pigmentation. The brown colour of the central spot from which the rings originated was also evidently owing to the colour of the fungus, just as the fawn colour of *Pityriasis versicolor* is due to the colour of microsporon furfur.

Pigmentation of Fungus.—This brown pigmentation is very readily observed if the scales of loosened skin are removed by prolonged immersion of the patient in a warm bath, and the vigorous use of soap and water and a rough towel. When by these means the scales are removed, the body is seen to be marked by parallel brown lines, which observe a more or less concentric arrangement just as the lines of scales do. The colour of these lines—which are about a tenth of an inch, or thereabouts, in breadth, and often many inches in length—is rather darker than the fawn colour of *P. versicolor*. If the skin of the patient be very dark they may appear lighter than the natural colour of the healthy integument. In Chinese whose skins have been darkened by much exposure, and in the dark-coloured negro races, the spots of *P. versicolor* may appear lighter than the surrounding healthy skin, the fawn colour, which evidently resides in the fungus, concealing the subjacent skin pigment; the same remark applies to *T. imbricata*. The brown colour resides in the fungus, and can be seen with the microscope as little granules in the interior of the mycelial filaments.

Diagnosis.—The following points may be noted as diagnostic between *T. imbricata* and ordinary body ringworm:—

In the first place, ordinary ringworm, though sometimes occupying a considerable area, seldom, if ever, involves the entire surface, or even an entire limb; whereas the scaly ringworm, or, as I prefer to call it, *T. imbricata*, if it has been in existence any length of time,

occupies a very large area, generally a great part of the trunk and much of some or all of the limbs. Second, ordinary ringworm affects, as is well known, the hairy parts of the surface; *T. imbricata* rather avoids these. I observe that Königer[1] says that it appears to cause falling off of the body hair. I cannot confirm or positively deny this statement. Certainly, though *T. imbricata* generally spares the scalp, in the cases in which this part is involved, the hair on it is in no way affected. Third, the active part of an ordinary ringworm is marked by inflammatory swelling of the derma, redness, often vesication, and much itching. In *T. imbricata* there is seldom, if ever, unless under peculiar circumstances, any sign that the fungus is exciting even the most superficial inflammation. Fourth, the ring of ordinary ringworm, in the great majority of instances, is single. After it has expanded very much it may include one or two little points of trichophyton infection, but practically it may be considered as a single ring advancing and opening out, the trichophyton living apparently on some element of the skin which is not quickly reproduced; thus a long interval of time must elapse apparently before the skin over which the fungus has passed can recover sufficiently to support a fresh crop. It thus advances like lupus, or a serpiginous syphilide, eating away and advancing at one border, while abandoning the ground it has travelled over and permitting it to heal at the other. *T. imbricata,* on the contrary, left to itself, never gives up ground it has once occupied. The fungus advances just between the epidermis and rete, destroying their continuity and scaling up the scarf skin, much in the same way as the gardener's spade raises turf. As soon as the epidermis is reproduced the spores which have been left behind germinate and the mycelia attack it, burrowing underneath and throwing it off again and again in never ending series. The distance between the periphery of one ring and the periphery of the succeeding ring is the measure of the time required to reproduce the epidermis. Fifth, if there are any scales about an ordinary ringworm, as a rule they consist of dried inflammatory exudation, or minute branny particles of epithelium. In untreated *T. imbricata* the scales are always present, always abundant, always large—often an inch in length by a quarter or even half an inch in breadth. So abundant are the scales that the air around the patient may be full of them when he takes off or shakes his clothes. It is often with difficulty that sufficient epidermis can be got from ordinary ringworm for microscopical examination, even by scraping the ring with a knife; no knife is necessary to procure specimens from *T. imbricata*, for, with

[1] Virchow's *Archiv*, 1878.

the assistance of a pair of forceps, it is generally an easy matter to pick off the patient in the course of half an hour enough epidermis to fill an ounce bottle. Sixth, in ordinary ringworm the fungus is often hard to find, and is seldom very abundant; in *T. imbricata* it is easily found, and is in great profusion, lying in layer upon layer of interwoven spores and mycelium. Seventh, inoculation with fungus of *T. imbricata* produces a *T. imbricata;* whereas, inoculation with fungus of *T. circinata* produces a *T. circinata.* I have inoculated the same subject with the two diseases simultaneously; twice I have made this experiment, one arm with *circinata,* the other arm with *imbricata,* and the resulting diseases had the distinguishing characters of their respective species. Eighth, *T. circinata* is world wide in its distribution; *T. imbricata* is confined as yet to a limited area.

I am particular in giving the points of contrast and diagnosis between these two diseases, as the authorities in Europe have shown great reluctance in admitting the claims of *T. imbricata* to the dignity of being a specifically distinct disease.

One would think that important evidence for the discrimination of the respective parasites of *T. circinata* and *T. imbricata* would be afforded by the microscope. This is far from being the case. I think there is a difference in the average dimensions of the fungi, and I think that the conidia are differently shaped and somewhat differently arranged; but in both there is great variety, and these varieties insensibly grade into each other. There is no question that in *imbricata* the fungus is infinitely more abundant than in *circinata;* but quantity is not a quality which can be regarded as absolutely distinctive and conclusive. These fungi are not alone in refusing to yield distinctive evidence from their microscopical features. In the case of minute organisms generally, morphological character is always an untrustworthy means of distinguishing species. The bacteriologist, for example, cannot always or even generally discriminate with his microscope one species of bacterium from another; and he must have recourse to a variety of cultivation and inoculation experiments, by watching which and the behaviour of the bacteria in different media he differentiates the various species. So with the fungi of *T. imbricata* and *T. circinata.* It may be difficult or impossible to say from microscopical examination alone that a given specimen is *circinata* or *imbricata;* but by observation of the disease it is derived from, and by an inoculation experiment, we can at once bring out specific characteristics. Nevertheless there are points which we may gather from a microscopical examination—more especially the situation of the

fungus on the under surface of the epidermis, its not implicating the hair, and its extreme luxuriance in *T. imbricata*, which afford very important, if not absolutely reliable evidence for recognition and differentiation.

Geographical Distribution.—The distribution of *T. imbricata* is peculiar. Its home undoubtedly is the Eastern Archipelago; here it is indigenous, and from this region it has spread east and south to many of the islands of the Pacific, and also northwards into China. There is historical evidence of this extension. For example (as pointed out by Dr. Turner),[1] Samoa some years ago was free from this loathsome disease, but a native of Tokelau (Bowditch Island) came to Samoa with the disease on him, and from him it spread and is now firmly established on the island. In the same report (1869) Dr. Turner writes: "It was unknown at Bowditch until about ten years ago, when it was introduced by a man, copper-coloured like themselves, and said to be a native of Tamana (one of the 'Gilbert group'), who landed from a whaler that called there. His name was Peter, and hence the disease is called at Bowditch *le Pita*—the Peter." From this little history we see how the disease has crept from island to island, and how, doubtless, as communications extend, it will continue to spread.

By far the best account of the distribution of the disease in the Eastern Archipelago is that given by Dr. Guppy.[2] This accomplished and very reliable observer gives references to his authorities in proof of the existence of the disease in the following islands: the Solomon Islands, where in some parts two-fifths of the inhabitants are affected; the Florida Islands, where quite one-half are affected; Treasury Islands, where four-fifths of the people are affected; the Elice group; New Guinea and neighbouring islands, Sumatra, Timorlaut, Buru, the Philippines, and the Ladrones. I would supplement this record by adding, on the authority of Dr. Rowell and Dr. Simon, of Singapore, the Malay peninsula; and, on the authority of the late Dr. Storey, of Sarawak, and Dr. Walker, of Sandakan, and other sources—the island of Borneo. Lately, Mialaret[3] has met with the disease in Maré, Loyalty Islands, and in New Caledonia. It is common in the Fiji Islands, and, according to Meerdervoort,[4] is plentiful in Gisser, Calietaroe, Ceram, Ceram Laut, Goram, and the Aroe Islands.

[1] Also by Königer, *loc. cit.*
[2] *The Solomon Islands and their Inhabitants*, 1887.
[3] *Arch. de méd. nav.* 1891, July and October.
[4] *Nederl. Tijdschr. voor Geneesk.* 1859, iii. p. 629.

It has also been reported as having appeared in the Sandwich Islands.

A carefully conducted inquiry by Dr. John Leslie, I.M.S., has resulted in proving the presence of *T. imbricata* in Burmah. In Rangoon jail a systematic inspection of the prisoners resulted in the discovery of four cases. Cases are also reported from Maubin, Tomeghoo, Akyab, and elsewhere in Burmah, and also from Chittagong, Bengal. Dr. Oswald Baker, I.M.S., who has studied the disease in Rangoon jail, remarks that *T. imbricata* seems never to attack the nails; another peculiarity he has observed in Burmah is, that it avoids the scrotum and flexures of the thigh—parts so frequently affected by *T. circinata*. Destruction of the finger nails is a very frequent occurrence here in *T. circinata*. The dry belt in Burmah does not seem favourable to the development of the disease. With the exception of Chittagong, I cannot find that it is endemic, or, rather, that it has been observed in any part of India.

The necessary climatic Conditions.—It seems to me that a warm, damp, equable climate is necessary for its rapid development and spread. Extreme heat, such as sometimes prevails in China, seems unfavourable, and so is the dry cold of the north-east monsoon there; the former seems to stimulate the growth of the fungus too much, until it acts as an irritant, and causes inflammation of the skin, which appears (just as occurs when in greater degree in *T. circinata*) to lead to the death of the fungus; dry cold, on the other hand, checks its spread by drying and hardening the epidermis. The effect of high temperature I once observed in the course of an inoculation experiment I made some years ago. An arm had been duly inoculated and the fungus grew and throve apace, and ring followed ring in regular order; but, the weather suddenly becoming excessively hot, the rings itched and inflamed, broke up into segments, and then finally vanished. Conversely, inoculations do not succeed well in the cold weather when the skin is very dry. An atmospheric temperature of from 80° to 85° F., and a skin kept soft with an insensible perspiration, seem the conditions most favourable for the spread of *T. imbricata*. The fungus is not so hardy as the tricophyton tonsurans, and is more readily killed by inflammation and peculiarities of climate.

Treatment.—The fungus, lying so superficially, is easily attacked and killed by almost any epiphyticide; but, owing to its profusion, and the great extent of surface usually involved by the disease, and consequent saturation of the patient's clothes with fungus elements, relapses very generally occur. In all cases

clothes and coverings ought to be burned or boiled during treatment. In China I was in the habit of applying the linimentum iodi, taking a limb or part of the body at a time. This quickly kills the parasite, and, if the precautions I have mentioned about clothes and coverings are taken, cures the disease. But it is very hard to get a native coolie to part with his venerable rags, and, accordingly, relapses are the rule. It appears that sulphur ointment is found efficacious in the labour ships of the South Pacific. As I have said, any epiphyticide will suffice, provided it is thoroughly applied, and clothes and coverings destroyed. In one patient I tried the effect of a bath of sulphuret of potassium; although persevered with for a good many days, it did not effect a cure, and I had to have recourse to the linimentum iodi treatment, which I have generally found to be the surest, safest, and most acceptable remedy for all forms of trichophyton disease in dark-skinned races. The liniment ought to be double the strength of the present pharmacopœial preparation. M.M. Bonnafy and Mialaret describe a treatment by sulphur fumigation adopted in Fiji which seems to be fairly successful; but they say as many as twenty fumigations, practised at intervals over a period of two months, may be necessary; this is by far too tedious a process.

An ointment prepared from the leaves of *Cassia alata*, as described under *T. circinata*, would probably prove effective. A great service would be done to the natives of those countries in which *T. imbricata* is so prevalent, by any one who would teach them to cultivate this plant and instruct them in its use.

2. Pinta.

Pinta disease, spotted sickness, mal de los Pintos, tina, carathès, carate, cute, cativi, quirica, are some of the names used to designate this disease. Most of them have reference to the peculiar appearance of the patients. The skin is piebald; patches of white, or red, or blue, or black colour are splashed, as it were, all over the body, giving it in some cases a peculiar and most grotesque appearance. The disease is further characterised by a peculiar and very offensive odour, variously compared to the smell of a mangy dog, damp and dirty linen, musk, cat's urine, and so forth. Desquamation and itching of the discoloured patches are other marked features. The disease is not attended with any constitutional symptoms, nor any danger to life.

Geographical Distribution. — Pinta is confined to the inhabitants of low-lying, hot, damp regions, especially along river

banks, in Mexico, Central America, Venezuela, Colombia, and perhaps to one or two places in Peru, Chili, and Brazil.[1] It is not equally distributed thoughout the whole of this vast area. It is unknown, unless as an imported disease, in many places. Probably the personal habits as regards dirt and cleanliness of the different communities and tribes have much to do with its distribution. It is rare among the whites and well-to-do people; the negro, likewise, is comparatively exempt. The Indians and poor half-castes are the most frequently affected. According to Gastambide,[2] who has written one of the best accounts of pinta available to the ordinary reader, quite 9 per cent. of the inhabitants of some districts are affected. So common was it in Mexico in 1826 that M'Lellan saw a regiment of soldiers there which was entirely composed of "pintados." In Honduras, Ariaga[3] says that the "mandrados," as they are called in that country, are exempt from military service. It is evident that pinta is a very common and well recognised complaint in these countries.

Symptoms.—The earliest accounts we have of pinta are very confusing; probably they include cases of leprosy, leucoderma, pellagra, ordinary ringworm, and other skin affections associated with scaly discoloration of the skin. Hence the deaths which were so frequently attributed to it. It is now known to be a purely local affection, dependent on the growth of a special form of fungus in the superficial layers of the integument.

Pinta may commence and continue as a single spot; more often several points are attacked nearly simultaneously, the parts affected in the first instance being the face, hands, or any exposed surface. A small patch of white, red, blue, or black discoloration is seen on the

[1] Professor Magalhães—to whose courtesy I am indebted for much information and assistance in preparing my contributions to this work—writes me, in reply to inquiries about pinta, that he has failed to get any reliable information about this disease as it exists in Brazil. It is probably, therefore, confined to the interior provinces of the country. He mentions a curious circumstance. He says: "Among the objects exhibited in the Medical Exhibition held in Rio some years ago, there was a picture of a disease characterised by blue patches on the skin. The painting was very old. I was informed that it came from somewhere in the Amazon regions. I did not see the picture, but my very competent informant told me, that the patches represented seemed so strange to him that he failed to understand what might have been the disease the artist had intended to represent. The catalogue did not mention the picture; but the organiser of the exhibition, Dr. C. Costa, librarian of the Faculty, confirms the fact of its having been exhibited. He explains the circumstance of its omission from the catalogue; he informs me that a number of irrelevant objects were exhibited in the cases and on the walls of the exhibition simply as ornaments. Unfortunately, it has been impossible for me to trace this picture; the existence of which, in our old collections, indicates a disease, characterised by blue spots on the skin, in individuals from the interior of the country, and has made me think of pinta, which, according to the description of Hirsch, sometimes presents a blue colour."

[2] *Presse Méd. Belge*, 1881.

[3] *Arch. de méd. nav.* 1885, No. 44, p. 464.

skin; this gradually enlarges, the surface becoming scurfy, and, after a time, scaly, and very itchy, especially on first going to bed. Gradually the patch enlarges, assuming various shapes. Other spots may form in the neighbourhood, and may merge into the original spot. In this way large patches are formed of discoloured integument. At first the various spots are usually all of one colour, whatever that may be; but after a time, and in nearly every case of long standing, differently coloured spots are developed, at the same time a particular spot is never observed to change colour, that is, a red spot always remains red, a black, black. Gomez[1] says that the white spots arise from the coloured as if from loss of pigment from prolonged irritation; Gastambide says they are independent of this, arising in previously healthy skin. The margins of the diseased areas may be sharply defined, or they may shade off gradually into the healthy integument; in the case of the white spots there is sometimes observed an increase of the natural pigment around the margin. Any part or all of the surface of the body, with the exception of the palms of the hands and soles of the feet, may be attacked. If extensive areas are involved the effect is often very grotesque, the unhappy victim looking like the painted clown of a circus. When the scalp is involved the hair becomes white and thin, and, in the end, falls out. At first the patches feel dry and harsh; later, probably on account of the continual scratching, they may become greasy, cracked, and perhaps ulcerated. Gomez says the white patches are not scaly nor itchy. Sensation and the functions of the sudoriparous follicles are not in abeyance; thus clearly differentiating pinta from leprosy.

There may be said to be two types of the disease—the superficial epidermic and the deep epidermic. The black and blue patches constitute the superficial epidermic, in which the disease seems to be more superficial. This variety spreads, as a rule, more rapidly, and at the same time is more easily cured than the deeper epidermic variety, which is either white or red, and involves the rete and deeper layers of the epidermis.

Pinta lasts for an indefinite time, often for a lifetime. It is decidedly contagious, spreading through a household if the members are not cleanly in their habits. It attacks both sexes and any age.

Pathology.—If one of the scales, which are flat, dry, polyhedric, thick, and dirty white, be placed under the microscope and moistened with liquor potassa, a number of black spores and white, highly refracting mycelium can be detected. The spores are round

[1] *Thèse de Paris.*

or oval, the spherical measuring about 8 μ in diameter, the oval 6 to 8 μ by 10 to 12 μ. They consist of a transparent capsule containing a yellowish fluid, in which float a number of black granules. The mycelial filaments are short non-branching, and taper from a broadish base to a blunted point, and are each attached to a single spore, like the stalk to a cherry; they measure from 18 to 20 μ in length by 2 μ in thickness. Gastambide says the fungus in the black and blue varieties, as in *Pityriasis versicolor*, is very superficially located; but that it is more deeply placed in the red and white. No satisfactory explanation has been given of the most striking feature of the disease—the different colours of the patches. The black, and perhaps the blue colour, is readily accounted for, just as the fawn colour in the case of *P. versicolor* or *Tinea imbricata*, by the profusion of the pigmented fungus. Possibly there are red and white varieties of the epiphyte which would account for the other kinds of coloured patches; or, it may be, that the red variety owes its colour to a congested state of the derma and the white to leucoderma, caused by the prolonged irritation produced by the fungus.

Treatment.—Several Mexican and Central American writers recommend the internal administration of mercury, iodide of potassium, sarsaparilla, and a variety of other so-called alterative drugs. It is difficult to understand in what way the internal use of these could possibly destroy a fungus growing on the surface of the skin. The most efficient remedies one would suppose to be those which have been found to be of use in other epiphytic skin diseases, such as chrysophanic acid, liniment of iodine, and preparations of sulphur. Old clothes ought to be boiled or burned, and domestic and personal cleanliness insisted on.

SECTION II.—SKIN AFFECTIONS CAUSED BY ANIMAL PARASITES.

1. Craw-Craw.

This term is applied by the natives of many parts of the West Coast of Africa to any itching, papulo-pustular eruption, and must therefore include several distinct forms of skin disease. Although Winterbottom[1] writes very briefly about "kra-kra," he mentions certain drugs reputed to cure it. The only detailed account we have of a specific form of this disease is that given in the *Lancet*, February 1875, by Dr. John O'Neil, in which, under this name, is

[1] *An Account of the Native Africans*, etc., 1805.

described a papulo-pustular affection said to be common in certain parts of the West Coast of Africa. He writes, apparently, of natives in the vicinity of the Addah Fort Hospital.[1]

According to O'Neil, this particular form of craw-craw at first sight suggests scabies; but, unlike what would happen in scabies, on the subject of it visiting a cooler climate,—such as the Cape of Good Hope,—the eruption and intense pruritus decline; only, however, to relapse on return to the hot, damp atmosphere of the West Coast. He examined six cases, all negroes. He describes the papules as being scattered over the limbs and body, arising singly and at irregular intervals, or arranged in rings. In two days the papule, he says, becomes a vesicle, in two more a pustule. If the top of the papule is shaved off with a sharp knife, and, after moistening with water, is placed under the microscope and magnified 100 diameters, a minute, filaria-like organism is seen wriggling about with great activity. After a few minutes this activity slows down, and, gradually ceasing to move, the little parasite stretches itself out and dies, apparently killed by the water. O'Neil's drawings represent an organism somewhat resembling *F. nocturna;* but that it is not this parasite is evident from the dimensions given,—$\frac{1}{100} \times \frac{1}{2000}$ inch,—and from the presence of two black markings near the cephalic end. O'Neil remarks that if the section of the papule be made at a sufficient depth, five or six of these filariæ may be seen in the field together.

Craw-craw is said by the natives to be highly contagious, to appear after an incubation stage of three days, and to be incurable by sulphur applications.

Writers who have commented on this subject generally allude to a case observed by Silva Araujo in Brazil, and by him diagnosed craw-craw.[2] On carefully reading this case I fail to see that it was other than one of ordinary *F. nocturna* disease—chyluria, with lymph scrotum, and a certain amount of skin irritation. In the account Araujo gives of the associated parasite no mention is made of the peculiar and distinctive markings mentioned by O'Neill; and as no other case of craw-craw seems to have been subsequently observed in Brazil, it is premature to conclude that the craw-craw of O'Neil is endemic in that country.

The only well-authenticated instance of a similar affection occurring in man is that recorded by Professor Nielly under the title "dermatose parasitaire."[3] This was the case of a French lad who

[1] Clover Expedition.

[2] *Arch. de méd. nav.* 1875 and 1878.

[3] *Arch. de méd.* April 1882; *Arch. de méd. nav.* No. 37.

had never been abroad, and who was attacked with an intensely itchy, papulo-vesicular eruption like ordinary scabies, in the pustules of which Nielly found a parasite very like that discovered by O'Neil in African craw-craw. It had the two peculiar cephalic markings, —apparently produced by the thick walls of a pharynx,—a well-defined alimentary canal, and very rudimentary organs of generation. Nielly distinctly states that the skin parasite was at one time associated with the presence of a nematoid embryo in the blood. It is probable, therefore, that the skin organism was an advanced form of the embryo worm in the blood, and that both were the offspring at different stages of development of a mature worm lodged somewhere in the tissues. The measurements of Nielly's parasite—named by Blanchard *Rhabditis Nielly*—were 333 μ by 13 μ.

It is evident that if O'Neil's craw-craw is of the same nature as Nielly's dermatose parasitaire, this form of disease cannot be so directly contagious as the negroes state; and that an incubation period of much greater duration than three days—that assigned to craw-craw—is necessary.

In this connection it is interesting to note that some of the lower animals are subject to similar affections. The parasitic dermatorrhagia and the "summer sores" of horses are of this nature. Probably the granular dermatitis of the horse, known in India as "Barsatti," is of a similar character; and so, likewise, are certain cutaneous filariases of the dog.[1]

Information is much wanted on the subject of craw-craw, and medical men on the West Coast of Africa would confer a service to helminthology and pathology by continuing the research commenced by O'Neil and Nielly. Particular attention ought to be given to the presence or absence of hæmatozoa in the blood in all cases of this and similar diseases. It is just possible, considering the geographical distribution of craw-craw and *F. perstans*, that the immature parasite in the papules of the disease we are considering may turn out to be an advanced stage in the development of this parasite. The structure and habits of *F. perstans* suggest that this animal leaves the blood vessels spontaneously; and it may be that it then lodges in the skin, where it undergoes a degree of development. By provoking a certain amount of irritation and pustulation, it causes the itching and scratching which eventuate in its liberation—whereby it obtains further opportunities to advance in development. Be this as it may, the parasite of craw-craw is certainly not *F. nocturna*, which never, while in the human body,

[1] For interesting information on these subjects, see Neumann's *Parasites and Parasitic Diseases of the Domesticated Animals*, translated by Fleming.

becomes possessed of an alimentary canal; nor is it a mature worm, as in this case the organs of generation would not be simply rudimentary. It is evident that the parasite of craw-craw is a transition form of some parasite on its way to further development.

2. Guinea-Worm.

Although instances of this form of helminthiasis are sometimes seen in the large maritime towns of this and other European countries having a large and direct sea-borne trade with the East and with Africa, guinea-worm is not endemic in any part of Europe or of North America. As an endemic affection it is strictly confined to certain of the warmer regions of Asia, Africa, and a very limited part of South America.

Guinea-worm is known under a variety of names, such as *dracunculus*, *F. medinensis*, *F. dracunculus*, and *dracunculus medinensis* (Cobbold); French writers often allude to it as "dragonneau." The particular form of helminthiasis it gives rise to is sometimes indicated by the term "dracontiasis."

The history of guinea-worm dates from remote antiquity. Indeed, the "fiery serpents" which so troubled the Israelites at one time during their wanderings in the wilderness of Sinai are believed by some writers—Bartholin, Kuchenmeister, and others—to have been none other than this parasite. Plutarch evidently refers to it as being endemic in his time in the countries bordering on the Red Sea; and Galen and other writers of antiquity make mention of it under a variety of names.

Geographical Distribution.—At the present day it is endemic in many parts of India; it is also common in parts of Persia, and exceedingly common in certain districts in Turkestan and Bokhara. It is met with in Arabia, in Upper Egypt, Abyssinia, and the Soudan. On the West Coast of Africa it is so common in certain districts that nearly every negro coming off to ships is affected with it. It seems probable that prior to the introduction of negro slaves the American continent enjoyed an absolute immunity; but the parasite, having been imported from Africa, has taken root in some districts, and is now to be found endemic in Curaçao, Demerara, Surinam, and in parts of the Brazils, notably in the country around Feira de Santa Anna, in the province of Bahia (da Silva Lima).

It has long been known that in the countries in which guinea-worm is endemic it is by no means generally or equally distributed; on the contrary, that it is strictly confined to certain limited

districts, often to the communities using the water of particular wells or pools, their immediate neighbours perhaps enjoying complete immunity. The explanation of this apparent caprice lies in the recently discovered fact that a certain species of fresh water cyclops, having apparently a very capricious distribution, is a necessary factor in the life history of the filaria; in fact, that the filaria at one stage of its existence must pass through the body of this minute crustacean, and undergo a certain metamorphosis therein, before it is qualified for life in the tissues of its final host. Consequently the distribution of the filaria is bound up with and dependent on the distribution of the particular species of cyclops which thus acts as its intermediary host.

Natural History.—*Lower Animals sometimes affected.*—A not unimportant fact, from the prophylactic point of view, is that the dracunculus is not confined to man, but has, not unfrequently, been found in the dog and horse. According to Avenzoar and de Marchais, the ox is not exempt—indeed the former calls dracontiasis "the disease of oxen."

Possibility of a Plurality of Species being included under the term "Guinea-worm."—Before proceeding with a short account of this parasite, I would point out that it is just possible that there may be more than one species of dracunculus affecting man. The length of African specimens is much greater—according to Bastian, generally attaining about 6 feet—than specimens from India, which rarely attain more than half that length. Hence, perhaps, the many discrepancies in the various descriptions of the anatomy of this parasite.

Description of the Mature Parasite.—The female guinea-worm (Fig. 82) alone is known with certainty; it cannot as yet be positively asserted that the male worm has ever been seen. Charles, in a recent number of the *Scientific Memoirs by Medical Officers with the Army of India*, part vii., describes a peculiar appendage in connection with two female dracunculi found in the sub-peritoneal tissue of a dissecting room subject, and which he believes to have been the male dracunculus *in coitu*. In the specimens referred to, filaria-like structures sprang from an orifice in the musculo-cutaneous wall of the female worm, and by gentle traction could be pulled out for some distance. Unfortunately, Charles' description and drawings are not sufficiently detailed; they leave the reader in doubt as to the exact nature of these appendages. Possibly they were male dracunculi; but, so far as the description and drawings go, it is also possible—as a writer in the *British Medical Journal*, 19th Nov. 1892, points out—that they were produced in both instances by

prolapse of the uterus through a wound of the integuments, a by no means rare occurrence in slightly mutilated specimens of the more delicate filariæ.

Immature specimens of the dracunculus are occasionally found in the sub-peritoneal tissue, and in the cellular tissue of the limbs and body, and may vary in length from a few inches to as many feet. The mature worm, with which we are more perfectly acquainted, is a long, milky-white, slender, cylindrical animal, having a uniform thickness of about the tenth of an inch. Its appearance is often compared to a thick fiddle-string. It varies in length from about 12 inches to as many feet. These are the extreme dimensions, which, owing to the elasticity of the structure of the worm, are very apt to be exaggerated; by a little traction it can be extended to nearly twice its natural length. In forty specimens measured by Ewart the shortest was 12¾ inches, the longest 40 inches.

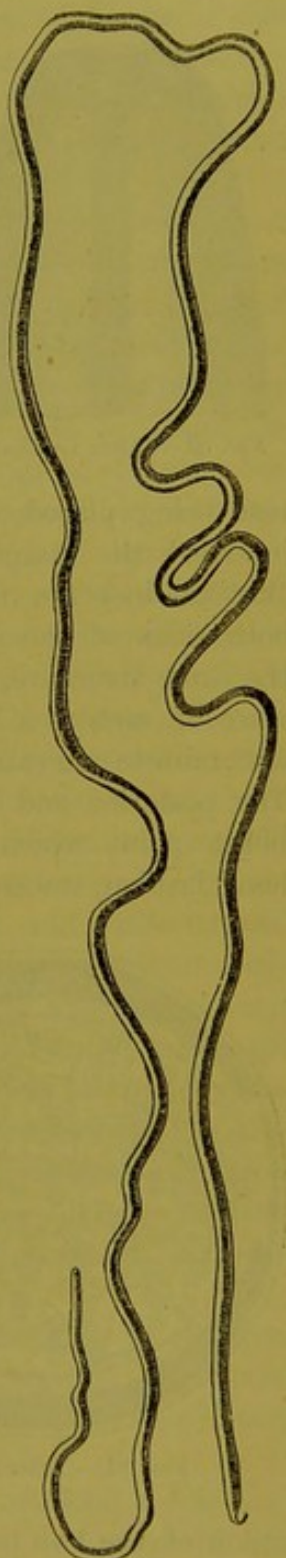


Fig. 82.—(After Leuckart.)

It is somewhat singular that the recorded measurements of the mature guinea-worm should vary so much. Slight differences in this respect occur in all organisms, but not to the extent attributed to dracunculus. I am inclined to think that much of this discrepancy is to be accounted for, not by the caprice of nature, but by the condition of the uterus of the parasite at the time of extraction. I shall presently show that during parturition the worm shortens herself. Accordingly, before this process commences she must be considerably longer than after it has concluded; consequently, I think it probable that the longer measurements recorded were made in specimens extracted before parturition commenced, the shorter from specimens in which this process had been partly or wholly completed.

The head end (Fig. 83), after a very short and slight taper, is rounded off abruptly, terminating in an oval, somewhat irregular surface—the "cephalic

shield." This so-called shield has its long diameter lying transversely as regards the dorso-ventral axis of the worm. At its centre is the triangular buccal orifice; this leads to the attenuated alimentary canal, which, running along the whole length of the animal, terminates near the tail on the ventral surface by becoming attached to the muscular integument; it does not open externally by an anus. Close to the buccal orifice, one on its dorsal and one on its ventral margin, are two well-defined papillæ; and at the circumference of the shield are six other equidistant and similar, though smaller, papillæ; two larger than the rest being placed, one on either side, where the two lateral lines intersect the margin of the cephalic shield; and four smaller—Bastian does not mention these—arranged symmetrically, one on both sides of the dorsal and ventral lines. These papillæ have all the same structure, and are believed to be sensory organs; at the apex of each is a well-marked depression, at the bottom of which are minute elevations to which nerve filaments are distributed. The posterior end of the parasite bevels abruptly, terminating in a blunt point which, though sometimes straight, is generally bent, hook fashion, towards the ventral surface (Fig. 82).

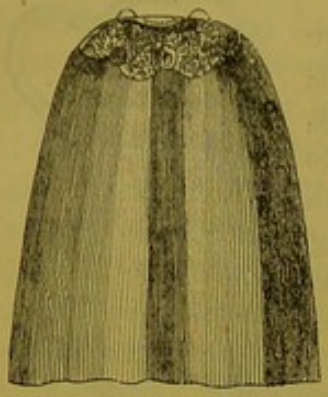


Fig. 83.—(After Leuckart.)

From within an inch of the head to close to the tail the musculo-cutaneous coat is completely filled by a long, tubular, embryo-stuffed uterus (Fig. 84), the alimentary canal — empty and flattened—being thrust to one side. At either end of the uterus there is a minute tubular appendix, the remains of the ovarian tubes. There being two ovarian tubes proves that originally, at an earlier period of the animal's history, the uterus was a double organ, which, at the union of the two horns, probably somewhere about halfway between the head and tail, had opened into a vagina; no trace of vagina can be detected in the mature parasite, however.

Fig. 84.—(After Leuckart.)

Process by which the Embryos are liberated.—The process by which

the millions of embryos, with which the dracunculus is crammed, quit the parent worm and obtain their liberty, and an opportunity to advance in development, has never hitherto, so far as I am aware, been properly explained. As I have had an opportunity lately of studying the subject, and as it is one not only of much biological interest, but of great practical importance in its bearing on treatment, I shall endeavour briefly to describe it.

It has been mentioned that in the course of her development and growth, the vagina, with which the young filaria is doubtless provided at the time of impregnation, and at the earlier stages of her parasitic life, disappears; no such organ has ever been found in the mature worm, although often and carefully sought for by competent observers. Thus the channel through and by which the young of most animals escape is entirely wanting in dracunculus. It is evident, therefore, that the process of parturition in her case must be of a somewhat anomalous character.

Some have suggested, or would have us infer, that the young filariæ obtain their liberty only on the occurrence of decomposition of the body of the parent worm after she has quitted, or been removed from, the tissues of her human host. This certainly seems a clumsy process for nature to adopt; it is certainly not the normal, or ordinary one. For, if we inspect a dracunculus which has been slowly expelled spontaneously, or which has been successfully extracted by winding out, we find that, with the exception of the last few inches of her body, she is shrivelled up and withered, and that any embryos she may contain are in the same condition, and dead. In many such specimens, even if we select the freshest and plumpest part of the caudal end for examination, hardly an embryo can be found in her; the enormous swarm, which we know must have occupied the uterus at one time, has almost entirely disappeared. Consequently, we are driven to infer that the embryos must in some way have escaped from the parent filaria before she herself commenced to leave the host,—that is, before the extruded and projecting part began to shrivel and wither up.

Some time ago I had an opportunity of observing in progress what I believe to be the normal process of parturition in dracunculus. What I then saw confirms in great measure the description given by Forbes,[1] now nearly sixty years ago.

When the embryos of the guinea-worm have attained a degree of maturity qualifying them for independent life, the head of the worm approaches the surface of the body of her host. Arrived beneath the skin she bores a minute hole in the derma, sparing the

[1] *Trans. Med. and Phys. Soc. of Calcutta*, vol. i.

epidermis. Then, either in virtue of some irritating secretion, or as a consequence of irritation set up by the mere presence of her head in this situation, a blister forms over the small hole she has made in the derma, raising up the superjacent epidermis for some distance around. After a time the contents of the bulla so formed become turbid, and in five or six days it breaks, or is ruptured, revealing a circular excoriation or ulcer having a minute hole, large enough to admit a fine probe, at its centre. Generally at this stage, and it may be for days or weeks thereafter, nothing of the worm herself is visible. If, now, a stream of cold water—such as can be most conveniently expressed from a sponge—is made to fall on the skin near the ulcer, but not on it, for that would conceal what ensues, one of two things may be observed. After the water has been running for some seconds, a minute quantity of clear fluid may be seen to rise up in the little hole in the centre of the ulcer. Presently the character of this fluid changes to a grey, grumous appearance, and, continuing to well up, it spreads over the surface of the sore. If a little of the fluid be taken up with a pipette, transferred to a slide, and examined under the microscope, it is seen to consist principally of a seething mass of active, wriggling embryo dracunculi. The quantity of discharge amounts in all to a droplet only, and soon ceases to flow, even although we continue to pour water on the neighbouring integument. But if we wait for an hour or two, and repeat the douche, the same welling up of embryo-laden fluid recurs, and continues for a second or two, again to cease abruptly. In other instances, and at another time in the same case, instead of fluid welling up on the application of the douche, a tiny, smooth, cylindrical, tense, shining, pellucid tube, perhaps about the twentieth of an inch in diameter, like a miniature sausage, is slowly extruded from the orifice in the centre of the ulcer. On its first appearance, the contents of the little tube are quite limpid; but, as it increases in length and becomes more tense, a milky-white, opaque material is seen to enter it, and quickly pervade its entire length. It then looks as if the head of the worm had been shot into the tube. This, however, is not the case. In a few seconds, and after perhaps half an inch, or even as much as an inch of tube has emerged, the free end ruptures, and the tube collapses into an almost invisible fibre, its contents escaping over the skin. Nothing remains of the tube but a ragged little fibre which can be traced into the hole in the centre of the ulcer, which has again become visible and gaping. On examining with the microscope the material which had escaped from the ruptured tube, it is seen to be almost entirely made up of

living dracunculus embryos. After the lapse of a few hours, a fresh protrusion and fresh rupture of the tube can be provoked; and so on, again and again, during many days, and until the entire contents of the worm have been evacuated. In other instances the source and nature of this little tube is apparent. Forbes, I believe, was the first to describe this; his description is very graphic and nearly correct. In certain cases of guinea-worm, which he had the opportunity of watching from the time the initial bulla began to form to their termination, in several on opening the bulla the head of the guinea-worm was seen to be protruding through the hole in the centre of the ulcer; and he also perceived, he says, a minute tube hanging from the free end of the worm. By pouring water on the ulcer the tube became distended and elongated, just as I have described above; by and by it discharged its contents,—dracunculus embryos,—also just as described above. Forbes says that, after discharge of the embryos, the tube is withdrawn into the parasite. This I am convinced is a mistake. The tube is not retracted, but shrivels up on collapsing. Each fresh discharge of embryos is preceded by a fresh protrusion of tube, the shrivelling up of which, after discharge of contents, effectually and permanently seals it up as far as its point of emergence from the worm.

This tube is the uterus of the dracunculus, protruded in response to the stimulus of cold water, the presence of which in its vicinity is so singularly and—considering the requirements of the embryo—so opportunely recognised by the parasite. Thus does nature get out of the obstetric dilemma which the absence of vagina in the parturient dracunculus entails. The mouth, now no longer required for purposes of nutrition or locomotion, is utilised as a vagina; through this the thinned uterine tubule is prolapsed. The contraction of the thick musculo-cutaneous wall of the dracunculus serves a double purpose, for it not only expels the uterus and its contents, but at the same time shortens the worm and decreases her diameter, thereby facilitating her escape or withdrawal.

Description of the Embryos.—The embryos (Fig. 85)—which can be readily obtained for microscopical examination by provoking their discharge in the manner I have described—measure, according to Cobbold, on an average $\frac{1}{30} \times \frac{1}{1000}$ of an inch. As first pointed out by Robin they are not cylindrical, but distinctly flattened. They taper towards the head end, and terminate posteriorly in a long, slender, sharply-pointed tail (Fig. 85, *h*). The tail, measuring from the commencement of the taper, is about two-fifths of the entire length of the animal. The integument is transversely and deeply striated, and is marked just where the body begins to slope off into

the attenuated tail by two depressions, one on either side, into which two narrow-mouthed sacs open (Fig. 85, *e*); the lips of these sacs are sometimes found everted after the death of the embryo. These organs are not always visible in newly procured embryos. They are more readily seen in languid specimens, and are particularly apparent in embryos killed by instillation of acetic acid below the cover-glass. The head end of the embryo is abruptly rounded off, its centre being marked by a minute three-lipped[1] mouth, which leads to rather a wide alimentary canal (Fig. 85, *b*, *c*, *d*), running back three-fourths of the way to the caudal sacs; so far as I have been able to make out it terminates cæcally; Bastian and others, however, say it terminates in a transverse slit—the anus. Surrounding the intestine, and stretching from the termination of the œsophagus to either immediately in front of, or immediately behind, the caudal sacs, is a peculiar, brown, granular-looking substance, dotted with bright shining, nucleated cells, regularly and diagonally arranged. This substance fills the posterior half of the embryo, and is rounded off posteriorly, not entering the tail.

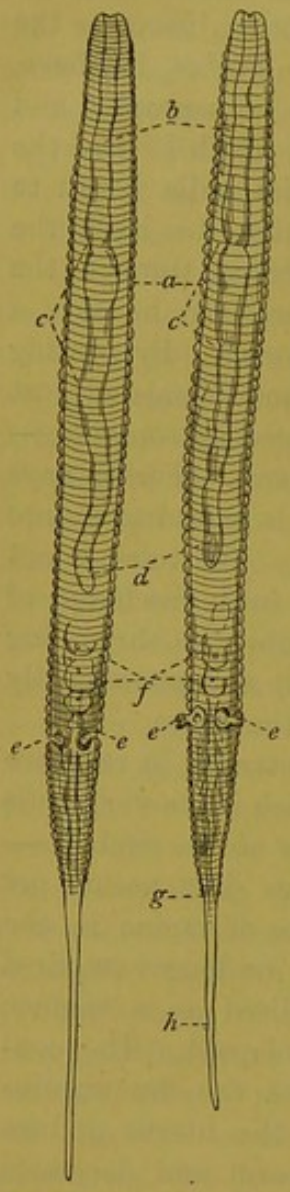


FIG. 85.—(After Bastian.)

The embryo exhibits remarkable activity and vitality; it will keep alive in water for six days; in dirty water, or moist earth for much longer,—at least fifteen or twenty days,—and can even be resuscitated after comparative desiccation by moistening with a little water. Its mode of swimming resembles very much that of a tadpole. It starts forward suddenly, and then, after swimming a second or two, pauses as suddenly, with its body stretched out and perfectly motionless. After a second or two it again proceeds to swim about, again pauses, and so on. In swimming the long diameter of the

[1] For a more detailed account of the structure of the guinea-worm the reader is referred to Leuckart's *Parasites of Man*, Blanchard's *Traité de Zoologie Médicale*, and other systematic works on helminthology. Busk (*Trans. of the Micros. Soc.* vol. ii. p. 80, 1846) and Carter (*Trans. Med. Phys. Soc.* Bombay, 1853 and 1861; *Ann. of Nat. Hist.* 3rd ser. vol. i. 1868; *ibid.* vol. iv. 1859) have written papers on the subject; the most elaborate account, however, of the anatomy of this parasite is probably that by Bastian in the *Trans. of the Linnean Society*, vol. xxiv. 1863-64.

body is vertical, but when it pauses in its movements it tends slowly to roll over, so that the flat and broader surface is exposed. It swims by lashing the tail from side to side with a corresponding undulatory movement of the fore part of the body. When it dies it is coiled up in one or two turns, the tail sticking out straight and not sharing in the curve.

Views regarding future History of the Embryo.—Formerly many

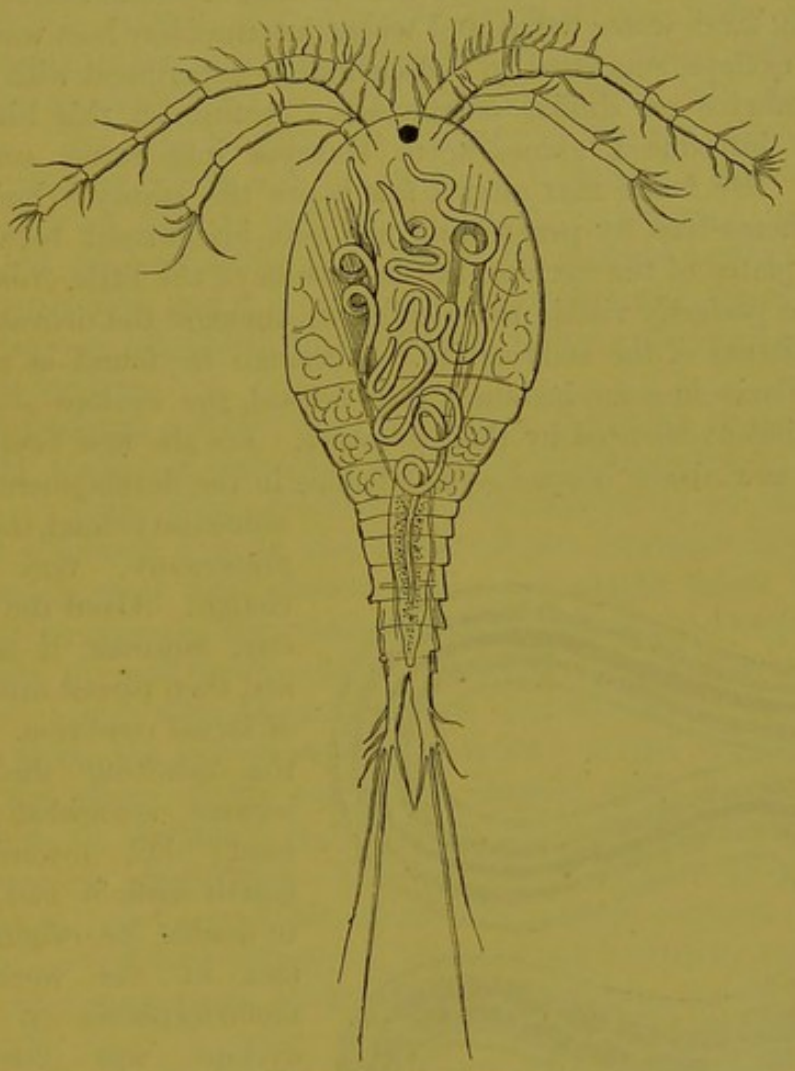


Fig. 86.—(After Leuckart.)

speculations were indulged in as to the subsequent history of the little embryo on leaving the uterus of the parent. Some supposed that after undergoing a degree of development in water or mud it again got access to the human body, either in drinking water or by penetrating the skin of bathers; certain statements about the frequency of guinea-worm in the backs of water-carriers seeming to give countenance to the latter hypothesis. Experiments by feeding

and inoculation were even tried in support of these hypotheses, but without success.

The Intermediary Host and Metamorphosis.—The Russian naturalist, Fedschenko, has shown that the first step towards development, after the embryo has escaped into water, is made in the body of a certain species of fresh-water cyclops (Fig. 86) found in the places in which guinea-worm is endemic. Leuckart, remarking the resemblance the guinea-worm embryo bore to that of *cucullanus*, a parasite attacking fresh water fishes, and whose intermediary host was known to be a cyclops, suggested to Fedschenko to experiment with dracunculus embryos on similar crustaceans. Acting on this hint, Fedschenko introduced guinea-worm embryos into water containing cyclops. He found that after a few hours the embryos had transferred themselves, by penetrating the soft integument between the ventral plates of the cyclops, to the interior of the little crustaceans, and were perfectly visible there coiled up amongst the delicate transparent tissues of the body cavity. Although he found as many as twelve filariæ in some instances thus housed, the cyclops seemed in no way inconvenienced by their presence. For the first twelve days in their new abode, beyond some advance in the development of the alimentary canal, the filaria underwent very little change. About the twelfth day, however, it moulted, and then passed into a sort of larval condition. After the moulting the filaria became somewhat shortened; but towards the fourth week it had grown to double its original size, and in five weeks the metamorphosis, so far as cyclops was concerned, was completed (Fig. 87). The mouth, anus, and alimentary canal had become still further developed, and traces of organs of generation were visible. The tail, too, had become changed in shape; it was now shortened and blunted, and its end was tripartite, like that of the larva of *cucullanus* in cyclops, and of *F. nocturna* in the mosquito.

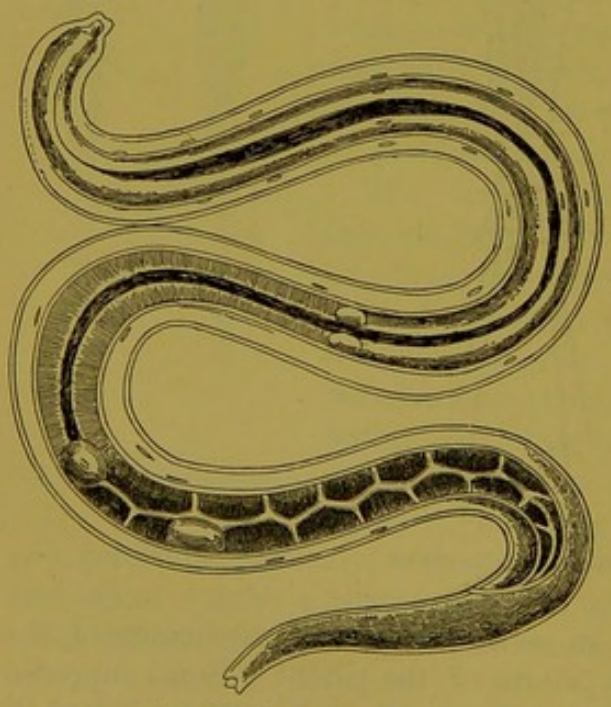


FIG. 87.—(After Leuckart.)

Process of Infection.—In this larval condition, lying in the body of the cyclops, it is supposed that the filaria waits an opportunity of being conveyed in drinking water to the stomach of a human host or of some other animal; whence—after obtaining its liberty on digestion of the tissues of the cyclops—it bores its way into the tissues. When or where the sexes come together, and what are the subsequent steps in the development of the female, and what becomes of the male worm, are all alike as yet unknown.

Incubation Period.—The next we know of the filaria is as an animal many inches in length, embedded in the cellular tissue of the host, with an enormously developed uterus packed with millions of embryos. The time between the entrance of the larval dracunculus and the time at which she has attained maturity is variously estimated at from nine to twelve months, or longer, even up to two years; but one year seems to be about the average duration of this incubation period, and it is one most in harmony with the recurrence of the seasons calculated to afford the embryo the opportunity of access to cyclops.

Symptoms.—When the guinea-worm is mature it gives rise to a certain amount of irritation leading to the formation of a small abscess or bulla, which presently bursts, disclosing, as already explained, a minute hole in the centre of the ulcerated integument. Occasionally some time, days or weeks, may elapse before the head of the worm appears, but sooner or later this is sure to be protruded. As a rule, the natives of countries in which the parasite is endemic proceed at once with its extraction, so that it is a rare chance to be able to study the natural course of events. If not interfered with, after the uterus of the worm has emptied itself in the manner described, the collapsed body will, in all or in most instances, be spontaneously discharged. This sometimes happens in the lower animals, in this instance the subjects of a happy neglect. Fleming relates a case of dracontiasis in a horse causing a swelling in the hock, which afterwards burst, discharging spontaneously a worm 18 inches in length.

Migrations in the Human Body.—Not unfrequently the presence of the filaria can be detected for some time before it seeks to effect its escape. It can be felt coiled up or stretched out under the skin; and, moreover, it has been observed to travel at the rate of several inches per diem from one part of the body to another, taking for the most part a downward direction towards the feet or ankles, where, as a rule, it finally emerges. These migrations of the worm are attended with considerable annoyance; sensations of tingling, burning, and pain even being complained of. It does not, however,

always, or usually, lie so superficially as to be traceable with the finger; very often its course is deeper, sometimes traversing the cellular tissue between the muscles and other organs. Da Silva Lima[1] relates a case in which a dracunculus, after travelling for some time about the head and neck, finally coiled itself up under the skin about the right nipple, its folds occupying an area as large as the palm of the hand. On examining the patient the following year all traces of the parasite had disappeared, and there was no history of abscess, surgical operation, or any evidence whatever that it had escaped from the body of its host. Possibly in some rare cases, such as this seems to have been, it may die and be absorbed; or, as Charles has pointed out, become calcified.

Accidents and Complications.—Frequently the filaria gives rise to extensive abscess, fistulæ, sloughing, and burrowing of pus among the muscles. Consequently its presence is not unattended with danger. Particularly is this the case when, in consequence of premature attempts at extraction, the worm snaps across and the young are poured out into the cellular tissues deep among the muscles. Severe constitutional symptoms may then follow, analogous perhaps to those occurring from absorption of hydatid fluid, and large abscesses may form and perhaps burst in some untoward direction, or give rise to septic trouble terminating in death.

Sites of the Parasite and Points at which it emerges.—As regards the region of the body at which the parasite elects to emerge, this, in the great majority of cases, quite 75 per cent., is the foot or ankle; next in frequency, perhaps in 10 per cent. of all cases, is the leg; then come the thighs, scrotum, trunk, arms, hands. On very rare occasions it has been found in the sheath of the penis, in the testes, tongue, upper eyelids, about the face, scalp, and elsewhere in different parts and organs of the body; but these sites are quite unusual, and it is by no means certain that the parasites reported as such, and as having been found in connection with liver, kidneys, and other viscera, were dracunculi.

Number of Parasites.—In the great majority of instances there is only one dracunculus visible at a time; in about a fourth of the cases two or three may be present, and once or twice in every hundred cases there may be six or seven. But such examples are rare. Cases, however, are on record in which as many as thirty, forty, or fifty were present at one time, the patients being literally riddled by dracunculi.

Seasonal Prevalence and Numbers affected.—It is found that the dracunculus is more prevalent at one season than at another. There

[1] *Veterinarian*, May 1879.

is no general law about this, however; in some places it is one season, in other places a different season. Its presence one year has reference, of course, to an infection the previous year. When the water supply becomes scanty and concentrated, we may infer that the chances of imbibing dracunculus embryos are proportionately increased; consequently droughts and the dry season have more to do with it than the rainy season, although the latter may concur with its final manifestations, as is the case in many places in India. In most places in which it is endemic it is observed that there is an annual periodicity about the recurrence of the epidemics. In some districts a very large proportion of the inhabitants are affected at these times. Aitken says that "its annual endemic prevalence in the Carnatic villages is in December, January, and February, during which time more than half the inhabitants are affected." Dr. Siciliano,[1] referring to the Slave Coast, West Coast of Africa, says that nearly half of the inhabitants are affected with guinea-worm; but he does not say that it is more common at particular seasons.

Prophylaxis.—From our knowledge of the life history of the guinea-worm, the direction which prophylaxis should take is clearly indicated. In the first place, individuals harbouring the parasite ought to be interdicted from washing their sores in, or in the neighbourhood of, the drinking water supply of a community. It is not sufficient to interdict washing *in* the source of water supply merely; the sores ought not to be washed even in the *vicinity* of this; for the embryo filariæ, falling on the ground, after desiccation might be readily blown into the water as dust. Second, in districts in which dracunculus is endemic, all drinking water ought to be boiled or carefully filtered. The importance of a pure water supply as a preventive of this as of so many other diseases is very manifest.

Treatment.—The practice in all countries in which guinea-worm is endemic is to ignore and interfere with the natural processes, and to attempt extraction of the worm so soon as the head can be laid hold of. This is done in various ways. A common method is to slip the free end of the worm into a cleft made in a piece of wood or bamboo, and gradually to attempt to roll out the animal on this. The rapidity with which extraction is accomplished depends on the amount of resistance experienced. Sometimes it comes out readily enough, inches at a time, at other times only one or two turns of the stick can be made daily. What has been gained at a sitting is maintained by fixing the piece of wood to the skin by strips of diachylon or by other means. The Arabs sometimes maintain constant and steady traction on the worm by attaching to it a piece

[1] *Arch. de méd. nav.* June 1892.

of lead; and in some parts of India traction is made by tying a bunch of grass or of leaves to the protruding part, the patient then swimming about in a tank or wading in a running stream.

If the coils of the parasite are closely approximated, and it is felt in a bunch below the skin, it will shorten matters to cut down and extract the worm at once. Some of the Indian hakims are very dexterous at this; after cutting down and hooking up a loop, by gentle traction and kneading the limb, they often succeed in removing it entire at one sitting of an hour's duration.

I mention these methods only to condemn them and their blind rule of thumb application. If, when any of these or similar plans of treatment is put in practice, the parasite has already, happily, emptied her uterus, well and good; but if her uterus be still stuffed with embryos, it is difficult to understand how rupture and the violent inflammation consequent on the extravasation of millions of living and very active embryos into the tissues are to be avoided. For, as the winding out progresses the contents of the uterus must, necessarily, be more and more driven back towards the tail end of the worm—squeezed backwards, as it were, by the reeling out on to the piece of wood. In this way the diameter of that part of the worm lying in the tissues is augmented, and resistance consequently increased. In addition to this, the eccentric pressure from the accumulating uterine contents must rapidly rise, and the breaking strain of the worm—11 oz.—being reached, rupture is the inevitable consequence. So I interpret the teachings of helminthology.

It has been shown that left to herself the filaria, after expelling, as above described, all her embryos, becomes flaccid and empty, and, according to Forbes, in from fifteen to twenty days from her first appearance, begins to be discharged spontaneously, and without pain or swelling. Treatment by rolling the worm on a piece of wood lasts on an average some fifteen to thirty days, and often causes considerable pain. So that little seems to be gained by an interference which, should it lead to rupture, may be disastrous. It seems to me, therefore, that the adage "that meddlesome midwifery is bad" has an application here; and that the best treatment for guinea-worm, unless it is evident that it can be cut down on and removed at one sitting, is to leave it alone. The worm should be protected from injury by some simple water-dressing (containing no poison such as antiseptics usually are), and encouraged to empty herself by pouring, two or three times a day, cold water—the natural uterine stimulant—on the leg, or by sending the patient to bathe frequently in some stream or pool not used for drinking purposes. When the uterus is quite emptied,—which can easily

be ascertained by the douching experiment above described, or by observing that the protruding end of the worm is commencing to shrivel up,—if the worm be not spontaneously discharged, its extraction by reeling out might then be cautiously attempted, the parts being douched with cold water several times daily, dry antiseptic dressings being applied in the intervals.

3. Filaria Loa.

We have very little precise information about this parasite, although it appears to be fairly common in the districts it haunts on the West Coast of Africa. Its habitat is the subcutaneous cellular tissue. It possesses considerable powers of locomotion. In its peregrinations it sometimes arrives at the subconjunctival cellular tissue, where it is easily seen, and where it may create considerable irritation.

Symptoms.—In a letter to Dr. Morton, U.S.A., the Rev. Dr. Nassau, a missionary at Gaboon, gives the following graphic account of his personal experience of this form of helminthiasis:—

> When I first came to Africa, in 1861, I heard of worms in the eyes of natives, and in the eyes even of white foreigners. Even when people came to me with swollen eyes, and said there was a worm in the eye, I did not believe it; after a few years I had the same form of swollen eye myself; still I did not believe in the parasite. Before I went to America in 1871, I was fully convinced I saw the worm wriggling under the skin of one of my fingers. In America I felt some of the symptoms of its presence. Since my return here in 1874, I have seen the worm again under the skin of my own fingers and the fingers of others; and a few months ago in the skin of my left lower eyelid. Using a glass, I seized it with a forceps, and cut at it with a scalpel, but it wriggled away across my cheek. An English trader, living near me up the Ogowe, was afflicted for months with what he called neuralgia in his head. He described the pain in his eye as unendurable. Finally a worm was discovered wriggling under the conjunctiva of the eyeball, making directly towards the cornea. The worm returned, disappeared, returned, and was extracted by a native using a thorn as a needle. My own worm I have never observed in the eyeball, but always in the eyelids, hands, or fingers. The symptoms are—swelling; the skin becomes tense, hot (not inflamed), very itchy, and it is impossible to refrain from rubbing the eye or hand; at once the swelling rises harder and harder. The eyes involuntarily weep, and then become inflamed; but the latter, I think, only from the rubbing. In the eye, in the line of the worm's track, at frequent intervals, are felt sudden stinging pains, like neuralgia; the constant sense of tension and itching are unendurable, but I have dreaded, as the sharp sting of one pain died slowly away, to await its repetition, which I knew would follow in a few minutes. This stinging pain I have not felt in the fingers, but in the centre of the palm of my hand, and I do not remember to have felt it in both hands and eyes at the same time, nor to have seen more than one worm at a time. I think I have

observed the stinging pain worse at night. The worm never stays, with all these effects, more than a day or two at a time, and may not be felt for two weeks again. There are no guinea-worms on this part of the coast; they are at the Accra coast, and Cape Castle in Ashanti.

Anatomical Characters.—According to Leuckart and Leidy, the loa measures from 16 to 70 mm., — usually from 30 to 40 mm., and is about the thickness of a fiddle-string. It is pointed at one end, blunt at the other. The latter is probably the head, and is provided with a prominent papilla, but no special armature. The intestine is straight, but the situation of the anus is as yet unknown. The genital tube contains embryo-bearing ova, measuring 35 μ by 25 μ, and, at its lower part, free embryos. From a sketch of the latter, kindly sent to me by Professor Leuckart, it is evident that they resemble very closely *F. diurna* and *nocturna*, although in the sketch referred to, a sheath is not represented.

Life History.—If little is known of the anatomy of this parasite, still less is known about its life history. Probably its young enter the circulation as a first step towards development. Considering the size of its embryos, their anatomical characters, its geographical distribution, and the fact that it had in one instance been present at a previous date in a negro in whose blood I afterwards found *F. diurna*, it is not unlikely that *F. loa* may turn out to be the female parental form of *F. diurna*.

Geographical Distribution. — *F. loa*, as an indigenous parasite, is confined to the equatorial region of the West Coast of Africa, so far as known. Formerly it was not uncommon in America among the imported negroes, but since the abolition of the slave trade it is no longer found there; so that it has not become acclimatised on that continent, probably from the absence there of a suitable intermediary host.

Treatment.—The natives in certain localities extract the parasite, using very primitive instruments. It seems to me that by passing a sharp hook underneath the creature's body as it lies under the skin or conjunctiva, and so fixing it, and thereafter cutting down on it, extraction could be easily effected. My patient informed me that it is the custom, among the negroes, to drop a few grains of salt into the eye when a loa appears there; this has the effect of causing it to retreat. It was so treated in his own case, and did not return, although very probably it is still lying elsewhere in the cellular tissue, and breeding the embryos which are so numerous in his blood.

4. Filaria Volvulxus.

Professor Leuckart informed me some time ago that he had received from a German medical missionary, practising on the Gold Coast, West Africa, two tumours, each about the size of a pigeon's egg, which had been removed from negroes,—from the scalp and chest respectively. Both tumours contained several mature filariæ —3 to 4 females, 1 to 2 males. These parasites were of considerable length—the female specimens from 60 to 70 cm., the males about half this. They were coiled up together in the form of a ball, about the size of a hazel-nut, and were difficult to unravel. Apparently in the first instance the individual parasites had occupied separate tunnels, but the partitions between them having broken down, these tunnels had become merged into a common cavity. This cavity, in addition to the mature filariæ, contained a fluid laden with free embryos.

Professor Leuckart very kindly sent me a slide containing a fragment of the uterus of one of these parasites. It was stuffed with outstretched embryos, resembling in shape and dimensions *F. diurna* and *F. nocturna*. Possibly, compared to these parasites, the embryo *F. volvulxus* was somewhat shorter, and also somewhat broader proportionately, and more abruptly truncated at the cephalic end. Little value, however, can be attached to this observation, as in glycerine or spirit preparations, shape and dimensions are apt to alter considerably. One important feature of *F. diurna* and *F. nocturna* I did not see represented in the embryo *F. volvulxus*, viz., the sheath. Professor Leuckart makes the same remark. It is probable, therefore, judging from the absence of this structure, that *F. volvulxus* is not the mature form of *F. diurna*, and it is certainly not the mature form of *F. nocturna*. Judging also from the absence of sheath, it may be that the life history of this parasite is somewhat different in character from that of *F. diurna* and *F. nocturna*.

Doubtless, in certain districts of Africa this helminthiasis is not uncommon; but, beyond the facts just stated, we possess very little information on the subject. Observations on this and allied matters are very much wanted. From the numerous discoveries, notwithstanding very limited opportunities, made in recent years in African pathology and helminthology, it is evident that many novelties await the investigator of disease in that country.

5. The Chigger or Sand-Flea (Pulex Penetrans). (Fig. 88.)

This exceedingly troublesome little insect was formerly confined to the central part of the American continent, having a geogra-

phical range from Mexico to the Argentine (30° N. to 30° S.), including the West India Islands, and being as abundant on the high grounds as on the low-lying plains. Lately, in 1872 or 1873, it has been introduced into Africa, and has spread over the greater part of that continent with incredible rapidity, being found now as far north as Algeria, and as far south as Benguela. It has also extended to Zanzibar, and is already probably in full possession of the Soudan. Doubtless, before very long, it will have invaded the tropical parts of Asia.

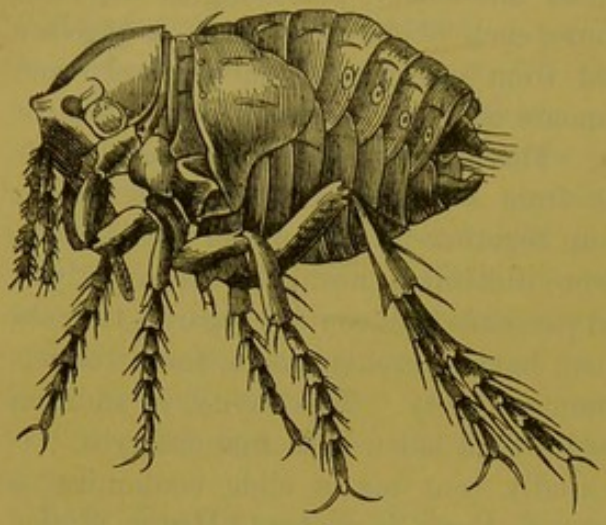


Fig. 88.

The chigger lives on the ground among the grass, woods, or plantations. It is most abundant in dry sandy soils, particularly in the sand by the seashore. A favourite haunt for it is the dirty, unswept native cabin, among the dust and ashes; and it is especially common where cows, horses, sheep, oxen, pigs, fowls, and other domestic animals are kept and imperfectly cleaned. It attacks all warm-blooded animals, fixing itself indifferently on the first that comes in its way.

The chigger is much smaller than the common flea, being only about one half or one third the length. It is reddish-brown in colour, and may be further distinguished from the common flea by its proportionately larger head and broader, deeper abdomen.

Both the male and the female are for the most part free parasites—the male always, and the female up to the time of impregnation. They suck the blood by piercing the skin whenever they get a chance, dropping off when gorged. When the female has been impregnated, she proceeds to bury herself in the integuments; in the case of man, in those parts nearest the ground, preferably the soles of the feet, especially about the great toe, underneath the free end of the nail, and in the digito-plantar folds. She does not confine herself exclusively to the feet, but is frequently found on the scrotum, thighs, and other parts of the body. She penetrates the skin, by passing obliquely through the epidermis to the corium, from the capillaries of which she obtains an abundant supply of nourishment. The head and thorax do not undergo

any change, but the abdomen becomes rapidly enlarged from the development of the eggs it contains.

The insect now swells out enormously (Fig. 89), raising the skin into a small tumour, perhaps the size of a small pea, the nature of which is betrayed by the attendant itching, and the presence of the small hole of entrance at its centre. The dilatation of the abdomen is made entirely at the expense of the second and third segments, the first and posterior segments retaining their original size, the latter plugging the orifice of entrance in the epidermis. When the abdomen has attained its full size, the surrounding tissues become inflamed, and a purulent fluid bathes the body of the parasite. Presently the overlying epidermis gives way, and the intruder is cast off by ulceration. The abdomen of the insect then ruptures, and its eggs fall on the ground. In due time these are hatched, producing a larva which, after enclosing itself in a cocoon and passing through a nympha stage, emerges in from eight or ten days as the perfect insect.

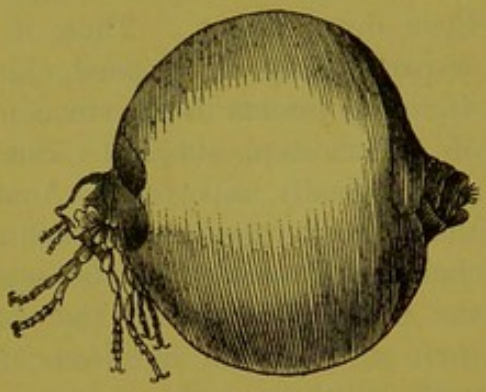


Fig. 89.

The number of chiggers lodged in the skin at any one time is very variable; there may be only one or two, or there may be dozens. As the tumours they produce approach maturity, they give rise to a great amount of irritation or even pain. As a rule, attempts at extraction are made; and if this little operation be not skilfully and successfully performed, ulceration and much suffering may ensue. If the insect be ruptured, ulceration is sure to follow; nails may be lost, and sores produced incapacitating the sufferer for a long time for locomotion.

In districts where the sand-flea abounds, sleeping on the ground, or walking barefoot ought not to be indulged in. Houses ought to be thoroughly swept, and ashes and all sorts of dust and dirt removed, and the floors sprinkled with water or carbolic acid and water, before occupation is entered on. Insect powder will probably be useful. If it be discovered that a chigger has lodged itself in the skin, its extraction ought to be set about very carefully, and, if possible, the services of a native expert employed, care being taken that the operator uses clean instruments. The orifice of entrance should be enlarged with the point of a needle till the periphery of the sac is exposed, when the entire body is to be neatly shelled out.

Should the insect be ruptured in the attempt at enucleation, the little pit in the skin should be swabbed out with a strong solution of nitrate of silver, and dressed antiseptically.

6. Cutaneous Myiasis.

Man, in common with the lower animals, is frequently the host of the larvæ of certain species of flies to which a temporary sojourn in the organs of a warm-blooded animal is a necessary factor for their development. Thus, it is by no means a rare occurrence —particularly in tropical climates—to meet with the larvæ of dipterous insects in the stools of patients, or in the alimentary canal of post-mortem subjects. Such, however, are more curious than pathologically important. Another class of larvæ find their opportunity for development in wounds or sores, or in such accessible but sheltered situations as the nostrils, the auditory canal, the vagina, the prepuce, and even the conjunctival sac. In consequence of their voracity, and by means of the powerful masticatory apparatus with which some species are provided, they rapidly destroy the tissues they are lodged in; in this way they may become a source of very great danger, and, unless intelligently and promptly treated, may even cause death. A third class of larvæ proceed in development by first penetrating and obtaining a lodging underneath the skin of their temporary host. Here they grow at the expense of the tissue on which they feed, causing much pain and irritation, and not infrequently leaving troublesome sores which, in malarious and unhealthy climates, are not without danger.

Of late years three forms of this kind of ecto-parasitism occurring in tropical climates have been studied, viz. that caused by the larvæ of *Lucilia macellaria*, of *Dermatobia noxialis*, and of *Ochromyia anthropophaga*; doubtless there are many more.

(*a*) ***Lucilia macellaria*** (*L. hominivorax*) is an inhabitant of America, being found from the United States in the north to the Argentine Republic in the south. It is most common in the warmer districts of this extensive range. The perfect insect measures from 9 mm. to 10 mm. in length; thorax blue, streaked with darker bands of a purple tint; pads black, and wings brown at the base. The larvæ measure from 14 mm. to 15 mm. The fly deposits her eggs in wounds and sores, and in the ears and nostrils of men and animals. When hatched out the larvæ, by means of their two powerful buccal hooks, attack the tissues, which they devour rapidly, producing extensive mutilation. Should they enter the external auditory canal, they attack the tympanum, destroy the

internal ear, and give rise to meningitis and intracranial suppuration. When the nostrils are affected, the larvæ devour the soft tissues in every direction, passing even into the frontal sinuses, soft palate, pharynx, and the deep structures at the base of the cranium. They even strip the periosteum from the bones, which then become carious or necrosed. Therefore, in the haunts of this fly, bloody and offensive nasal discharges, with severe headache and fever, should suggest this most dangerous form of parasitism, which, if neglected, may lead to coma, convulsions, and death. Of 31 cases collected by Maillard, 21 died.

(*b*) ***Dermatobia noxialis*** is also a native of the American continent. The larvæ, according to the district in which they occur, are known by a variety of names. In New Grenada they are called *Nuche* or *Gusano;* in Cayenne, *Ver Macacque;* in Brazil, *Ura;* in Costa Rica, *Torcel;* and in Mexico, *Moyoquil.* The perfect insect is about 17 mm. in length; face and forehead yellow, with short silky brown hairs; thorax brown, striped with grey; abdomen flat, blue, and covered with small black hairs, their bases being of a dirty white. This fly frequents the outskirts of woods and lands covered with scrub. They deposit their eggs on cattle and dogs principally, but also occasionally on man. The larvæ, when hatched out, penetrate the skin, and increasing in size produce much irritation, diffused congestion, and, at times, an inflamed swelling like a boil. From the aperture of entrance, which is visible at the centre of the swelling, a sero-purulent fluid mixed with black particles—the fæces of the larva—exudes. The larvæ attain a length of about 3 cm. They are elongated, and pointed at one end. The anterior half of the body is alone provided with spines. It is also furnished with two strong buccal hooklets.

(*c*) ***Ochromyia anthropophaga*** is a native of Senegambia, and is particularly prevalent in the district of Cayor; hence the name of its larva, *Ver du Cayor.* The fly is of a greyish-yellow tint, measuring from 8 to 9 mm. in length; head testaceous, and covered with small black hairs; style of antennæ plumose; thorax marked in front with two longitudinal black bands; wings slightly smoky; abdomen covered with large black spots, particularly behind. This insect is believed to lay her eggs in the sand, whence, an opportunity occurring, the larvæ emerge and penetrate the skin of man or domestic animal. Underneath the skin the larvæ grow rapidly, giving rise to a small inflamed swelling covered with a brown crust. In six or seven days the larvæ leave their temporary host and pass into the pupa stage. The small sores so produced usually heal rapidly.

Treatment.—In the case of *Dermatobia noxialis* and *Ochromyia anthropophaga*, the larvæ may be removed by first enlarging slightly the orifice of entrance, and killing the intruder by the application of a little turpentine, carbolic acid, creosote, or sublimate lotion; they may then be cautiously expressed or picked out with a forceps. The larvæ of *Lucillia macellaria*, when their situation permits, may be easily got rid of by syringing the sores and sinuses with some antiseptic fluid. When the nostrils or ears are attacked, the employment of poisonous lotions may be dangerous; in this case tar, or creosote, or chlorine water may be used instead. The vapour of chloroform is said to cause the larvæ to relax their hold, when they drop off and may be washed away. In the event of the larvæ entering the frontal sinuses, trephining may be necessary.

7. Leeches.

In many tropical countries much annoyance, and sometimes even fatal results, are brought about by various species of water and land leeches. In the South of Europe and in North Africa the common horse-leech—*Hæmopis sanguisuya*—frequently attaches itself to the mucous surfaces of the nose and pharynx, and even enters the larynx and air-passages of men and animals. Horses and cattle are much debilitated by them at times; and, in the event of their entering the air-passages, death by asphyxia may ensue. Men have also been killed in this way.[1] Doubtless there are other species besides the *hæmopis*. In the island of Formosa, in the course of ten or twelve years, I heard of three cases of this form of parasitism. Monkeys are likewise attacked there in this way. In one man—an American—I removed the leech from the nostril. It had been taken in with drinking water, and had attained considerable dimensions, and caused much suffering from headache, repeated attacks of epistaxis, and a considerable amount of anæmia. In this instance the parasite did not protrude externally; but in the two other instances the leech could be induced to show itself, and wander about over the upper lip, by the patient stooping and laving his face with water.

There can be no doubt that these leeches enter the mouth in foul drinking water. Creeping round the velum, they attach themselves, by means of their powerful suckers, to the mucous membrane which they pierce with their jaws. They frequently shift their hold, and so give rise to recurring attacks of profuse hæmorrhage, which is further augmented by the fact that they expel a large

[1] See an excellent paper by Chavaase on this subject in the *Arch. de Méd. et de Phar. Milit.* Feb. 1893.

proportion of the blood they imbibe. In countries in which these parasites occur, repeated attacks of epistaxis without apparent cause, with severe headache and progressing anæmia, ought to excite suspicion of the presence of leeches in the nostrils, and lead to a careful examination with suitable specula. If a leech be discovered, it may be easily removed by attaching to it a spring forceps, and syringing out the nostril with salt and water.

Land leeches are a very common and troublesome pest in many tropical countries. One of the best known species is the *Hæmadipsa Ceylonica* (*Hirudo tagalla vel Ceylonica*), which, as its name implies, is found in the island of Ceylon. It is about an inch in length, and no thicker than a knitting needle, but of prodigious activity. It lies among the grass and leaves in the woods and jungles, and attacks with great avidity any man or beast passing near it. Similar land leeches occur elsewhere, as in Japan, the Philippines, the islands of the Malay Archipelago, the Himalayas, Africa, Australia, Chili, and, doubtless, elsewhere. They are easily removed. Travellers passing through leech lands require to protect their legs and feet very carefully, otherwise the repeated small bleedings these leeches give rise to may bring about a state of great debility; death even has been known to be so caused both in men and in domestic animals.

SECTION III.—SKIN DISEASES CAUSED BY MICRO-ORGANISMS.

1. Boils.

Boils are common in all climates, but especially so in the tropics, where in some years they are almost epidemic. Few young children there get through a summer without one or more, and they are common enough in adults as well. Doubtless the sodden state of the skin arising from perspiration, the broken epidermis resulting from the scratching associated with prickly heat, ringworms, insect bites, and such circumstances, together with the debilitated condition of the system generally brought about by prolonged heat, poor living, and perhaps by repeated attacks of fever, favour the invasion and germination of the micrococcus,—*Torula pyogenica*,—which Pasteur has shown to be the specific cause of this disease, the inoculability of which had already been demonstrated by Startin.

As a rule, the starting-point of a boil is a hair follicle or a sweat gland. It commences as a minute, excessively itchy vesicle or papule. The papule or vesicle may pustulate; but although the pustule may open, the disease does not come to an end. On the

contrary, the base of the little pimple enlarges, indurates, inflames; the surrounding integuments become congested, inflamed, swollen, and tense, and there is very great local pain. Constitutional disturbance ensues, with headache, malaise, slight shivering, and pyrexia. Unless in the case of what are called "blind" boils (that is, boils which do not suppurate, but which gradually become absorbed), in the course of three or four days the apex of the swelling ulcerates, a certain amount of blood and pus are discharged, and, on this occurring, the central slough or "core" is exposed. The pain and feverishness immediately begin to subside, and in the course of a day or so the core is discharged, picked out, or expressed. The resulting cavity granulates and heals rapidly. A permanent scar results which, along with a portion of the surrounding skin, frequently becomes pigmented, assuming a dark coppery colour. This pigmentation may last for years, and is often erroneously mistaken for evidence of syphilitic antecedents. If one boil occur it is very liable to be followed by others, either near the site of the first, or elsewhere about the body. Doubtless these secondary boils are, in many instances, brought about by auto-inoculation. Sometimes it happens that the interval between the successive boils is one of months, suggesting that a necessary factor in the production of the furunculosis in such cases is something of a constitutional nature, and that the specific microbe possesses a certain faculty of latency. It would seem that occasionally infection takes place through the blood, as in furunculosis produced by decaying food, foul water, and sewer gas. It is likely, however, that in the majority of instances of tropical furunculosis the boils are produced by germs which have fallen on, or been applied to the skin from without, and which, on finding the local and constitutional conditions favourable, germinate.

In all cases of recurring boils the urine ought to be examined for sugar and albumen.

Treatment.—This resolves itself into constitutional and local. The discovery of sugar or albumen in the urine calls for treatment appropriate for a disease in which furunculosis is a comparatively insignificant incident. A history of malarial fever, or of sewer gas, or other form of septic poisoning, indicates quinine. Anæmia and debility point to ferruginous and quinine tonics, mild aperients, good food, and wine; stout enjoys a reputation in such cases. It is customary with some to prescribe sulphide of calcium in quarter grain doses three times a day, or in tenth of a grain dose every three hours; as a matter of routine I have often employed this drug, but cannot say I attribute to it any very marked virtue. Fresh yeast, half a wineglassful night and morning, has also been favour-

ably spoken of. Tar water, a quart per diem, is a favourite remedy with the French. Arsenic, phosphorus, liquor potassæ, ammonia and bark, hyposulphite of soda, have each had their advocates.

When a boil has formed, and there is much hardness and a considerable amount of swelling and inflammation with constitutional disturbance, all that can be expected of local treatment is to palliate somewhat the suffering by rest, elevation, and the application of some cooling, soothing lotion. Poultices, unless strongly impregnated with an antiseptic, are apt to spread the trouble, and, as a rule, should be avoided. A boil, if it be tending to point, unless there is excessive tension, had better not be incised. In certain parts, especially in the axilla, where boils are slow in reaching the surface, and in the scalp, where they are very apt to become abscesses, and to burrow, especially in young children, it is better to use the knife as soon as matter has formed. A boil should never be squeezed or irritated in any way, as by so doing it is further inflamed and enlarged. When discharge begins the parts ought to be freely and frequently washed with corrosive sublimate lotion, 1 in 1000; and, when the lotion has dried, freely powdered with boracic acid and starch, and a dry, absorbent, antiseptic dressing, such as oakum or salicylic wool lightly bandaged on.

Many methods for aborting boils have been recommended, and some of them are successful if intelligently practised. To be useful they must be employed at an early stage. Salicylic acid in 10 per cent. solution to the apex of the initial papule, or as 10 per cent. ointment, plaster, or mull has been recommended. Other methods consist in the application of compresses of spirits of camphor for a few minutes at a time, three or four times a day; painting with tincture of iodine or saturated solution of boracic acid; mercurial ointment, liquor ammoniæ, acid nitrate of mercury, opium plasters, subcutaneous incision, scraping, nitrate of silver, potassa fusa, an incandescent lucifer match, injections of carbolic acid solution, etc., have all had their advocates. The following plan I found very successful, both in aborting incipient boils and in preventing their spread:—I impressed the patient or, in the case of a child, the mother or nurse with the contagious nature of the discharges, and directed them to wash very frequently the neighbourhood of all discharging boils with corrosive sublimate solution, to powder the body freely with boracic acid and starch, and to dress all boils antiseptically with a dry, absorbent, antiseptic dressing. I told them to keep a sharp look out for the appearance of any itching vesicle or papule, the herald of a fresh boil. Should such appear, they were directed to drill into the centre of the papule with a sharp pointed

piece of hard wood, fashioned like a pencil, which had previously been dipped in pure carbolic acid. Just sufficient carbolic acid should be taken up on the piece of wood to form a minute droplet at the pointed end when it is directed downwards. If the drilling be done very slowly and steadily, and with not too much pressure at first, it causes little pain, the carbolic acid acting as an anæsthetic. In this way a small hole, an eighth of an inch or more in depth, may be drilled in the centre of the boil. From time to time during the drilling the piece of wood is recharged with acid, so that the antiseptic is well driven into the tissues surrounding the hole. If this be done sufficiently early and thoroughly the boil will generally be aborted. I regret I cannot recall the name of the inventor of this proceeding; it is certainly a very effective one.

Change of air is very desirable in severe and persistent furunculosis.

2. Tropical Sloughing Phagedæna.

It is certain that in addition to that associated with syphilis, cancer, tubercle, lupus, and leprosy, and such well defined endemic forms as Oriental sore, parangi, yaws, verruga, etc., there is at least one other type of ulceration of a specific character to be met with in the tropics.

Like so many diseases occurring in foreign countries, this form of ulceration is very generally named after the place in which the describer for the time being encounters it, and, consequently, it has received a great variety of local names. From this circumstance the inference is apt to be drawn that each of the differently named ulcers is a variety by itself, and specifically distinct from all others. Thus we read of Mozambique ulcers, Cochin-China ulcers, Yemen ulcers, and so forth. It would be wrong, however, to conclude from this multiplicity of names that there is a corresponding multiplicity of species, and that almost every community and country has an ulcer peculiar to itself. Without denying that there may be other forms of acute necrosis of the soft tissues with a limited geographical range, it is now generally recognised that the great majority, if not all, of these differently named ulcers are really one and the same disease, for which "tropical sloughing phagedæna" seems to be a suitable name.

It is difficult to say positively if this form of ulceration differs from the type at one time common enough in Europe, and known as "hospital gangrene"; probably they are not the same disease. Be this as it may, the conditions under which sloughing phagedæna is met with in the tropics are very well understood. Given a body of

men badly fed, badly housed, exposed to the vicissitudes of the weather in a hot damp country, and with health deteriorated by overwork, malaria, and scorbutus, sloughing phagedæna is sure, sooner or later, to break out. Thus it has often attacked our soldiers, and the soldiers of other nations, and their retinue of native camp-followers, when campaigning in tropical countries. In books on African travel we read of the terrible ulcers which attack the legs of the native porters, and decimate the slave gangs on their way to the coast. Agriculturists in hot countries, especially those employed in opening plantations in jungle lands, wood-cutters, grass-cutters, rattan collectors, men who are particularly exposed to wounds from thorns and other causes of slight abrasion of the integuments of the legs and feet, are particularly liable to this form of ulceration. In the cachectic conditions alluded to any wound, no matter how trivial, a mere scratch, a mosquito bite, a small boil, a scabies pustule, a guinea-worm or chigger sore, a chronic ulcer, the chafing from a badly fitting boot or sandal string, is liable to take on a phagedænic action, and rapidly to develop into an enormous putrid sore.

If the initial lesion be an old ulcer, or a wound of some extent, the raw surface becomes dry, grey, and quickly assumes the character of a slough; but if the initial lesion be minute, as in the case of a mosquito bite or small pustule, the first sign of the development of phagedæna is generally the formation of a large bleb with sero-sanguinolent contents. This bleb enlarges rapidly and then bursts, disclosing an ash grey slough an inch or more in diameter. Day by day, often apparently without much pain or constitutional disturbance, the sloughing process extends until an area of tissue as large as the open hand, or even larger, is converted into a yellow, stinking, gangrenous mass of putridity. I have seen such a sore involve the whole of the front of the leg from above the knee to the toes. The centre of the sloughing area in the course of a few days tends to liquefy and disintegrate; it is then seen that not only is the subcutaneous tissue destroyed, but that muscles, tendons, nerves, and vessels may be equally involved, and, possibly, the subjacent bone be laid bare and its periosteum stripped off. The small bones of the tarsus and metatarsus and entire toes may necrose and sphacelate and be cast off. The surrounding tissues are congested and swollen, but not usually to what one would consider an extent proportionate to the amount of the adjoining sloughing, unless the patient has been moving about much. If the disease continue to spread constitutional symptoms of an adynamic character supervene, and diarrhœa, low delirium, hiccough, and septic fever set in, and death

may ensue. Usually, however, and sometimes even when the case seems to be hopeless, and without any apparent reason for the arrest of the phagedæna, the sloughing processes ceases to extend, the dead tissues are cast off, granulations spring up, and, after many weeks or months, the huge cavity fills up, and skins over or becomes converted into that form of chronic sore so frequently seen in the poverty-stricken natives of all tropical climates.

Often much deformity results from the ravages of these sores. Toes are frequently sacrificed; the articulations are deprived of their movements from the binding, constricting effects of adherent contracting cicatrices; great and permanent swelling of the foot may ensue from the strangulation caused by a cicatricial ring around the calf of the leg or ankle; the heel is frequently drawn up, and even the movements of the knee impaired by the extensive scars.

Doubtless these terrible sores are the result of the growth of some micro-organism which has found access to the tissues through the original wound. Under healthy conditions this germ would have been quickly disposed of by phagocyte or other innate means of protection possessed by healthy tissues; but, falling on weakened and undernourished tissues, it gets the upper hand for a time.

Why when once started it and similar organisms should cease to spread is still a moot point in pathology, and one which it is difficult to understand.

Some attempts have been made to isolate this hypothetical organism of sloughing phagedæna, but as yet they have led to no definite or practical conclusion.

Treatment.—Good food, fresh vegetables, wine if possible, quinine, and opium, are of the first importance. Without the first very little can be done to arrest the necrotic process when once fairly started in an individual, or to prevent the extension of the disease in a camp or community.

At one time I had to treat a great many of these sores, and I found that the most successful local treatment, and the best, not only as regards the individual himself, but as regards the comfort and safety of his fellow patients, was to remove at once the entire mass of sloughing tissue by the free application of pure carbolic acid. The patient should be put under chloroform. A good sized mop of lint is tied round a stout piece of wood and soaked with pure carbolic acid; with this the slough is thoroughly stirred up. The carbolic acid has the property of dissolving and disintegrating the slough, and in a few minutes the whole of the dead tissues can be thoroughly broken up and washed away. The patient should then be given a hypodermic injection of morphia,

and his sore irrigated constantly by a slow stream of tepid, antiseptic solution until healthy granulations begin to spring up. It may then be dressed with some ordinary antiseptic dressing. When the cavity has been filled in with granulation tissue, free grafting with many minute pieces of skin should be frequently practised. Generally by these local means, when they are combined with good food and quinine, the necrotic process is at once arrested. Should, however, sloughing continue, the carbolic acid ought to be applied again, and repeated daily until the morbid process ceases.

The chronic ulcers which result from this disease are to be treated on ordinary surgical principles. It often happens, however, that the poverty stricken native cannot spare the time necessary for treatment in hospital. In such cases I have found that the old Edinburgh plan of a shield made of thin sheet lead, and cut a little larger than the sore, is not only the best protective, and, under the circumstances, the cleanest and cheapest, but that it is also very effective in producing cicatrisation. The Chinese sometimes use a copper-plate in the same way, tying it on with a string, and providing for the escape of discharge by placing underneath the plate a drain made of two or three strands of rush pith. It is singular, as in this application of the principle of drainage, how often some of our greatest improvements in medicine and surgery have been anticipated by this very practical people.

3. Oriental Sore.

The term "Oriental sore" was suggested by Tilbury Fox as a suitable name for a specific form of sore endemic in North Africa, certain islands of the Mediterranean, Asia Minor, the Crimea, Syria, Arabia, Persia, the Tartar countries to the east and south of the Caspian, Hindustan, and—if the disease recently described by Dr. W. C. Brown,[1] of Penang, under the native name of "Puru," is to be regarded as Oriental sore—in the Malay peninsula. This affection is not equally diffused throughout these countries, but is more or less limited to certain towns and districts. Hence, as in the case of sloughing phagedæna, it has come to be called after these places, and considerable confusion has crept into the earlier literature of the subject in consequence. Thus it was spoken of as the sore, boil, bouton, clou, or evil of Biskra, Aleppo, Bagdad, Delhi, Mooltan, Penjdeh, and so forth. At the present day, however, the identity of these variously named sores is generally recognised. It

[1] *British Journal of Dermatology*, No. 56, vol. v. 1893.

is therefore desirable that the local names be abolished and some comprehensive name, such as "Oriental sore," adopted.

Symptoms.—Oriental sore is described as commencing as a small, itching, congested spot like a mosquito bite. Presently a minute papule, feeling like a small seed in the substance of the skin, forms in the centre of this congested area. The papule may then enlarge till it acquire a diameter of a line or two, or even as much as half an inch; usually, before it has attained any great dimensions, the epidermis over the centre of the minute tubercle begins to desquamate. This desquamating process sets in from five to ten days after the commencement of the disease. At first the scales of epidermis are very fine, dry, and shining; but as the process extends to the deeper layers the scales become thicker, coarser, browner, and more moist. The papule now softens somewhat, is dark, red, and shining; through the thinned epidermis dilated blood vessels, and also, by using a lens, several very minute yellowish spots, can be seen. After a variable interval the thinned epidermis gives way, or a small vesicle or acne-like pustule may form, and the stage of ulceration commence. At first the serosity oozing from the surface of the papule dries into a crust, which, by continued accretions from below, acquires a semi-rupial form; usually after a short time this crust becomes detached, and an ulcerating surface is exposed. Gradually the ulceration spreads by erosion at its periphery, and also by the formation around it of similar papules, which in time break down in the same way, the resulting sores falling into and becoming continuous with the central or primary ulcer.

As a rule these ulcers are oval in form, but not unfrequently they present an irregular sinuous outline. The margin, which is sharp, perpendicular, ragged, and slightly infiltrated, is surrounded by an inflammatory areola of variable extent. The surface of the ulcer may be smooth; generally it is irregular, and marked by little pits and elevations, and, at times, small dotted hæmorrhages. The sero-purulent discharge varies in quantity and in degree of viscidity; sometimes it is easily removed, at other times it clings to the surface of the sore, forming a thick, slough-like yellow pellicle. All around the ulcer, involving the skin and perhaps the cellular tissue, minute elevations, papules, and yellow spots show that the disease is advancing. Crusts may form now and again, covering part of or the entire ulcer; from underneath these the unhealthy discharge can be expressed.

In size the sores vary very much; sometimes they may be only half an inch in diameter, in other cases they are several inches across.

The appearance of the sore is, of course, liable to great modification, according to the treatment it receives. It may be irritated and inflamed; or it may be inoculated with the virus of phagedæna or erysipelas; or it may be associated with a certain amount of lymphangitis. There is also a variety in which ulceration never takes place, but the original papule spreads till it acquires the size of a pea or a a bean, and then, after a few months, with or without desquamating, gradually recedes and disappears. In other cases the ulcerated surface is never exposed, but is covered with a crust from the commencement; it heals in time, the crust not dropping off, however, till the cicatrising process is complete. These are exceptional forms.

After a variable interval of from three, five, ten, or twelve months, or even longer, healing sets in; healthy granulations spring up, and cicatrisation commences—usually at the centre of the sore. Relapses may occur, but finally the sore heals, leaving a violet coloured congested scar, which, after a time, becomes dark brown, and, finally, a light yellow. As the ulceration has destroyed the glands and follicles of the skin the hair is not reproduced, and a permanent somewhat depressed cicatrix, with a tendency to contract and produce some degree of deformity, especially if situated on the face, remains. This scar has acquired the name of "date mark" in some countries.

The Number of Sores varies from one or two to as many as fifteen or twenty, or more. Their *situation* in adults for the most part is on the arms, hands, legs, feet, more rarely on the face or neck. In infants and young children the face is the usual situation. Wounds, abrasions, scratched insect bites are very often the starting-points for sores, and consequently the liability of any particular region of the body to attack depends in great measure on its liability to injury.

Inoculation.—There can be no doubt about the inoculability of this disease. Experiments on man and many of the lower animals have frequently given positive results, although the effects vary somewhat according to the species of animal inoculated. It is very commonly stated that dogs are liable to be attacked by the ulcer on the nose. It has also been asserted that horses likewise are subject to Oriental sore; the affection called in India "barsatti" is considered by some writers as being of this nature. It may be that some of the ulcers to which the horse is liable are true Oriental sores, but it has been ascertained that "barsatti" is in many instances a helminthiasis, and quite distinct from the species of endemic ulcer we are considering.[1]

[1] Neumann, *Parasites of Domestic Animals.*

Incubation.—It is difficult to determine precisely the duration of the incubation period. That some time elapses between exposure to the virus and development of the characteristic signs of the disease is certain, but this time is probably very variable. The inoculated disease seems to develop most rapidly. Weber [1] inoculated a man who showed signs of the disease within three days. On the other hand, there are cases on record in which the disease did not show itself until a considerable time after the patient had left the endemic area; Vidal [2] mentions a case, of African origin, in which it did not appear until the patient had been in Paris fifteen days.

Age, Sex, and Occupation.—All ages and both sexes are equally liable to attack, and occupation seems to have no influence one way or another. In places in which the disease is very common no one seems to escape—most, indeed, suffering in infancy or early childhood, or, in the case of visitors, not very long after their arrival. Bagdad is, or was, such a place. Colvill [3] says, referring to this city, that few children escape. According to Sirus-Pirond and Oddo, Gafsa, in Tunis, seems to be equally afflicted. At one time I saw a great many Eastern Jews from Calcutta, Bagdad, and other Eastern cities. I could always recognise a Bagdad Jew by the "date mark" that was sure to be visible somewhere about his face. They frequently told me that all children in, and all visitors to, Bagdad sooner or later are attacked.

Protection.—The fact that a specific sore heals, is in itself a proof that a certain degree of immunity is established. It may be that the immunity is of a local or temporary character only, but it is immunity of a sort. Colvill states that the Bagdad Jews at one time inoculated their children so as to give them the disease in a situation where the resulting scar would not be seen; from this circumstance it is evident that these Jews considered one attack as protective. It seems, however, that the Delhi Commission found that Oriental sore could be inoculated on individuals showing the scars of a previous attack; and apparently, according to Hickman,[4] certain French observers have arrived at the same conclusion. The conditions of artificial inoculation are somewhat different from those of ordinary infection, quantity as well as quality being an element to be reckoned with in attempts to establish some infective diseases. It is possible that an individual partly protected by a previous attack may be able to resist the minute dose of virus

[1] Fox and Farquhar, *Skin and other Diseases of India*, etc.
[2] *Semaine méd.* 6th April 1887.
[3] *Trans. Med. and Phys. Soc. of Bombay.*
[4] *Practitioner*, Jan. 1886.

introduced by ordinary and accidental means, and yet be unable to resist the large amount introduced in an artificial inoculation. This is a subject which, as regards Oriental sore, deserves, on practical as well as on scientific grounds, further attention.

Histology.—The histology of Oriental sore has been very carefully studied, both by English and continental workers; amongst others by Lewis and Cunningham, by Cunningham independently, and, more recently, by Riehl.[1] Their several observations were made on sections of the initial tubercle prior to ulceration, and they agree very closely. The essential histological feature of the initial tubercle appears to be the infiltration of the skin and subjacent areolar tissue with lymphoid and epithelioid cells, and disassociation of the tissue elements thereby. Towards the periphery of the tubercle the cells of the neoplasm occur in isolated clusters or grouped about the blood vessels and lymphatics; as the centre is approached the cells become more numerous, and about the focus of the disease, save for a few scattered and degenerate remains, they entirely replace the original tissues. The individual cells of this infiltration vary considerably in size and shape. On an average they measure from 7 μ to 9 μ. The nuclei for the most part are single, and are relatively large, 3 μ to 6 μ. The infiltration does not primarily affect the hair follicles or the sebaceous glands. Oriental sore must therefore be classed among the granulomas.

Bacteriology. — Numerous attempts have been made to demonstrate the micro-organism which has been assumed to be the irritant giving rise to the characteristic neoplasm. Cunningham—and the observation has recently been confirmed by Firth[2]—observed that by staining sections with gentian violet, and afterwards washing them in spirit till all colour had nearly faded, peculiar violet-stained bodies could be seen scattered about among the smaller lymphoid cells. These were found to be cells enclosing certain round, stained bodies, which varied considerably in size and grouping. The number of cells containing these bodies varied in different fields, some fields having only one or two, others showing them in abundance. Cunningham was inclined to regard these minute bodies as representing various stages of some simple organism constituting the virus of the disease. Riehl, on the contrary, regards them as hyaline bodies, the result of degenerative processes in the cells containing them, and therefore not parasitic—consequences of the disease, and not the cause. This observer found isolated

[1] *Twelfth Ann. Rep. San. Com. Gov. in India; Scient. Mem. by Med. Off. of the Army of India*, part i. 1884; *Ann. de Dermat.* 2nd ser. ix. 1888, p. 404.

[2] *Brit. Med. Journ.* Jan. 10, 1891.

micrococci, in size from 0·9 μ to 1·0 μ, in the protoplasm of the granulation cells in great abundance. They were found in sections which had been placed in aniline solution for half an hour, and afterwards washed for one minute in 1 per cent. acetic acid, dehydrated in alcohol, cleared in cedar oil, and mounted. Riehl was not successful in cultivating this micro-organism; as, however, it was found in a tubercle which had not ulcerated, and which was plunged into alcohol immediately on excision, the micro-organism was certainly not the consequence of accidental contamination nor of decomposition. He is inclined to look upon it as the germ of the disease.

Etiology.—From time to time many theories have been advanced on this point, but none of them can be considered as established. It is certain that the disease can be conveyed by inoculation, and therefore that it depends on an organised virus. The fact that the sores generally occur on those parts of the body which are most frequently exposed, suggests that the inoculation may be effected by flies, mosquitoes, or other insects, or by the accidental application of the virus to a broken surface in washing, or as dust floating about in the atmosphere. The insect hypothesis is supported by the circumstance that in infants the face is the part usually attacked, and that this is the part most often exposed in their case to the bites of insects which they do not understand to brush away. The fact that the disease, although it may be introduced into hitherto unaffected districts and spread,[1] clings only to particular districts, seems to indicate that there are local conditions of soil, or water, or vegetation favouring the multiplication of the virus outside the human body. As to the nature of the virus itself, many theories and speculations have been started. Lewis and Cunningham regard it as of a chemical nature, bound up in some way with hard water; Flemming and Smith suggest that the ova of a parasite have something to do with it; Carter and Weber consider that a fungus, which they find in the lymph spaces, is the virus. Duperet and Boinet separated by cultivation a micrococcus from the discharge of a sore, which was found on inoculation of the culture into various animals to give rise to sores more or less like Oriental sore—this micrococcus they regard as the cause of the disease; their observations have in a measure been confirmed by Chantemesse and Duclaux;[2] but none of these observers have succeeded in demonstrating this micrococcus in the tissues. It is possible, however, that the recent observations by Riehl, above detailed,

[1] Carter in Fox and Farquhar's *Skin and other Diseases*, p. 76.
[2] *Bull. de la Soc. anat. de Paris*, Oct. 1887.

and somewhat similar observations by Poncet,[1] fill in this important lacuna.

Treatment.—An abortive treatment seems occasionally to be successful in the earlier stages; but it requires to be thoroughly carried out, the actual cautery being preferred to acids. When ulceration has set in an expectant treatment is the best; for it seems to be impossible to forestall, by any line of treatment, the development of an insusceptibility to the virus, which can only be attained by the presence of a sore or sores on the body for many months. Antiseptic poultices should be employed to remove inconvenient crusts, great cleanliness should be observed, and all sorts of irritating treatment avoided. Some mild antiseptic application, such as boracic, salicylic, or weak citrine ointment, should be used on the dressings. Hickman, who has had a large experience, recommends painting the sore with a solution of iodoform in collodion, and he also mentions the use of the actual cautery and scraping with a Volkmann's spoon as a possible treatment.

Generous diet, with wine or beer if thought necessary, ought to be prescribed. Signs of scrofula, syphilis, or malaria may call for appropriate specific treatment. Whenever convenient the patient ought to be sent away from the endemic area, and, if in India, ordered to the hills or to England.

4. Pemphigus Contagiosus.

In the Straits Settlements, in Madras, and, during the hot season, in China, and probably elsewhere in damp tropical countries where the skin is more or less constantly bathed and sodden by perspiration, there is to be found a form of contagious bullous disease, which at times becomes exceedingly prevalent. In many respects this disease resembles some of the forms of the *Impetigo contagiosa* of colder climates; but there are points about it which seem to me to justify its being regarded as an independent species of skin disease. On the strength of these I have presumed to give it the name of *Pemphigus contagiosus*.[2]

This skin lesion is not associated with any kind of constitutional symptom whatever. When a large extent of surface is involved, as sometimes happens in very young and neglected children, the consequent irritation may set up a certain amount of fever; but this cannot be considered in any way as an essential feature of the disease.

[1] *Ann. de l'Instit. Pasteur*, Nov. 1887.
[2] *Trans. Hong-Kong Med. Soc.*

The elementary lesion of *P. contagiosus* is a large, hemispherical, clear, tense vesicle or bulla. It varies in size, from a split swan-shot to half a large marble. It commences as a minute papule, or rather speck of erythema, which rapidly, in the course of a few hours, passes on to vesication, the bulla or blister springing abruptly, and without any surrounding areola of congestion, from apparently sound skin. At first the bulla is tense, and filled with a clear fluid. In a few hours this fluid becomes somewhat cloudy, and the vesicle, which continues to enlarge at the periphery, becomes flattened and flaccid. At this stage it is usually accidentally ruptured. But although the fluid contents of the bleb escape, and the lesion cease to have the characters of a vesicle, the disease continues to spread eccentrically, as shown by the undermining of the epithelial layer of the skin. This undermining may gradually extend, before it dies away, over a circle an inch or two in diameter. The process then stops spontaneously, and by degrees the dried scarf skin is thrown or rubbed off, a pinkish patch of delicate new epidermis remaining for a time as evidence of where the disease had been. Meanwhile, by a process of auto-inoculation, fresh bullæ are developed wherever the hands or clothes have transferred the fluid from the original vesicle; and so the disease, unless properly treated, or unless the weather become cold or dry, is continued indefinitely.

It is evidently exceedingly contagious, for once introduced into a household or school, especially where there are many young children, it quickly spreads through the entire establishment.

In children, the bullæ may develop in any part of the integument; but in adults the axillæ are the parts most liable to be affected, although from time to time the characteristic lesion may show itself elsewhere. Thus I distinguish two principal types of *P. contagiosus*—an axillary or adult form, and a diffuse or infantile form. In the axillæ of adults, and also in the folds of the skin in fat children, the constant apposition of diseased surfaces tends to create considerable irritation; thus raw, weeping, angry-like patches, looking as if the epidermis had been removed by a blister, may be produced. The irritation in these places leads to scratching, pustulation, and scabbing, and often paves the way for the development of painful boils.

The successive crops of bullæ in young children are very apt to be mistaken for chicken-pox; but careful inquiry into the history of the case, and the absence of all constitutional symptoms at the outset, together with other and obvious considerations, ought to make the diagnosis an easy matter. In the adult the disease in the axilla is very apt to be mistaken for body ringworm; but the

presence of large vesicles—especially about the margin of the patch, the absence of a red, elevated, festooned border, and the absence of trichophyton elements in the scrapings, are sufficiently distinctive. Under the microscope, in stained preparations of the contents of the bullæ and of the epidermis, micrococci, arranged in pairs and fours, can usually be readily detected. Whether these bacteria constitute the virus of the disease I cannot say; doubtless, cultivation and inoculation experiments would readily settle the matter.

Treatment.—The attendants of children ought to be thoroughly impressed with the highly contagious nature of this complaint. All underclothing after being worn ought to be passed through boiling water. When a bullæ shows itself it should be opened, and the parts around and the spot itself thoroughly soaked with 1 in 2000 corrosive sublimate lotion, then dried and dusted with a powder of equal parts of boracic acid, oxide of zinc, and starch. This ought to be repeated several times a day. In the axillary form in adults the same treatment is speedily followed by a cure. As relapses are very liable to occur, both in children and in adults, the dusting powder ought to be used systematically, morning and evening, for some time after the bullæ have ceased to recur.

5. Verruga.

In certain narrow, steep-sided, confined valleys on the western slope of the Andes, between the 9th and 16th degrees of south latitude, and at an elevation of from 3000 to 10,000 feet above the sea-level, there is to be found a curious and often fatal disease, called by the natives "verruga," a Spanish word signifying a wart, and by the French "verrue peruvienne," or "bouton des Andes." This disease is characterised by an irregular, prolonged, and often intermittent febrile condition, attended by severe muscular, osteoscopic, and articular pains; and, also, associated sooner or later, if the patient survive, with the eruption of fungoid vascular growths on the skin and mucous membranes. It is inoculable, and frequently attacks dogs, cats, fowls, horses, mules, cattle, and pigs.

Although verruga has been known since the invasion of the land of the Incas by the Spaniards, and although it is a rare thing for any one, or any beast even, living for a length of time in the endemic area to escape an attack, very little mention was made of the disease, even in local medical literature, until recent years; and at the present day its morbid anatomy and pathology are still very imperfectly understood, and absolutely nothing is known about its

etiology; nor has any reasonable hypothesis been advanced in explanation of its peculiar endemicity.

For purposes of description, verruga may be divided into three stages—1st, the period of incubation; 2nd, the period of invasion and fever; 3rd, the period of eruption and convalescence.

1st. *The Incubation Period.*—This has been variously estimated at from eight to forty days. In the case of Carrion, a medical student who caused himself to be inoculated with blood from a verrucous tumour, symptoms of the disease began to show themselves on the twenty-second day after inoculation. Prolonged residence in the endemic districts is not necessary; there have been cases in which the disease attacked travellers who merely passed through the country, and neither ate nor drank during the journey.[1]

2nd. *The Period of Invasion and Fever.*—In addition to malaise, languor, headache, gastric derangement, and similar symptoms, fever is generally a prominent feature at the commencement of the disease. It is of a very irregular character, sometimes resembling a malarial remittent, sometimes being distinctly intermittent, like an ordinary ague, with a tendency to recur every afternoon, and having a cold, a hot, and a sweating stage; and, like a malarial fever, it is always associated with the rapid development of a profound anæmia. More characteristic of the earlier stages are the excruciating pains which harass the patient, especially at night. These are connected with rheumatic-like inflammatory swelling of the joints, large and small, which may be attacked one after the other. In addition to his articular sufferings the patient is racked with osteoscopic, rachialgic, and myalgic pains, often culminating in agonising muscular cramps and spasms. The anæmia gradually becomes very marked, the mucous membrane assuming a waxy white appearance, the skin looking grey, earthy, and coarse; hæmic bruits may be heard; languor becomes extreme, and dropsical effusions may take place. Dysphagia is a symptom frequently complained of. The liver enlarges somewhat and is tender, and the spleen may swell to three or four times its normal volume.

3rd. *The Period of Eruption and Convalescence.*—After these symptoms—the fever, the pains, and the anæmia—have persisted with varying degrees of intensity for a period of from twenty days to six or eight months, or even longer, the characteristic eruption begins to appear. When this comes out the constitutional symptoms become markedly ameliorated, particularly if the eruption be copious and rapidly evolved. The anæmia, however, persists, and if there be hæmorrhage from the tumours, may even be intensified. The erup-

[1] Dounon, *Arch. de méd. nav.* Nov. 1871.

tion usually appears first on the face and extremities; it then spreads to the trunk, and sometimes to the mucous membranes. It may either be discrete or confluent; and it may come out at once in one crop, or different parts may be affected at different times, so that the eruption may be fading at one part while it is progressing at another. From first to last the skin affection may remain out for several months.

The lesions are of two kinds, a cutaneous and a subcutaneous. The former commences as minute red specks or vesicles, which rapidly pass into bright red papules. These gradually increase until they attain the size of a very small pea. They are soft, and evidently very vascular, their dimensions being much affected by the state of the cutaneous circulation; muscular effort or warmth causing them to increase in size, tranquillity and cold causing them to shrivel somewhat. The eruption is very itchy, and the consequent scratching ruptures the verrugæ, which may then crust over and heal. Those not so destroyed remain in a stationary condition for some time; they then become darker in colour, and slowly subside, leaving a small dark spot which gradually, after desquamating, becomes lighter in colour and then disappears without mark or scar of any description. The eruption is usually most abundant on the face and extensor surfaces of the extremities, where it may be very abundant, even confluent. The crop of verrugæ on the trunk is usually much more scanty.

The subcutaneous growths are felt at first as minute, hard, smooth, movable tumours, like small peas underneath the skin. After remaining in this condition for a time they may gradually disappear; or, they may go on increasing in size. In the latter case they rise up as prominent tumours, the skin over them becoming dusky red, shining, adherent, and very itchy. After a time the skin gives way, and the morbid growth appears as a fungating fleshy swelling (Figs. 90 and 91) of a dirty grey or black colour, exuding an offensive sanguineous secretion. The skin surrounding the tumour is congested, and may so constrict the mass as to give it a mushroom-like shape (Fig. 92, *a*, *b*); in other instances the tumours are sessile (Fig. 92, *c*, *d*), being long (Fig. 92, *d*), hemispherical, or irregular in form. In bulk they vary from the size of a pea to that of an orange; and there may be only one or two, or they may be very numerous. Their favourite sites are the knees, the elbows, the face, the front and outer and inner surfaces of the legs, and the back of the malleoli; but any part of the skin may be affected. Sometimes these tumours may be converted into abscesses before they have broken through the skin, in which case an ulcer may

form. Sometimes they ulcerate after rupture of the skin; and if several in close contiguity thus break down, large sores are formed. They also may become encrusted and rupial in character. They are exceedingly vascular, and bleed spontaneously or on the slightest

FIG. 90.

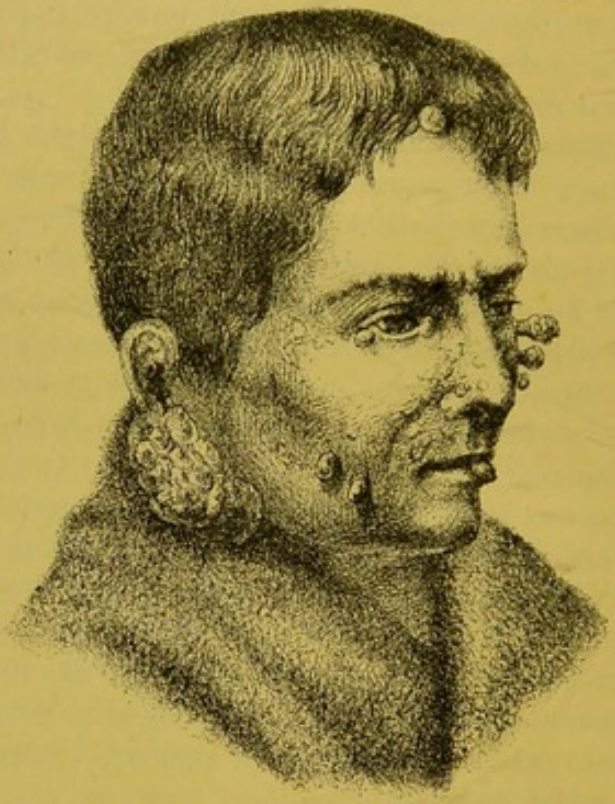


FIG. 91.—(After Dounon.)

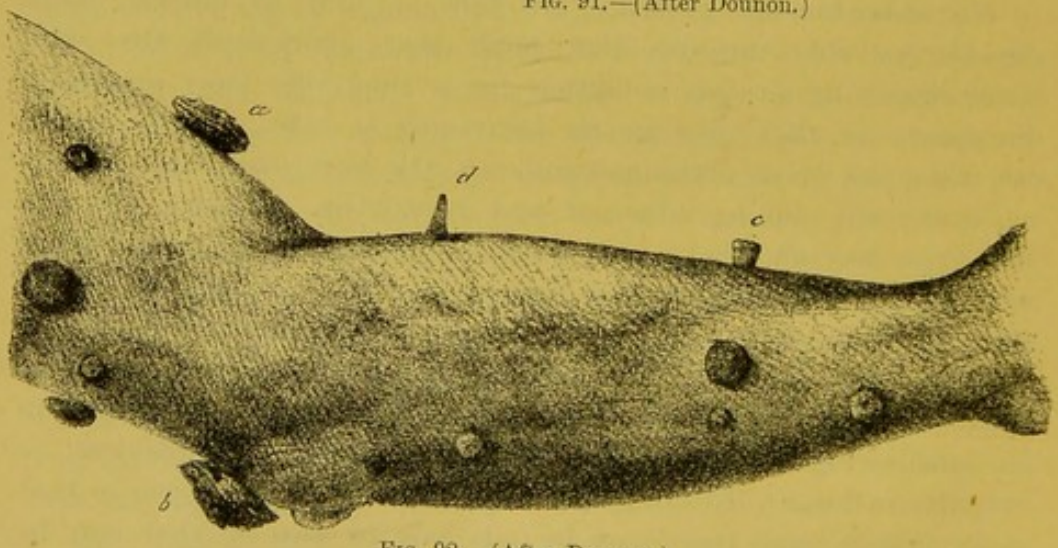


FIG. 92.—(After Dounon.)

provocation, draining the already emaciated and anæmic sufferer. Like the smaller tumours already referred to, they may gradually subside, or they may be destroyed by ulceration or sloughing, or they may be enucleated by the surgeon.

Dysphagia, and hæmorrhages from the bowels, and occasionally ascites may be explained by the presence of tumours in the œsophagus and stomach, and elsewhere in the alimentary canal and abdominal viscera.

Tumours may form on the conjunctivæ, in the nostrils, on the tongue, and on the mucous membranes of the mouth, pharynx, glottis, vagina, uterus, and bladder. Great inconvenience may result from growths in these situations; and in the case of tumours about the glottis, respiration may be seriously interfered with.

It would appear that a copious eruption relieves very much the urgency of the constitutional symptoms; and as cold has a repressing effect on the evolution of the skin lesion, high altitudes have an unfavourable effect in thus preventing its development. Moreover, a diminished barometric pressure favours hæmorrhage very much. Consequently, verruga, which at the sea-level is not a very dangerous disease, becomes exceedingly fatal in the higher valleys, its fatality apparently being directly related and in proportion to the lowering of atmospheric temperature and pressure.

Pathological Anatomy.—Salazar mentions that he found verrucous tumours in the stomach and on the surface and substance of the liver. The liver and spleen are both enlarged; but beyond this, apart from the skin eruption and visceral tumours just referred to, there are no special lesions to be found post-mortem.

The mass of the tumours is made up of small lymphoid cells (Fig. 93, *c*) held together by an exceedingly delicate fibrous stroma, and a capsule (Fig. 93, *b*) of concentrically arranged layers of connective tissue. The tumours are all very vascular; and in the case of the larger specimens the centre of the mass is cavernous (Fig. 93, *d*), the large spaces being filled with blood and communicating with each other. Some tumours are entirely made up of this cavernous tissue. The smaller kinds spring from the papillary and more superficial layers of the skin, the larger from the subcutaneous tissue, and the medium sized from the deeper layers of the skin.

According to Isquierdo, a bacterium is to be found in the morbid growths in great profusion. It is a little larger than the tubercle bacillus, and is made up of closely set, spore-like bodies enclosed in a sort of sheath; the spores are so closely set that the bacterium has the appearance of a streptococcus. These microbes were found between and not in the cells. In most tumours many of the capillaries are varicose, distended, and stuffed with a micrococcus, which is also to be met with in the vessels of the apparently healthy skin in the neighbourhood.

Etiology.—Neither age, sex, nor race have much influence as regards verruga; a very few seem to be proof against it.

Although it is generally supposed that the disease is confined to Peru, it is just possible that its distribution is not quite so restricted. Beaumannoir[1] describes a case which he saw in Réunion, and which, after having seen the Peruvian disease, he believes to have been genuine verruga; the eruption, fever, pains, and anæmia were in his opinion, identical. Similarly, Dr. de

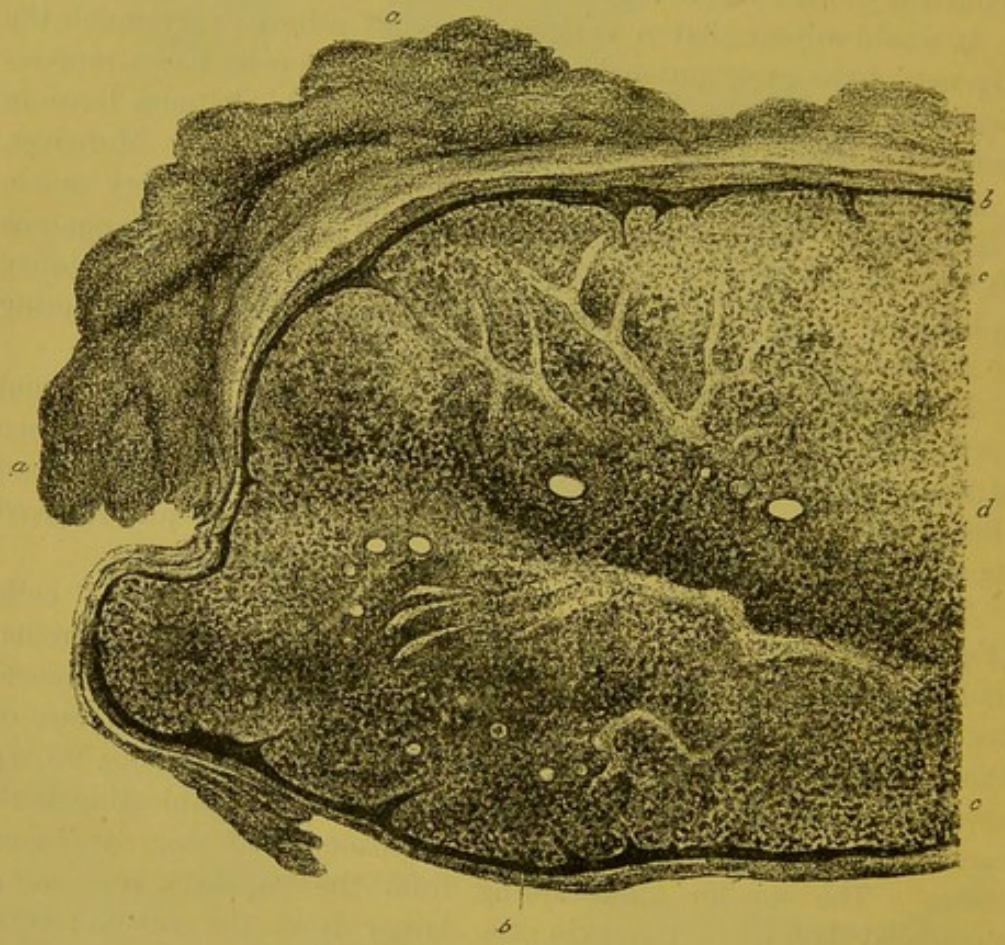


FIG. 93.—(After Dounon.)

Haviland Hall, described a disease which a correspondent informed him is to be met with in Zaruma, Ecuador, and which, if not identical with verruga, at all events closely resembles it.

Although the fever of verruga is very like some of the forms of malarial fever, yet it cannot be of this nature, as although malarial fever is common in many of the valleys of the Peruvian Andes, it is only in a very few of these valleys that verruga is found.

There is a native idea that the disease is contracted by drinking

[1] *Arch. de méd. nav.* Jan. 1891.

the water of particular springs and streams. In the valley of Huarochiri, the centre of the endemic district, there is a stream which is actually called Agua de Verrugas, and the natives firmly believe that its waters confer the disease. There are several other streams in the neighbourhood which have an equally evil reputation; but whether this is well deserved or not, in the absence of all scientific demonstration or body of well-observed facts, it is impossible to say.

Since Carrion's fatal experiment there can be no doubt about the inoculability of verruga; but the medical profession in Peru evidently does not regard contagion as the ordinary or a likely mode of contracting the disease. In the hospitals of Lima patients suffering from verruga are treated in the general wards, and they do not appear ever to communicate the disease to the other patients.

One attack seems to confer permanent immunity, as a general rule. There must be exceptions to this, however. Mr. Hutchinson[1] had a patient in the London Hospital who, five years previously, suffered from verruga while working in a silver mine at Agua Verrugas, Peru. Seven months prior to his admission he left South America. During the voyage to England he contracted dysentery, and it was for this disease that he came to hospital. Five weeks after admission the verruga eruption showed itself, and was readily recognised by the patient himself.

Mortality.—There are no statistics from which reliable conclusions on this point can be drawn. The influence of temperature and altitude has already been referred to. Undoubtedly, many die of the fever before the eruption comes out. A fever, called Oroya fever, has proved very fatal to the workers on the Oroya railway, which traverses the verruga country. Since Carrion's experiment, which eventuated in his case in the development of a fever exactly like the Oroya fever, this disease is believed by many of the Peruvian physicians to be the initial fever of verruga. It is very deadly.

After the appearance of the eruption the risk to life seems to diminish, but there is still considerable danger from hæmorrhage from the tumours, and from septic trouble in connection with the unhealthy and extensive sores.

Treatment.—There is no specific treatment for verruga, and no suitable prophylaxis other than that of quitting the endemic area. When the disease appears all that can be done is to maintain the strength of the patient by a generous and suitable diet,

[1] *New Sydenham Soc. Atlas of Portraits of Diseases of the Skin.*

and to keep the skin as aseptic and clean as possible. Gangrenous and sloughing tumours should be removed; should hæmorrhage occur, styptics and graduated pressure must be employed. Transfusion or saline injections may be necessary. Iron, arsenic, and other tonics are indicated, especially during the eruptive stage and during convalescence. If possible, the patient should be transported to a lower altitude, as it has been found by experience that, not only are the pains and tendency to hæmorrhage increased by high altitudes, but the chances of recovery are very much diminished thereby.

SECTION IV.—UNCLASSED SKIN DISEASES.

1. Prickly Heat (Lichen Tropicus, Miliaria Papulosa).

Owing to the unwonted physiological strain thrown on it by the high atmospheric temperature, the skin of most Europeans on their entering a tropical climate for the first time, and even the skin of some of the natives, becomes affected with what is called "prickly heat." In some the trouble recurs every hot season; but, as a rule, unless under unusual circumstances, such as great exposure to the sun, especially if combined with violent exercise and profuse consumption of fluids,—in other words, unless an unusual amount of sweating is induced, the crop of prickly heat becomes yearly less and less troublesome, and finally, after a few years, ceases to come out altogether. In most people the skin seems in time gradually to accommodate itself to the physiological demands the heat makes upon it.

Prickly heat consists of a crop or succession of crops of minute, slightly-raised, acuminated papules. Though often very closely approximated the papules are discrete, with here and there between the tiny elevations, which feel like grains of sand on the skin, small shining vesicles scarcely the size of a pin's head. The skin of the back,—especially between the shoulders,—the forehead, the scalp, the neck, axillæ, chest, abdomen, arms, and backs of hands are favourite seats for the eruption; the legs and face, although by no means always exempt, being more rarely affected. The development of the eruption is attended with intense itching of a stinging, pricking character, always much aggravated by anything which tends to cause sweating, such as exercise,—especially in the sun,—large draughts of fluid—especially hot fluid, such as tea, soup, etc. The eruption may keep out or recurring during an entire hot season. A decided fall of temperature rapidly effects a cure. When the

irritation and congestion of the skin subside, there may be a little roughness and branny scaling, but in a day or two all trace of the disease clears away.

Although generally an affair of little importance as affecting health, under some circumstances prickly heat becomes a serious disease, and must not be despised. Especially is this the case when it attacks young and delicate children, parturient females, and sick persons generally. In these cases the irritation from the eruption is often so great that sleep becomes impossible almost, and a low, feverish, restless, nervous condition, exceedingly prejudicial to recovery, is induced. In severe cases, boils, intertrigo, and other eczematous conditions are very apt to supervene, and much suffering ensue.

The pathological condition in prickly heat is one of congestion or inflammation of the sudoriparous glands, from hyper-activity. This is the elementary lesion; but the scratching, scraping, and rubbing provoked by the intense irritation is apt to bring about pustulation, vesication, scabbing, and various degrees and kinds of traumatic dermatitis.

Treatment.—Everything tending to induce sweating—such as violent exercise, hot rooms, thick clothing—must be avoided. The consumption of fluid must be reduced to a minimum, and what fluid is taken should be taken cold, and never in large draughts at a time. Hot tea, hot coffee, hot soup, and alcoholic drinks are most prejudicial, and their consumption is generally immediately followed by an outburst of perspiration and an aggravation of the pricking and stinging of the skin. Night clothing ought to consist of light flannel, or better, of a gauze singlet and thin pyjamas. The mattress and pillows should be hard, and covered with a fine matting. Some device for keeping opposing surfaces of the body from coming in contact should be adopted in all severe cases, or as a matter of habit, in hot weather. A long, hollow cylinder, 8 inches by 4 or 5 feet, of open rattan basket work,—what is known in the East as a "Dutch wife,"—over which the arms and legs can be thrown, is a great comfort in hot, steamy weather, often enabling the victim of prickly heat to procure a little sleep.

There have been many local remedies proposed; but without self-denial in the matter of fluid consumption these are of little avail. Opposing surfaces, such as the axillæ, crutch, under the breasts in females, and in the folds of the skin in children and fat persons, should be well powdered after the morning bath and before going to bed with a dusting powder of equal parts of powdered boracic acid, starch, and oxide of zinc. A solution of sulphate of copper, 10

grains to the ounce, is highly spoken of by French;[1] he directs that it should be applied with a sponge daily after the bath. Another application recommended by Lesinge[2] I have sometimes found of use; it consists of sublimed sulphur 80 parts, oxide of magnesia 15 parts, oxide of zinc 5 parts, and is applied by means of a wet sponge, and gently rubbed into the affected areas for some ten or fifteen minutes daily. Solutions of carbolic acid relieve the itching somewhat, and so do zinc or calamine lotions.

Mild saline purgatives occasionally may be beneficial; but the cooling drinks so commonly recommended, inasmuch as they are sure to be rapidly excreted by the skin, can only be prejudicial.

2. Keloid.

Although occasionally met with in Europe, keloid is a comparatively rare affection in the fair-skinned races. The remarkable

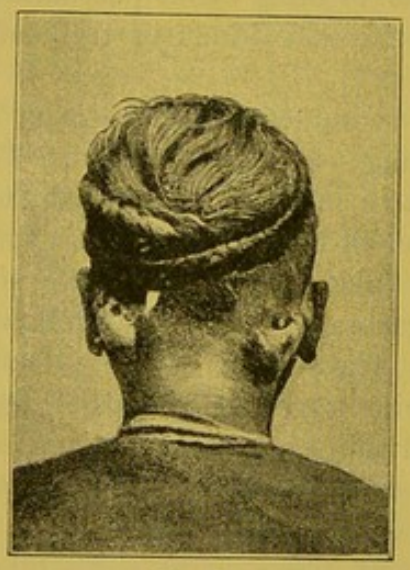


Fig. 94.

Fig. 95.

tendency displayed by cicatricial tissue in the integument of the black and yellow-skinned races to take on this form of growth has long been remarked. Frequently with them the scar of nearly every wound or sore, no matter how trivial, becomes keloid, more particularly if the healing has been long delayed by accidental or intentional irritation. Thus the scars resulting from vaccination, from pustules (Fig. 94), or from boils, from ear-piercing (Fig. 95), and other trifling causes, may become thick and indurated, pushing out claw-like processes into previously healthy skin. The resulting

[1] *Lancet*, August 19, 1876. [2] *Ibid.* May 5, 1872.

tumour may assume large and disfiguring proportions. I have frequently observed that the disposition to take on a keloid action seems to be symmetrically located in the skin. Thus, if one ear-ring hole—a favourite site—becomes keloid, it is generally found that the other ear is similarly affected; or if the scars left by a pustular eruption on the chest or head become keloid, those so affected are more or less symmetrically disposed. The natives of certain parts of Africa avail themselves for purposes of love, social distinction, or war, of this pathological peculiarity of their integuments to work symmetrical patterns of keloid growth on their bodies (Fig. 96). A similar habit obtains among certain tribes of aboriginal Australians. They cut themselves with flints along the lines of the bony skeleton, and afterwards irritate the wounds by rubbing into them the juices of certain plants, with the result that in time a keloid outline of the skeleton is produced, an effect calculated to intimidate their enemies.

Keloid is not amenable to treatment; even if excised it returns.

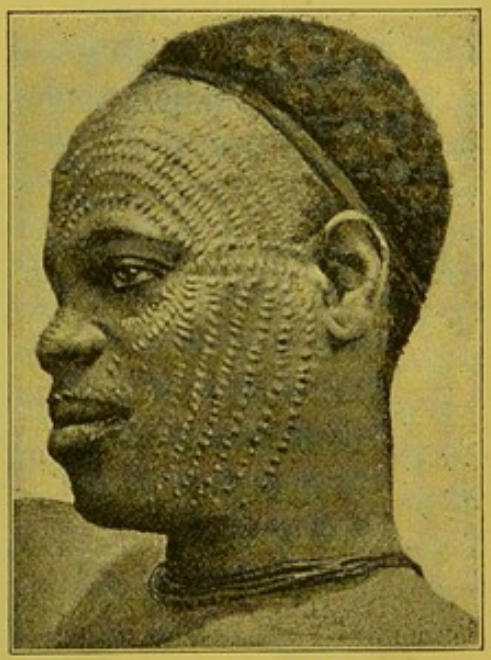


Fig. 96.—Artificial keloid in a Congo negro. (From a photograph by Dr. Grattan Guinness.)

3. Ainhum.

Ainhum (a Nagos negro word signifying *to saw*) is a disease more curious than important. Hitherto there is no record of its occurrence in an European or Mongolian, but it is common enough among the negroes of certain parts of tropical Africa, and occurs occasionally among those of the West Indies, Brazil, and the United States of America; it has also been seen in Madagascar, in Réunion, and in one or two places in India—Dacca, Pondicherry. Whether from hereditary or endemic causes, in some instances it seems to have a predilection for particular families. It is most common in adults and in the male sex, although children and females are by no means exempt.

The essential feature of the disease is the slow amputation of one or more toes—usually the little toes—by a deepening, non-ulcerating furrow (Fig. 97, *b*, *d*) which surrounds the base of the affected member.

Although the little toe is the one usually affected, it does occa-

sionally happen that the fourth toe is also attacked. There is only one well authenticated instance of another toe being affected; in this instance it was the second toe. Sometimes both little toes are symmetrically involved, sometimes one is attacked after the other, and sometimes only one suffers. The amputation may be through a joint, or in the continuity of a bone. Gongora [1] has recorded a case in which, after the second and third phalanges had been thrown off, the disease recurred in the stump, which in due time was also removed.

Ainhum commences by the formation of a narrow groove on the inner and plantar aspect of the digito-plantar flexure of the little toe. This groove gradually deepens and, without sign of inflammation or irritation, as it deepens extends to the inner aspect of the toe, then on to the upper, and finally to the external surface. As the groove deepens the distal part of the toe increases in size, as if hypertrophied by slow strangulation. It is not altered in colour, however, but is smooth and plump, and perhaps two or three times the natural size, looking like a plum or a small potato.

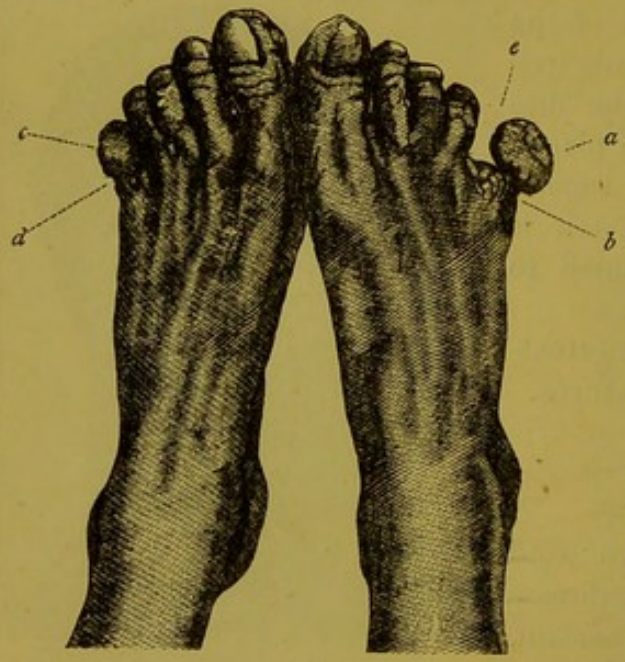


FIG. 97.

The dorsal surface of the toe tends to become everted, so that after a time the nail (Fig. 97, *a*, *c*) comes to look outwards. By degrees the neck of tissue connecting the toe with the foot becomes so narrowed that the part waggles about freely in all directions, getting in the way, and interfering with locomotion. As the result of injury or of dirt, ulceration may ensue in the depths of the constricting furrow, and finally, if not previously removed by accident or surgical interference, the toe drops off spontaneously or becomes gangrenous. The progress of the disease is very slow, lasting, from the commencement of the groove to the shedding of the toe, from one to ten years, or even longer.

On section of the amputated member, it is found that the dermal

[1] *Arch. de méd. nav.* 39.

tissues are but slightly altered. The panniculus adiposus is very much hypertrophied, and the bones are infiltrated with fatty matter, thinned, or altogether absorbed. At the seat of constriction a band of fibrous tissue, more or less intimately connected with the derma, is found to surround, in whole or in part, the pedicle.

Several speculations as to the nature of this singular disease have been indulged in, but it cannot be said that as yet the problem has been satisfactorily solved. Some have suggested that it is artificially produced by ligature, with the object of malingering. But such an explanation does not fit in with the facts, seeing that, in the first instance at all events, only a limited part of the circumference —the inner and plantar quadrant—is affected; moreover, it occurs in some individuals who can have no object in thus mutilating themselves. Another view is that the constriction is brought about by a linear scleroderma; but Corre points out that the fibrous band lies below and not necessarily in the derma during the earlier stages. The same writer suggests that the lesion may be a tropho-neurosis; but he brings forward very little, if any evidence in support of this hypothesis; symmetry is not sufficient evidence for such an origin, for symmetrically-disposed tissues are liable to be symmetrically attacked by non-constitutional or non-nervous disease, *e.g.* ringworm, keloid, itch, and many other forms of external parasitism. Possibly the starting-point of the disease is a wound or irritation of some sort—to which the little toe, from its position, is particularly exposed—which afterwards starts a fibrotic change analogous to keloid, an affection to which, as is well known, the African race is particularly liable.

Treatment.—When the disease has advanced so far as to render the toe useless and inconvenient, it ought to be amputated. In the earlier stages, division of the fibrous band at the bottom of the groove in one or two places might possibly delay the progress of the strangulation.

APPENDIX.

Malaria, Medium of Infection, p. 144.—The editor overlooked to mention, in the section on the Etiology of Malaria, that milk has been recognised as a medium for the malarial infection in the United States; see Pepper's *System of the Practice of Medicine*, Philadelphia, 1885, vol. i. p. 590.

Dysentery, transportable and in some instances spreads from the sick to the healthy, p. 559.—Since the article on dysentery went to the press, an account has appeared of a disastrous epidemy of that disease which deserves to be recorded, although unfortunately the details are defective. The following extract should be read in connection with the observations on other epidemies given on pp. 557–60:—"I have intelligence from Futuna, one of the New Hebrides group of islands in the Pacific Ocean, some 1400 miles from Sydney, detailing the ravages of an epidemic of dysentery of the most malignant and fatal type. . . . A labour vessel from Queensland landed a Futunese woman with a half-caste child, suffering from dysentery. The disease soon spread; it cut off one-fourth of the population. It has destroyed so many of the children, that in other ten years the Futunese people will be extinct. Dr. Gunn and his wife have recovered, but two of their children have died. . . . In 1842 the same disease attacked the Futunese, when they killed the Christian teachers from Samoa; now the majority are more intelligent, but the small race is doomed."—*Scotsman*, July 31, 1893.

This is only another illustration of the importance of epidemic dysentery, and the necessity for bestowing on it a more careful study than it has hitherto received.

Life History of *Bilharzia hæmatobia*.—At Gabes Dr. Sonsino obtained a sufficient supply of ova for the experimental rearing of

embryo *Bilharzia* in association with different kinds of fresh-water animals (molluscs and arthropods). The following extract from the *Lancet*, September 9, 1893, gives the result of his researches respecting the life history of the *Bilharzia hæmatobia*:—

"After many experiments I succeeded with a small crustacean (amphypoda), and obtained evidence that this same crustacean is an effective intermediary host of *Bilharzia*, and so discovered the secret of the life history of the African parasite. Not being quite satisfied on all points, I did not at once publish the discovery, but contented myself, in view of establishing the date of my discovery, with sending a sealed letter on August 3rd to the Secretary of the Società Toscana di Scienze Naturali in Pisa. The conclusions therein detailed I have now decided to publish. They were as follows:—1. *Bilharzia hæmatobia* has a life history differing from the typical one of the digenetic trematodes as represented by *Fasciola hepatica*. 2. Its life history is less complicated than that of the digenetic trematodes; it requires an intermediary host and undergoes a metamorphosis, but there is no alternation of nor asexual generation. 3. In this way it resembles in its life history the holostomes rather than the distomes. 4. The intermediary host is a small crustacean. 5. The free embryo, which swims actively about, on encountering the crustacean attacks the latter at a vulnerable point, and, by means of the papilla at its head, bores and forces its way into the body of this animal after having rid itself of its covering of cilia. Having effected an entrance it proceeds to encyst itself. 6. The part of the crustacean in which the *Bilharzia* cysts are most frequently located is that corresponding to the first segment and near the eye. 7. The encysted larva, being transferred with the crustacean in drinking water to the human stomach, is then set at liberty. Afterwards, penetrating the intestinal walls, it arrives in the portal vein, where, presumably, it completes its development. After I had despatched these conclusions, I continued my researches in Gabes, and discovered that the larva of an insect (probably a species of ephemerida) was also an efficient intermediary host. With the view of making control experiments I proceeded to Gafsa, as I had been assured by the principal medical officer of the military hospital of that place that Bilharzia disease was endemic there. Here I had access to several cases of indigenous origin. I found that not only is the disease very common in Gafsa in the native population, but that the cases were sometimes of a grave character, such as I had previously frequently met with in Egypt. I may say that my researches in Gafsa confirm the conclusions above detailed in so far as I

succeeded in rearing *Bilharzia* embryos in the aquatic larva of an insect; but, strange to say, I did not meet with this insect larva at Gabes; nor did I succeed in finding in Gafsa the larva of the insect nor the crustacean which served me as subjects for my experiments at Gabes. Thus I concluded that several kinds of aquatic arthropoda may act as efficient intermediary hosts for *Bilharzia*, whilst molluscs seem to have nothing to do with its life history. I may add that in these intermediary hosts the cysts of *Bilharzia* resemble very closely the asexually generated cysts of young distomes. They are globular, having a double outline and a diameter of from 0·075 mm. to 0·120 mm. If the included parasite be not moving spontaneously, movement may be elicited by adding to the slide a little warm water. The cyst is elastic and compressible, but it is difficult to rupture. Up to the present I have failed to liberate the parasite without completely destroying it. As seen in the cyst it presents no features by which it can be distinguished from other kinds of encysted distomes, with the exception that its integument is firmly and characteristically beaded, just as in the earlier stage of the embryo *Bilharzia*, as described by Cobbold[1] in 1872, and as I figured it in my first paper[2] on this parasite in 1874. I may mention that as regards the anatomy of the embryo *Bilharzia* I can confirm what has quite recently been announced by Railliet,[3] but which I have long known, that whilst still *in ovo* it is provided with two pairs of ciliated infundibula, one pair being in the anterior third and one pair in the posterior third of the body. These infundibula are very apparent, and are to be best seen by slowly lowering the focus of the microscope whilst examining the embryo. As the larval phase of *Bilharzia* found encapsuled in the intermediate host is, in a biological sense, different from the encysted cercaria phase of the typical distomes,—inasmuch as it is a direct encystment of the embryo, and not, as in the case of the cercaria of the distomes, the encystment of the asexually generated progeny of a redia developed from an embryo distome,—it is desirable to provide this larval encysted stage of *Bilharzia* with a distinctive name. As the corresponding larval phase of the holostomes—which, as regards this part of their life history, *Bilharzia* closely resembles—is designated "tetracotyle," the term "dicotyle" appears to be

[1] "On the Development of *Bilharzia hæmatobia*," being a paper read before the Metropolitan Counties Branch of the British Medical Association on May 17th, 1872. *Brit. Med. Journ.*

[2] "Ricente intorna alla Bilharzia hæmatobia in relazione colla Imaturia Endemica in Egitto," *Rendiconto della R. Accademia della Scienze di Napoli*, 1874.

[3] "Observations sur l'embryon du Gynæcophorus hæmatobius Bilharzia." Par A. Railliet, Paris, 1892. *Bulletin de la Société Zoologique de France*, 1892, tome xvii. p. 151.

appropriate, the *Bilharzia* larva having only two suckers. The insect aquatic larvæ in which the dicotyle of *Bilharzia* is found encysted have a length of 7–8 mm. and a thickness of about 0·3 mm. The crustacean is about the same size. The magnitude of these intermediary hosts renders it easy by a little attention being paid to the drinking water to preserve a community from infection by *Bilharzia*, as the investigation I have made would tend to show that the drinking water which is obtained from pools, tanks, and streams which contain arthropoda is the medium by which this parasite gains access to the human body, and it is probably the only medium; therefore bathing in such water, if the bather should swallow the water, is dangerous. Filtration, even rough filtration through linen, will afford complete immunity. It will thus be seen that the rules I laid down many years ago for the prevention of Bilharzial infection, which were repeated in a paper read by me at the last International Congress of Hygiene (1891), and which appeared in the *Lancet* of August 22nd and 29th, 1891, are still appropriate."

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INDEX OF SUBJECTS.

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