

Spirillum fever : synonyms famine or relapsing fever, as seen in western India / by H. Vandyke Carter.

Contributors

Carter, H. V. 1831-1897.
Royal College of Physicians of Edinburgh

Publication/Creation

London : J. & A. Churchill, 1882.

Persistent URL

<https://wellcomecollection.org/works/rnw29wme>

Provider

Royal College of Physicians Edinburgh

License and attribution

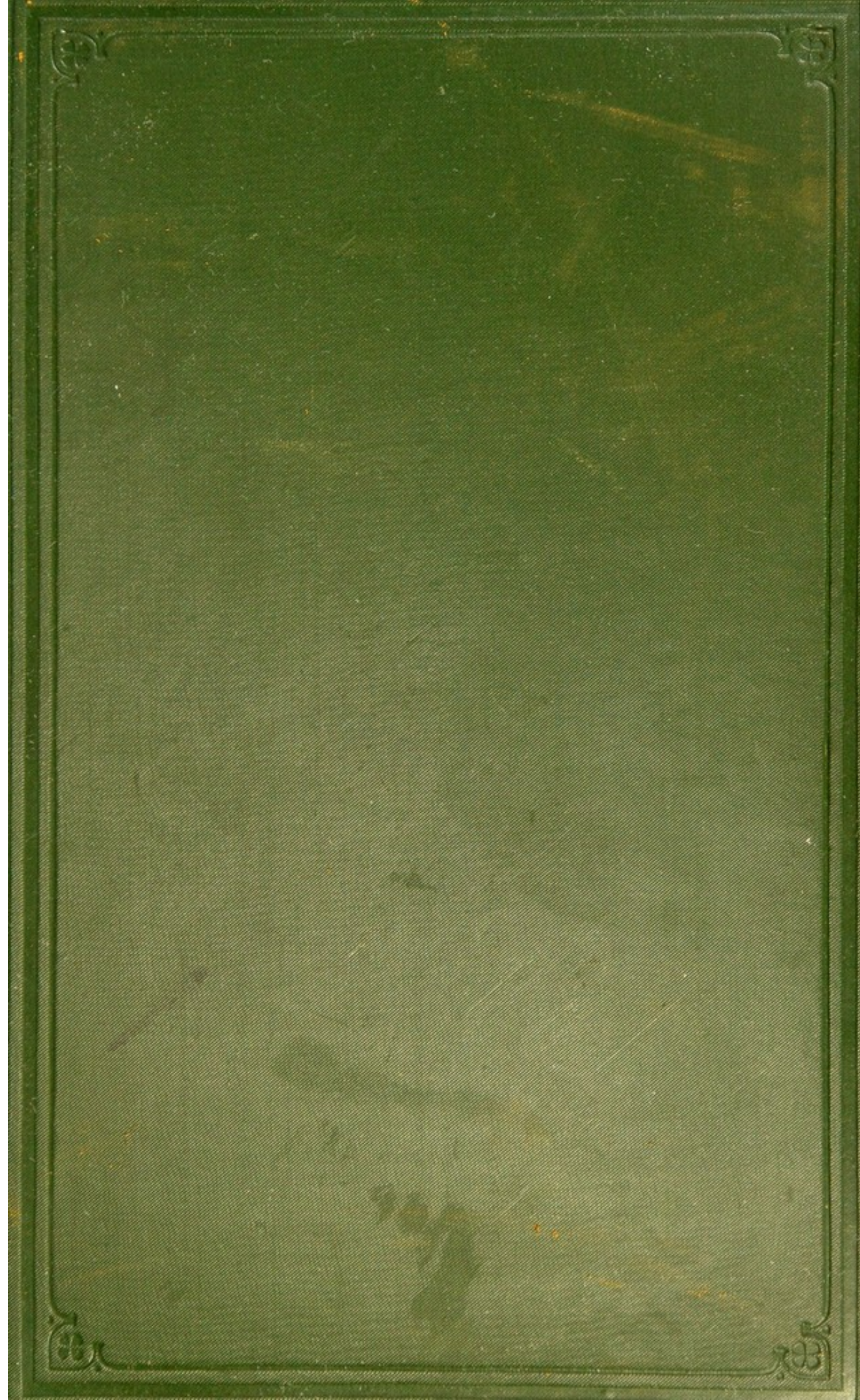
This material has been provided by This material has been provided by the Royal College of Physicians of Edinburgh. The original may be consulted at the Royal College of Physicians of Edinburgh. where the originals may be consulted.

This work has been identified as being free of known restrictions under copyright law, including all related and neighbouring rights and is being made available under the Creative Commons, Public Domain Mark.

You can copy, modify, distribute and perform the work, even for commercial purposes, without asking permission.



Wellcome Collection
183 Euston Road
London NW1 2BE UK
T +44 (0)20 7611 8722
E library@wellcomecollection.org
<https://wellcomecollection.org>





Ms. 7.15



21/-

W. A. M. E. N.

Fb * 7.15

R39997

SPIRILLUM FEVER

RECEIVED

SPIRILLUM FEVER

SYNONYMS

FAMINE OR RELAPSING FEVER

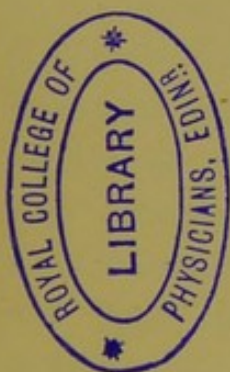
AS SEEN IN WESTERN INDIA

BY

H. VANDYKE CARTER, M.D. LOND.

SURGEON-MAJOR I.M.D.

SURGEON IN CHARGE OF THE GOCULDAS TEJPAL HOSPITAL,
LATE OFFICIATING PRINCIPAL AND PROFESSOR OF MEDICINE, GRANT COLLEGE, AND
FIRST PHYSICIAN, JAMSETJEE JEJEEBHAY HOSPITAL, BOMBAY



LONDON
J. & A. CHURCHILL
NEW BURLINGTON STREET

1882

SPIRILLUM FEVER

STUDIES

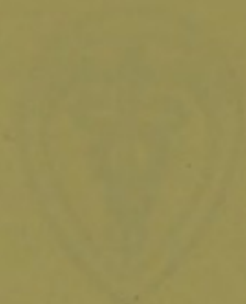
IN THE HISTORY OF THE

SPRUE OF THE

SPRUE OF THE

SPRUE OF THE

SPRUE OF THE



1900

NEW YORK

NEW YORK

1900

PREFACE.

THE following data were collected at Bombay between April, 1877, and March, 1880 ; and during this interval (which includes the height and decline of the late fever-epidemic) having been placed in executive or administrative charge of both local Native Hospitals, I had ample opportunity for clinical observation.

Genuine famine or relapsing fever had not been previously recognised in Western India ; and early perceiving the sanitary importance of establishing its identity, I spared neither leisure nor effort to procure the materials needful for adequate diagnosis. Whilst well aware of the imperfections of the record ensuing, I have endeavoured to note and duly estimate the prominent points of this remarkable disease ; and to ensure accuracy, no instances are here analysed except as such by means of the microscope were demonstrated to belong to the spirillar infection. Experience leads me to insist upon the desirability of careful examination of the blood, on all practicable occasions ; and upon its absolute necessity, in cases deviating from the average state. The spirillum fever of man, may not long retain its peculiar suitability for advanced clinical research ; but, as yet, it affords the best known data for fresh pathological inference.

I had the benefit (if so it can be termed) of repeated personal experience of this fever : unfortunately, the last of these attacks brought to a premature close some more recondite enquiries I hoped to undertake.

Amongst the many obligations I am under for collateral aid, should be mentioned the valued co-operation of my colleagues at the J. J. Hospital, both European and Native ; also the judicious suggestions of the late lamented Dr. Murchison, and of Dr. J. Burdon Sanderson ; and the generous friendliness of Dr. Ferdinand Cohn of Breslau, and Dr. Robert Koch (then of Wollstein) : besides other equally appreciated assistance later rendered at Geneva, Rome and London.

Such references as time permitted to the recent literature of Relapsing fever, are embodied in the text ; they serve to complete the proof that the Spirillar infection is one and the same disease, in both East and West.

A few parts of the following Chapters have already appeared in the Transactions of the Royal Medical and Chirurgical Society of London, and in those of the recent International Medical Congress.

The two Appendices contain data supplementary to and illustrative of the main topic.

CONTENTS.

SECTION I.

HISTORY OF THE EPIDEMIC.

CHAPTER	PAGE
I. History of Fever sickness in the Mofussil	I
II. History of the Fever epidemic in Bombay	14

SECTION II.

CLINICAL HISTORY OF THE SPIRILLAR INFECTION.

I. Source and sum of Data	31
II. Definition and Clinical Summary. Illustrative cases.	33
III. General phenomena : prodromata, physiognomy, body-weight ; acme, crisis and lysis. Special symptoms : headache, pains, thirst, appetite, vomiting, sweats, sudamina ; the tongue and state of bowels. Heart, pulse, lungs ; liver, spleen, urine	69
IV. Description of the Pyrexia. Definitions. Normal temperature and pulse. Abortive and relapsing series. Pyrexia at succes- sive stages of ordinary spirillar disease ; comparisons of tem- perature and pulse. Variations of pyrexia at the successive stages and in fatal cases	125
V. Complications of spirillum fever. Secondary fever, affections of the nervous, respiratory, circulating and digestive systems, of the urinary organs ; abortion	170
VI. Antecedents and Sequelæ	228
VII. Clinical modifications of spirillum fever. Essential and in- cidental ; modifications from conjoined enteric, typhus and malarious fevers. Bilious typhus	231
VIII. Mortality of spirillum fever. General features—1. Death-rate. 2. Date of Death. 3. Apparent cause of Death. 4. Mode. Detailed features. 5. Influence of Sex. 6. Age. 7. Season. 8. Period of epidemic. 9. Social station. 10. Birthplace. 11. Race. 12. Habits. 13. Previous disease. 14. Mental depression. 15. Bodily exhaustion	241

CHAPTER	PAGE
IX. Anatomical lesions. Introductory observations. Summary. Description of lesions. Lesions according to stage of fever : final remarks. Memorandum on European observations	248
X. Diagnosis. Recognition of Relapsing fever ; its identification ; its clinical discrimination	300
XI. Prognosis	321
XII. Treatment	325

SECTION III.

PATHOLOGY OF SPIRILLUM FEVER.

I. Aspects of the Blood, general and special. Methods of examination. General description of the blood ; normal aspect ; appearances according to stage of disease. Detailed description of blood-elements according to stage of disease. Account of the spirillum	333
II. Etiology of spirillum fever. Predisposing causes. Contagion	369
III. Nature of the Disease. Explanation of its essential relations, its characteristic symptoms, and its epidemiological conditions.	411
APPENDIX A. 1. Artificial production of spirillum fever in the Monkey. 2. Some Culture-experiments	429
APPENDIX B. Concurrent Fevers at Bombay. Cerebro-spinal meningitis, Typhus, Enteric fever, Ague, Remittent fever	436
INDEX	447

ILLUSTRATIONS.

Chart No. I. of Famine and Fever data	30
Sphygmographic Pulse-tracings	102
Skin-eruption	194
Correlated data of First Relapse, Charts II. and III. ¹	358
Microscopic Appearances of the Blood, Lymph, Heart-muscle, Culture-products	368
Temperature-charts. Plates IV. V. VI. and VII.)	<i>At the end</i>
Temperature-charts of the Monkey. Plate VIII.)	

¹ By oversight numbered 1 and 2 respectively.

SPIRILLUM FEVER

SECTION I.

HISTORY OF THE EPIDEMIC.

CHAPTER I.

HISTORY OF FEVER SICKNESS IN THE MOFUSSIL (COUNTRY DISTRICTS).

Normal Data of Western India.—The Presidency is mainly inter-tropical, extending over 125,156 square miles, and having a population of about 17,000,000. Excluding the province of Sind, its major part lies coastward, and rarely suffers from want of rain ; but there is a large area of raised upland (known as the Deccan plain), where, as the country recedes from the western sea-line, the rainfall gradually diminishes from 40 to 20 inches per annum, or less. It is here that the late famine occurred, the nine affected districts embracing a continuous area of 54,355 square miles, with a population of 8,000,000 ; of these, however, only five suffered severely, viz. Sattara (a part), Belgaum, Dharwar, and especially the more easterly sections of Sholapore and Kulladghi; the whole lie between N. latitude 14° and 18°, and have an area of 33,873 square miles, with a population of 5,000,000, or about 147 individuals to the square mile. Here the soil is mostly shallow, and derived from trappean rock ; tree vegetation is sparse, and the incipient rivers comparatively small. Cultivation in this semi-torrid region chiefly depends upon rainfall, which, in proportion as it decreases is also apt to fluctuate in amount and distribution ; the result being an annual liability to variation in the harvest supply. Particularly is the meteorology of the last named eastern districts worthy of attention, owing to their position upon a nearly vertical line where the two alternate monsoons of peninsular India may be said to meet and subside ; and either rainy season failing the land must suffer. Mean temperature of air 75° to 80° F., mean

monthly range 60° to 90° , or wider; mean humidity of year about '60, monthly mean '45 to '86, the minimum often less; barometric pressure 27.5 in., the mean elevation being about 2,000 feet above sea-level.

The inhabitants of the affected districts are mostly Hindoo Marattas, other races and Mussulmans being found to the east and south. A very small proportion of the Hindoos are strict vegetarians, but animal food is nowhere largely consumed. The great majority of the people are agriculturists, the remainder consisting of village officials, menials, and petty traders; there are few large landholders or manufacturers on a wide scale, or populous towns; and as but one line of rail exists, transport is mainly effected by bullock-cart and pack-animals. The population is habitually too poor to store either food or money; and marriage being religiously inculcated, families abound. The common buildings are of clay, seldom of brick, not raised, and usually crowded; regular village or house drainage is unknown, and the water supply rarely selected. The people, however, are not deficient in comfort and intelligence; they are industrious and abstemious, and preserve strict social discipline.

In average years the population is not unhealthy, and it tends rather to increase; the normal death-rate may be not more than 25 per mille per annum. The prevalent disease is 'fever;' 'small-pox' and 'cholera' are nearly endemic; 'bowel complaints' are common; under these four heads, together with 'injuries' and 'other causes,' all the deaths are registered according to a plan now several years in operation, under native unprofessional agency. The returns are supervised by the Sanitary Commissioner and his assistants, who are commissioned medical officers.

Abnormal Events in the Deccan Famine Districts.—The localities named above were free from distress and unusual disease, until the abrupt climatic derangements of 1876-77; and thence followed in succession drought, dearth, and disease. In the first of these years the rainfall, beginning as usual in June ceased generally in August, or long before the crops were grown; and in 1877, the same rainy season (S. W. monsoon) was early interrupted, not until the autumn being supplemented by the N. E. monsoon; so that for a second time the chief grain harvests almost totally failed, the loss being such as could not be at once made up even with good years. After September 1877, heavy rain fell, but where not excessive, it was of little immediate avail; and the subsequent seasons of both 1878 and 1879, proved to be not particularly favourable. The resulting scarcity was especially felt in Sholapore and Kulladghi, where the total rainfall in 1876 had been only 6.19 inches and 4.76 inches respectively, or no more than one-fourth to one-sixth of the normal yearly mean; hence here total failure of grain crops, earliest and greatest distress, and the maximum fever mortality. Similar contingencies, in like order, simultaneously ensued in the other districts named. In 1877, the general experience was of the same kind and local distribution; and it was partially repeated during the spring and summer of 1878 and 1879.

Course of Distress, 1876.—As early as August apprehensions of scarcity were expressed, and prices of the staple grains began to rise;

soon afterwards relief works were established by Government in suitable localities, and by the close of November upwards of 150,000 labourers were collected together. Food was at famine rates, and in some places could hardly be procured at all; much of that offered for sale being old, and of bad quality. As pasturage and agricultural operations, with other sources of remunerative labour, ceased to be available, emigration began on a large scale; and about the end of the year, 13 per cent. of the affected populations had left their homes.

1877.—This year witnessed the severest sufferings, and descriptions have been published by eye-witnesses of the prevalent distress, in terms which it would be difficult to surpass. Cattle died of starvation, and the fields went untilled; there was no food but what was imported, and every sort of edible plant was consumed; even water to drink was often scarce. The aspect of the parched land in the eastern areas was compared to that of a desert, with a sky of gloom. Houses or whole villages were abandoned, and cherished personal ornaments were given up for coinage in large quantities by the better class of ryots. Migrations further extended; but many of the infirm, poorer, and low caste would not leave their houses, though confronted with the prospect of death from privation. Whilst facilities for the importation of grain, with means of direct public aid, were liberally afforded by the authorities, and private charity abounded, yet it would seem impossible to concur in the view that at this time only a few persons died of actual want; and how many thousands of all ages sank under the consequences of prolonged starvation, has never been adequately estimated.

As the best available gauge of public distress, I have introduced into the appended Chart, No. 1, the statistics of Government relief works (Column A). In general there was a gradual increase of applicants for aid from November 1876 (beginning with 98,422) until June 1877, when the maximum was reached of 529,951, or about 10 per cent. of the entire population of the five worst districts; after this date, in prospect of the usual rains, the people were encouraged to return to their villages, and the works were closed in the ensuing November.

1878.—Distress augmenting in the spring, relief works were again opened; the rainy season began late, and the fever mortality continued to be excessive, pursuing the course shown in column B, under this date.

1879.—Distress was again prevalent before seed-time in the South Deccan and elsewhere (*e.g.* in Kattiawar); 'deficient harvest owing to excessive and unseasonable rain, high prices, and the injury done to the later crops by locusts, grubs, and rats, all combined to bring about the recurrence of this calamity' ('Admin. Rep. of Bom. Gov.,' 1878-79).

Sickness and Mortality, 1876.—At the close of this year the deaths from fever were beginning to augment: see the Chart, Column B.

1877.—It is recorded that total deaths in the Presidency this year were 627,708, or 259,448 (41·3 per cent.) in excess of those of the previous year—a remarkable augmentation, which was mainly due to 'fever'; the mortality under that head amounting to 336,865, or 116,032 in excess of 1876. Of this surplus 106,818 (upwards of 90 per cent.) pertained exclusively to the nine famine districts under notice, there

being elsewhere no such excess; within the affected area the fever deaths were 223,388, indicating a rate of decease per mille under this head double the mean, *i.e.* 28·4 as contrasted with 14 per mille; and in the two worst districts the rise of fever mortality was even higher, *viz.* from normal means of 13·1 and 11·4 per mille to 40·4 and 50·5 per mille respectively. In the most easterly and poverty-stricken area of Kulladghi, where 9,757 persons died of fever in 1876, no fewer than 41,248 died from the same cause in the succeeding year; and this after a large emigration of families.

I should here remark that amongst the 49,187 deaths occurring in the several Relief-establishments, the casualties from 'fever' not being discriminated, could not be included above; I note, however, that a disproportionate part of these inclusive deaths (*viz.* more than one-fourth) occurred in the same district of Kulladghi alone.

On comparing columns A and B, it would appear that the fever mortality did not rise so promptly as the public distress, lagging somewhat behind, as it were; this might, indeed, be anticipated, yet it should be remembered there was a large emigration of people early in 1877, and a partial return of the same after June, when deaths soon became more numerous. A similar correspondence of fever mortality with movements of the population, is noticeable in the following year; and more emphatically was it established in Bombay city, during the chief famine year.

Striking as are these statistics, they do not show the entire loss of life referable to dearth-fever; for besides the hundreds of migrating villagers dying far from home, account should be taken of the many casualties everywhere due to the sequelæ of fever, yet not included under the same heading. The official tables are necessarily silent regarding the amount of sickness not fatal; applying, however, my data from the Bombay hospitals, it might be said that for every fever-death above the mean, ten persons were ill.

Lastly, I note that, whilst the high death-rate of the famine districts was due mainly to fever, yet 'Cholera' (asserted to be veritable), 'Small-pox,' and 'Injuries' are returned as being unusually fatal; moreover, there was an excess of deaths beyond the mean, amounting to 26,012 from 'Bowel complaints,' and 68,460 from 'Other causes.' Assuming that the three first-named headings had no essential connection with the dearth, it may be supposed that under 'bowel complaints' would come famine-diarrhoea or dysentery, so widely fatal during 1877 in the adjoining impoverished areas of Madras. I find, indeed, that upwards of one-half the total deaths from bowel complaints in the whole Bombay Presidency, took place in the five worst famine districts alone; or a number higher than the total for the Presidency during any year since 1868. Particularly in Kulladghi did the deaths from bowel complaints rise, *viz.* from 1,402 to 6,102—a proportional augmentation exceeding that of even the fever mortality there; and in all the affected districts the main increase from this cause occurred strictly within the famine period, or contemporaneously with the increase from fever. It has been conjectured that the term 'Other causes'—embracing so considerable an excess—did this year include 'privation' or starvation in its acute and chronic forms (under the latter head coming exhaustion, atrophy, anæmia, scurvy, dropsy);

some such adjustment seems needed, but in the absence of information recorded when events were transpiring, discussion becomes futile.

1878.—Concurrent with existing distress was a continued high fever mortality, the year-deaths amounting to 201,418, or upwards of 70 per cent. beyond the mean; it rose considerably soon after the rains set in. The distribution of surplus deaths this year underwent a remarkable change; for whilst the excess in the South Deccan declined, yet in the North districts, where the famine had not been very severely felt, there was now a considerable augmentation. I could not understand how this radiation of fever mortality was produced, but such a transporting of infection as occurred in the direction of Bombay was here, too, possible and would account for the phenomenon; it was demonstrated that spirillum or famine-fever existed in the Deccan during this year, at Sholapore. (See below.)

1879.—Want still prevailing in the areas lately most affected, the fever mortality did not persist (*vide* Chart, Column B); and it is expressly stated, as regards the Sholapore district, that fever hardly seemed 'more frequent among the very emaciated than among those in better condition.'¹ The official records indicate at this date an experience resembling that of the famine period in Madras, in the greater prevalence of such diseases as ulcers, stomatitis, dropsy, dysentery, and diarrhœa; and the mortality rate seems to have been often very high. With this subsidence of fever mortality where much distress and other sickness continued, I again observe that in some areas adjoining there occurred, on the other hand, a striking augmentation of deaths from 'fever'; thus, in the Concan and Gujerat proper the increase was very great, and in the absence of any allusion to coincident increase of malaria, it is open to conjecture whether or not the hunger-pest of 1877-78, whilst subsiding at its centre of origin, had spread to districts around.

TABLE I.

SUMMARY OF COLUMN B.				
	Year	Fever deaths	Totals	Excessive mortality in famine years
Normal years {	1875	119,910 }	236,480	} 188,326
	1876	116,570 }		
Famine years {	1877	223,388 }	424,806	
	1878	201,418 }		
	1879	129,840 }		

MEMORANDUM ON THE CONTEMPORARY STATE OF PROVINCES ADJOINING THE FAMINE DISTRICTS OF BOMBAY.—Within Presidency limits, the effects of the local dearth were extended (as would appear) only by migration of individual sufferers; and of such diffusion the instance of

¹ Quoted from Dr. Mackellar's memorandum in the Report Sanitary Commiss., Bombay, 1879, where it is added that 'circumstances did not permit of observations exact enough to make a statement about any peculiarity in the sequence of attacks.' In future, it may be understood that an adequate use of the clinical thermometer and microscope, for even a brief period, is often sufficient to establish the diagnosis of a new fever; but nothing short of this may suffice.

Bombay city is a remarkable illustration : there may have occurred others like it. In the nearer native states, however, towards both east and west, equal dearth and sickness prevailed, from the same climatic causes, during 1877. Thus, in Mysore, the total reported mortality of 1876 being 54,265, during the first five months only of the following year it amounted to 85,915—an enormous augmentation, due chiefly to so-called 'cholera,' the fever mortality (deaths 12,871) then also rising. Further data were not accessible, nor have I yet learned any particulars respecting the state of the conterminous Nizam's territory. Westward, the Kattiawar peninsula of Gujerat was nearly equally afflicted with drought and fever ; and thence was a nearly continuous stream of immigration to Bombay.

With regard to certain districts of the Madras Presidency, not far separated from the worst affected in the Deccan, and like them suffering severely from deficiency of N.E. monsoon rains in the autumn of 1876 ; it is recorded by the Sanitary Commissioner that whilst the mortality from famine-diarrhoea was very great in the early part of 1877, yet at this time (*i.e.* when the fever mortality was so high in Bombay districts and city) no cases were anywhere seen resembling famine-fever, and that the famine people in Madras did not show symptoms of fever of any kind.

Whence it seems that within an area, wide but continuous and uniformly afflicted, on the one side of a conventional mid-line (*viz.* the western) a fatal form of fever was very rife, whilst upon the other side (*i.e.* the eastern) such disease was absent. Here a positive datum being confronted by a negative one, the position amounts to discrepancy rather than contradiction. It is yet very perplexing, however viewed ; and, assuming the general accuracy of official statistics, I cannot conceive any reconciling term as regards the western area, except the supposition that famine-fever had been first introduced into the Deccan from Bombay, and did not spread beyond Presidency limits. Such an idea is inconsistent with recorded dates and places of fever mortality, yet I would not reject it were there any valid evidence in its favour.

In detail, evidence is furnished by the Reports of the Sanitary Commissioner of Madras that the fever mortality of the most affected districts did not subside during 1876, and it was there doubled in 1877. During 1878 it was still undoubtedly high. From the Report of 1879 it seemed still in excess of the average previous to the famine, the statistics given showing that since 1866 the fever mortality in those districts, and in them alone, had augmented as much as in the Bombay famine area. It is understood that the fever was still of malarious origin : yet as regards one district, that of Chingleput, it is noticed 'that there is great tendency when the periodic attacks have once been established, for them to recur at intervals of a week or fourteen days ; the fever seemed to be contagious.' Such a statement appears to me suggestive of other than miasmatic fever, and were particulars admissible in official Reports, further information would have been of interest.

Appreciation of Events in the Mofussil.—Owing to the absence of definite information in accessible records, one has to rely chiefly upon data collected in Bombay, and partly upon rational inference and analogy.

The term 'fever' in the Mofussil returns is held to mean (a) malarious remittents, and (b) pyrexia symptomatic of local irritation or inflammation. Admittedly its application is very ill defined; yet in the absence here of the 'continued' fevers of Europe, and in the very probable rareness of acute local disease independent of malarious influence, the employment of this word for fatal febrile sickness seems hardly more inexact than the current use of the expression 'remittent fever.'

Normal years (*vide* Chart No. 1, col. B, 1875-76).—The rise of fever mortality in and after the rains is comprehensible on the supposition that malaria then more abounds, and its persistence in the succeeding cold and hot seasons of the year may point to local diseases of the head, chest and abdomen (*e.g.* cerebral congestion, bronchitis, pneumonia, hepatitis, splenitis), variously attributable to climatic changes, age, sex, and hardships, especially in the subjects of malarial cachexia. A previous long residence in the Deccan does not enable me to add more, than that precise data respecting 'country fever' are needed at the present day as much as ever.

Famine years (col. B, 1877-78).—The old forms continuing in use it was impossible that any new febrile disease, even if present, could have been specified; nor to meet the requirements of a grave sanitary crisis, were fresh medical terms introduced. There remains, therefore, to search for indications in the available data, and by suitable arrangement of these the Chart becomes more than suggestive. Thus, it shows that prior to the famine was no unusual fever sickness, and that with dearth the mortality rose so promptly as to indicate clearly a direct relationship: throughout, too, it was evident that the surplus death-rate could not be a simple exaggeration of the previous normal state, but was more likely due to a superadded disease. This new sickness did not seem attributable to intensified malaria, because it began during an unusually dry season, at the end of the rainless autumn of 1876, and long before the scanty rainfall of June 1877;¹ and had there been present

¹ At Kulladghi the rainfall, in 1875, was 25.51 inches and chiefly autumnal; the fever mortality greatest from August to October (mean monthly deaths 1,000 with narrow range) and least in February (562). In 1876 the rainfall was only 4.76 inches, and solely estival, yet the deaths from 'fever' did not diminish as they should have, upon the hypothesis of contingent malarious influence; and further, in spite of emigration and the continued absence of rain, they continued to increase throughout the first six months of 1877, rising from 948 to 3,776 in July, when only 5.3 inches of rain had fallen, or not more than the mean to date. During the corresponding period of the previous normal year, the rise was 705 to 953; and in no earlier record of these dates do I find any augmentation of fever-deaths comparable to that of 1877. In the first eight months of the chief famine-year, with moderate rain, the deaths were 13,450 in excess of the same period of 1875 (normal year); and as regards the dates from September to December, the large increase of 19,517 fever-deaths beyond the mean seems hardly explicable by a distributed rainfall of 18.65 inches, even after making widest allowance for the debilitating effects of want; indeed, the more reasonable supposition would be that malaria had then co-operated with pre-existing causes of disease, rather than itself become the main cause.

In an adjoining coast district unaffected by drought, the deaths from fever rose, with a nearly identical rainfall, to 10,662 in 1877, as compared with 6,452 in 1876; and this augmented mortality was attributed to the effects of malaria upon famine immigrants seeking relief in Kanara. Yet if this were so, how is it that the mortality began with immigration before any rain had fallen? In March (a dry month) there were 836 fever-deaths in 1877, as contrasted with 518 during the same month of the previous normal year.

In these notes I assume the ordinary theory of malaria to be borne in mind; combined heat and moisture being the *sine quâ non* of miasm-production. It is also presumed that the natural history of famine-fever is remembered.

any time during this period even the ordinary degree of miasmatic influence, it is difficult to suppose that the death-rate would have continued to rise with increasing aridity of the soil. It is true that public distress was then augmenting, yet I am not aware that the destitute are specially susceptible to malarious influence, or likely to die therefrom in proportion to their destitution. Still, were this so, the difficulty remains of accounting for so large a mortality when miasm-production was at a minimum. That the increasing mortality in the hot season of 1877 was due to local inflammations, is an idea nowhere advanced; and besides, local complications occur in fatal famine-fever nearly as often as in fatal remittents, pneumonia (*e.g.*) being much the commonest in both infections.

After the rainfall of September severe malarious remittents possibly prevailed in the Bombay famine districts, as they are reported to have supervened in those of Madras; and persons enfeebled by want may have suffered unusually therefrom, with the result of an augmented mortality. At the same time, the public distress did not subside or at once diminish with the advent of rain; on the contrary, it persisted, and the population of the districts becoming augmented by immigrants returning in hopes of a favourable season, which did not arrive, there were then more subjects liable to all forms of disease.

1878.—Though the number of casualties diminished, and their distribution in time of necessity was more uniform, yet the proportionate loss of life may have been hardly less than in the previous year. There is no better indication of malarial exacerbation, and the presence of spirillum fever was demonstrated at a chief famine centre.

1879.—The striking diminution of fever sickness and mortality with the advent of this year (when also they became less frequent in Bombay town and hospitals—*vide* Chart, Cols. B, C, D) points to some particular influence, which I must assume to be that of epidemic decline within primary areas. Much public privation certainly persisted, but the stress of infection subsided; and, as intimated a few pages back, it may have spread centrifugally to adjoining areas free of want. Depending upon the official returns, I see here signs of the course of disease corresponding, in great measure, to known epidemic laws.

Evidence from Analogy.—The malaria hypothesis not satisfactorily accounting for the late excessive sickness, experience of dearths in Europe or elsewhere might furnish the required explanation; and here the analogical inference becomes clear, being to the effect that contemporarily with distress in the Deccan there may have arisen a febrile epidemic disease, not malarious, but comparable to the famine-fever of other countries. An undefined pyrexia attendant on the last stages of privation, has been mentioned in this connection: what it could be was worth testing. Fatal spirillum fever was highly variable in its manifestations at Bombay, and often by no means corresponded with descriptions in medical text-books.

Direct Evidence.—It might seem impossible that non-resident enquirers could learn the true character of a comparatively fugacious complaint like relapsing fever, especially when separated from the nearer seats of the disease by a distance of 200 miles or more. Yet the obstacles

to proof are not insuperable, the following contingencies being possible :
a. A Deccan resident while affected with 'fever' might be brought in a few hours by rail to Bombay, and be at once admitted into hospital and be examined. *b.* The verbal testimony of patients showing specific fever might be accepted as to the date and place at which their sickness arose. *c.* Specimens of blood taken from 'fever' subjects resident in the Deccan, might be preserved and scrutinised at the Presidency town. As only by accident were new comers brought to hospitals or early came of their own accord, instances like those under *a* and *b* would necessarily be very rare ; and I consider it fortunate that there are any of the kind to be met with. The following cases, therefore, bear a significance which is not to be measured by their numbers. I regret that blood-specimens were not earlier procured from the Mofussil, but was not aware how to prepare them until the height of the epidemic had passed.

Proof under a. CASE I.—J. F. H., æt. 35, Mussulmanin, female domestic, was brought to hospital by the police on May 30, 1877, from the adjoining railway-station of Byculla, where she was found to be in a state of delirium : 'a friend with her states she has been suffering from fever for eleven days while at Poona (Deccan) ; she arrived at Bombay this morning to go to her brother's house, but became delirious, and the friend not knowing where to take her to, reported her condition to the police' (quotation from original notes of case). On admission the axillary temperature was 102°, pulse 120, feeble, respirations 25, shallow ; tongue dry, furred, brown ; thirst excessive, cough and sibilant râles in chest ; next day, the blood was thus described by Dr. A.—coagulation slow, plasma clear, plasmic bodies a few, white corpuscles many, spirilla seen. With prompt subsidence of the fever immediately following, jaundice and hepatic uneasiness were marked ; she remained free from fever for nine days, when a pronounced relapse took place, lasting four days, on three of which the blood-spirillum was again found. Jaundice and delirium also returned ; there was a prompt fall of temperature preceded by the *perturbatio critica*, and much exhaustion subsequently. She remained in hospital eleven days longer, and then left in a convalescent state.

I saw this woman frequently, and at the time noted there was no reason to doubt the truth of her history. How she acquired relapsing fever at Poona is unknown ; it may have been by contagion. The case further shows that residents in the chief Mofussil town were in the way of catching the disease so early as May 1877. This is similar to experience in Bombay.

Evidence under b. CASE II.—B. K., æt. 20, country lad, Hindoo, immigrant from the Deccan, destitute but not starving, was admitted on April 30, 1877. History—had tramped from Poona in search of work and food ; was seized with fever above the Ghâts (eighty or ninety miles by road from Bombay), and resting two days, descended to Thanna (twenty miles off), again rested one day, and then walked to Bombay, when he was so ill that the day after his arrival he came to hospital, and was admitted into my clinical ward. At this date seven days had elapsed, according to his plain and repeated statement, since the fever began, five of which had been spent on the road ; when seen on the evening of day of admission, he presented several usual symptoms of the new fever—temp. 104°, pulse 108, small and soft, respirations 28 ; bronzed *facies*, coated tongue, frontal headache, nausea, hepatic fulness and tenderness, jaundice, some cough, pains in the bones of the lower limbs, but spleen not visibly enlarged ; the blood was found by me to contain many active spirilla. Next day a marked crisis occurred, the temp. falling 8° F., then a rebound to the normal temp., and, excepting dysenteric diarrhœa with much debility, no interruption to convalescence ; discharged May 31.

A common typical case of spirillum fever seen late, with no indication of relapse except some periodic splenic fulness ; the absence of a

febrile recurrence not, however, impairing the patient's testimony in any degree. Date of immigration about the height of the Bombay epidemic, when crowds of immigrants were arriving.

I took occasion to point out to my class of students, the great probability that this lad's fever had been contracted on the road or possibly in his village home, his long journey on foot not, according to my observation in Bombay, militating against this view; and the whole case, like that of the woman above, affords an illustration not only of spirillum fever prevalent in the interior, but also of its direct conveyance to the Presidency, where it might spread.

CASE III.—F. R., æt. 23, Christian half-caste, carpenter, resident at Madras; set out thence on foot for Bombay; he was at Poona about a week ago (close of July 1877) and had fever; leaving he had chills and renewed fever on the road; arrived in Bombay yesterday evening (31st), coming straight to hospital; then temp. $104^{\circ}\cdot4$, pulse 128. August 1—Pyrexia, dry skin, headache, pains, bilious vomiting, jaundice, constipation; blood examined on admission and also this morning; the spirillum found, though sparingly (Mr. S. A.), and again found in the evening, when the temp. rose to $104^{\circ}\cdot8$, next day declining to 97° , by critical fall; much weakness; discharged after three days at his own request.

It could not be said whether or not this specific attack was the first, for the man asserted that he had been suffering from 'ague' for a month previously; and if his statement were true, it in no way invalidates the significance of the case as one of specific fever acquired in the Mofussil, but would also serve to show that particular means are needed to distinguish this peculiar form of pyrexia, which otherwise might not be recognised. Neither of the temperature-charts of these two cases, nor of No. 4 below, is in the least degree characteristic of ordinary relapsing fever; but, on the contrary, to an observer without practical experience of spirillum fever, the charts would seem absolutely insignificant, as they show only the last 1-3 days of fever, followed by an apyrexial state.

CASE IV.—M. B., æt. 35, Hindoo, ill-fed and anæmic, admitted with fever into the J. J. Hospital, July 2, 1877; he gives the following history. Left his native place in the Kurar Petta (famine district of Satara) twenty days ago in consequence of want of food, travelled on foot to Bombay and arrived here four days ago, putting up at Oomerkari, had fever on arrival in the town and now comes to hospital; on admission was very ill, temp. $105^{\circ}\cdot6$, pulse 120; the pyrexia remitted on two days and on the third subsided by crisis, the temp. sinking to $96^{\circ}\cdot4$, pulse 95; there was some secondary fever with diarrhoea, and later on two isolated paroxysms which might represent the relapse. The blood was examined on the day of his entering hospital and on the next day also, the characteristic parasite being found on both occasions; it was not seen after the critical fall of fever.

Here, again, supposing that reliance can be placed upon the simple statement of the patient that he had been in Bombay only four days, the clinical evidence would emphatically point to the conclusion that the specific fever exhibited by this man had been contracted before his arrival in the town. I have no hesitation in making this assertion, for whatever doubts be suggested as to the previous duration of his fever, the fact remains that in accordance with large clinical and experimental experience, the incubation period of the disease would necessarily carry back the date of infection prior to the man's arrival. Were his attack a 'relapse,' the inference of extra-mural origin of the disease would be still more strengthened.

There are before me the notes of several other instances of demonstrated spirillum fever dating back to day of arrival in Bombay, or to one or two days afterwards; and in my own opinion these, too, were valid evidence of the point in question, for no data are known to me tending to show that, amongst men, the contagion of this fever is capable of operating instantaneously in the production of pyrexia. On the contrary, in the remarkable series of six instances of infection by inoculation at autopsy of fever patients, which is detailed in the chapter on 'Contagion,' the periods of incubation were as follows:—3 days 14 hours, 3 days or possibly 4 days, 7 days, 7 days, 7 days 4 hours, 7 days 4½ hours; mean of the series, 6 days. As under ordinary conditions contagion is not likely to operate more quickly than by way of inoculation, it may be assumed these dates are not excessively long; and therefore any attack of fever supervening a day or two after an immigrant's arrival in Bombay, had almost certainly been contracted outside the town.

It may also be stated here that relapsing fever is communicable at all times when the blood contains the characteristic *bacterium*—namely, during fever of invasion-attack (mean duration in man, 7–8 days), of first relapse (mean duration, 4–5 days), and even of second relapse (mean duration, 2–3 days), or for a total of about fifteen days in the course of a prolonged illness. But these febrile periods are separated by apyretic intervals of about seven and nine days respectively, and therefore the whole time over which possibility of active manifestation of disease extends (and with this the faculty of propagation) becomes enlarged to thirty days or so; which is a term long enough for any journey a famine immigrant would make in traversing the country from his native town or village to the Presidency, inclusive of due intervals of rest *en route*. No allusion is here made to the fact that the fever is communicable during an additional period, corresponding to the few days immediately preceding visible pyrexia.

Lastly, I remark that a band of famine immigrants once infected might not only convey their fever weeks together amongst themselves, thus allowing of even long sea voyages becoming the means of transporting famine-fever; but by its propagation from their members to strangers coming into contact with the band, a still more prolonged manifestation of the disease becomes not only possible, but quite probable. In fact, thus do epidemics arise; and hence the great significance of the few cases I have now narrated, for it is evident that, famine-fever once appearing in the Mofussil, widespread sickness was at least likely.¹

¹ Amongst the many other confirmative data in hand, the following may be useful in this place:—*a.* It was matter of common observation to myself and other medical men, that some immigrants entering the town were visibly ill and had the aspect of low fever. *b.* From a trustworthy official source (non-medical) intimately acquainted with the town of Bombay, I have the written statement that there were seen, in 1877, a considerable number of immigrants who were suffering from fever and prostration on their arrival in the town; the men and young children suffered most (a noteworthy remark, since here, as in Europe, mothers of families seemed to get famine-fever first, and then to communicate it to their associates: H. V. C.) Again, it is reported that at a station near Bombay where immigrants were stopped in August 1877, 'some of the children and a few of the men had fever,' this statement coming from a medical authority disallowing the existence of famine-fever at the time. Again, in those quarters of the town where the fever was worst, 'cases like this occurred, a party of seven or eight immigrants arrived with, say, three of them having fever; in a day or two, there would probably be five or six of the same party attacked with the disease; there is no doubt at all of such cases having occurred.'

Proof under c.—Sholapore town and district suffered severely from famine and fever in 1877, and again in 1878. In May of this last year Dr. A. (who had taken part in the enquiries at Bombay) visited, at my request, the above-named town and neighbourhood; he promptly reported in detail eleven cases of fever, unselected, finding the blood-spirillum in eight. In consequence of this information I went to Sholapore after a few days, but was too late to confirm the observations; and not being able to remain away from the Presidency, had to return without fresh proof. However, upon examining the specimens of dried and stained blood which Dr. A. had himself prepared, I found the parasite still visible unequivocally in one, and probably in two other slides, which, like the majority, were too imperfectly prepared (from inevitable difficulties met with) to allow of thorough scrutiny. Of the cases, ten were Hindoos; five were males, eight were town residents (one in respectable position), five being wandering mendicants; three were seen in villages near; several of the poorest gave a history of famine suffering; all had symptoms more or less characteristic of relapsing fever, some describing the relapse, and this was so with the destitute woman J. S., whose blood furnished the decisive evidence; she was admitted a second time with fever (entered as being 'ague') into the Municipal Dispensary, and died there of dysentery ('bowel complaint'). Contagion in the family was indicated once; none of the patients had travelled to Bombay, and all denied having been in contact with travellers from a distance. I found the country parched up, and villages desolated, but saw few cases of fever; this is no matter of surprise, however, considering that the specific pyrexia is never continuous, and may be very brief. In practice, evidence from the blood is equally contingent, and a thorough investigation of relapsing fever may require a longer time and more watching than cases of ordinary malarious fever; not to insist upon special knowledge, skill, and patience with the microscope. How readily the truth regarding the nature of the late fever sickness in the Mofussil might have been elicited on the spot, was shown by the result of the brief visit above described.

SUMMARY OF THIS CHAPTER.

The late excessive fever sickness undoubtedly followed upon dearth; the circumstances of its origin and early course indicate its non-malarious character; upon analogical grounds it might have been a true famine-fever; and the presence in the Mofussil of relapsing or famine-fever (*i.e.* of pyrexia with spirillar blood contamination) was demonstrated in some cases. This combined testimony points to but one inference,

c. What kind of fever the above might be, is not obscurely indicated by the following instance belonging to the series of documents bearing on 'Contagion' which are quoted hereafter. It is that headed 'The Bala Family,' and contains a statement that the mother coming with the family from Satara, arrived at Poona, and fifteen days after her arrival there she became laid up with fever and remained ill for about a fortnight; when her eldest son, hearing of her illness, went from Bombay, and removed her from Poona, whilst in a delirious state, by the G. I. P. Railway to Byculla station. A few days after her arrival in Bombay, her husband became ill, and then all the five children, in succession, with spirillum fever; some of them died in hospital. In offering these remarks, I do not wish it to be supposed that all the febrile sickness seen amongst immigrants was of this specific kind; for probably no epidemic has had a wholly exclusive character.

which is not contradicted by other facts, and which legitimately explains all the chief phenomena of the epidemic.

The main valid objection to this conclusion, is the circumstance that famine-fever was not detected in the Deccan by resident observers ; and a suitable reply to this would be the fact that at the height of public sickness in Bombay, and under the most favourable conditions for enquiry, the prevalence of the new disease was both ignored and authoritatively denied.

CHAPTER II.

HISTORY OF THE FEVER EPIDEMIC IN BOMBAY.

Normal Data of the City.—Situation, N. latitude, $18^{\circ}57'$; E. longitude, $72^{\circ}51'$; site upon one of several low, level islands at the mouth of an estuary; population, near 700,000; subsoil, igneous rock (trap or basalt), clay shale, calcareous sand, and marine alluvium.

The compact native town (here alone concerned) is mostly built upon a central, flat, alluvial area, often little higher than spring-tide level; a part constructed upon the eastern raised and rocky foreshore (elevation 100–200 feet) did not escape the disease; whilst the similar western coast line, also of trappean hills, where Europeans and the wealthier natives reside, was practically unaffected. The streets are mostly narrow and tortuous, the houses high and constantly overcrowded; mean density of population 6 to 12 square yards per individual in central parts. Drainage of rain-water and liquid sewage as yet defective; street cleaning is attended to, house scavenging is chiefly manual. The food and water supply are good; drinking wells are now little resorted to.

The population is mostly Hindoo, about one-fourth being Mahomedan, one-twelfth Parsee, and a smaller proportion of other non-indigenous races; the several sections do not eat together or intermarry, and, on the other hand, particular subsections are closely aggregated. The humbler classes (alone in question here) consist of day labourers, mill hands, handiercraftsmen, servants, petty traders, and mendicants who freely wander. There is no system of poor relief, and the destitute paupers suffer much. In general wages are sufficient, and it could not be said that want is commoner here than in the cities of Europe. Of the sexes, male adults predominate; especially during the open season (October to May), when labourers from the interior annually resort for labour to Bombay, returning to their homes and fields before rain arrives.

The climate is tropical; mean annual temperature, 80° F., with daily range, 8° ; absolute range, 23° ; mean annual dew-point, 72° ; humidity, .75 (saturation being 1), with a range of only .19; mean barometric pressure, 29.815; daily range, .103; the sea-breeze blows 18 hours of the day; the rainfall averages over 70 inches per annum, and attends the S. W. monsoon from June to October.

Normal Mortality of the Town.—An elaborate mortuary registration has been in use for some years; of late nearly 20,000 deaths occur

annually from all causes, amounting to a rate of nearly 30 per mille. A chief and uniform heading is 'fever' (remittent), under which are reckoned more than one-fourth of all casualties at all ages ; it is a characteristic of the Bombay returns. Cholera, small-pox, measles, are items fluctuating, but never absent ; phthisis pulmonalis, 3,000 deaths ; other lung inflammations, 2,000 ; from dysentery and diarrhoea, 2,000 ; infantile convulsions add to the large mortality of early years, so significant in this town. Most recognised diseases of Europe are found in the mortuary lists, but some are unknown (as scarlet fever), some not very long familiar (enteric fever and diphtheria), and some await recognition (typhus, simple and recurrent).

Abnormal Events.—In the absence of local drought or dearth, or of other unfavourable change in the outward state of the town, there yet occurred in 1877 and subsequent years, a sanitary calamity which will not soon be forgotten. This consisted of an enormous influx of famine-stricken peasantry, and the concurrent increase of fever mortality shown in Chart No. 1, Column C.

Though the Presidency town is distant 300 or 400 miles from the *foci* of want in the Deccan, yet free communication was maintained by rail, road, and indirectly by sea ; there being, too, ready transit by rail from Central and North-West India, where also famine prevailed. As a centre of textile manufacture and remunerative labour, it early became a refuge of the remote needy populations, and the extent of immigration was enormous. Thus, in June 1877 (when the fever-deaths were most numerous) it was estimated that the population had increased to 1,000,000, or by 30 to 40 per cent. beyond the normal ; and to a mitigated degree the influx still continued, there being counted in the main roads from the chief area of distress during the last fifteen days of August, 18,884 paupers, in various stages of destitution. I am unable to offer any estimate of subsequent years, or of the numbers coming from Central India, and further northward or westward from Kattiawar ; but the total was doubtless many thousands of persons, who had no intention of immediately returning to their desolate homes. Contrary to custom, whole families migrated to the town, with a corresponding effect upon the statistics of sickness. The new comers needing food and seeking work, wages and the prices of grain were affected, and doubtless the quality of food sold was sometimes bad. Untrained labour was available chiefly in the new docks and on coal ships, and the influence of arduous exertion upon the exhausted frame of men and women may be readily surmised ; skilled labour was limited to the spinning and weaving mills, and to the many private looms for cotton and silk fabrics, in even more crowded buildings. The depressed mental aspect of these strangers was often too obvious to be overlooked.

To my mind, however, there remains the most important consideration of all, affecting both severity and propagation of the prevalent disease ; for house accommodation, never superfluous, had not materially increased in 1877, and the lodgment of so many immigrants necessarily led to excessive overcrowding, with its inevitable consequences. In most houses the subdivision of rooms was extreme, passages and light being reduced to a minimum ; cleanliness was

impracticable, and the removal of excreta interfered with ; workshops, and the lanes, temples, verandahs, and markets became sleeping places, and a distinct proportion of the sick were quite homeless.

I have not insisted upon the elevation and character of the soil in the chief fever localities of Bombay, from not detecting any uniform relationships of these conditions with prevalent sickness. Nor from the large additions to the population in 1877-78, could the true death-rate of the town be elicited.

Course of Sickness, 1876.—Concurrent with imminent distress in the Deccan, an unusually large migration to Bombay began in the autumn; yet the fever mortality did not immediately rise (*vide* the Chart). From hospital data I know that even early this year destitute individuals had arrived in the town from Northern India (where also scarcity prevailed), and that famine-fever had appeared.

1877.—This was the year of greatest suffering both in Mofussil and at Presidency. In Bombay, it could not be said that mere insanitation was so much worse as to account for the augmented death-rate ; nor was the local meteorology at fault; *e.g.* annual means—barometric pressure, 29·846 ; temperature, 81°·2 ; dew-point, 72°·7 ; humidity, 75, and rainfall 69·89 inches at Colabah. The mean temperature of May, with so much fever sickness, was 85°·5 ; dew-point, 75°·8 ; no rain fell from March until June, and not an inch during the previous five months—these data being adverse to the hypothesis of malaria as the cause of fever. More important, therefore, seems the conjunction, at this period, of maximum immigration with maximum sickness and mortality. The fever-deaths starting in January with a slight excess over 1876 (though not over earlier years), viz. as 686 to 489, in March had risen to more than double (1,265 to 527), and in April, May, and June they were treble as many, the greatest number being reached in May, viz. 1,617, as contrasted with 511 in the previous year.

With the rise of mortality in March, and the prospect of further augmentation through the crowds of famine-stricken then daily flocking to the Presidency, public attention became imperative ; and a Relief Camp capable of lodging 2,000 persons was erected on the outskirts of the town, whither were conveyed by the police such destitute or sick persons as they could best induce to move. It was here that the nature of the prevalent fever was surmised and demonstrated ;¹ and as this assemblage may be supposed to represent the population of the worst lanes and houses of the town, I may add that a few days after its formation, of 659 inmates 309 were found to be ill or ailing, others were much reduced, and some unaffected were in attendance on families or friends ; about one-half the whole were recent immigrants, the rest being residents of over six months' standing. The authorities soon after took measures to check further influx of the destitute, and the monsoon season being at hand, many immigrants returned to their villages ; by July, therefore, the mortality had declined to 976, but rain again failing, there was a temporary arrest of the exodus and fresh influx, the fever deaths rising

¹ To Mr. Thomas Blaney, an esteemed medical practitioner in benevolent attendance on the sick, and a respected town councillor, belongs the credit of independently recognising this disease and engaging actively in its mitigation.

to 1,006 in August ; thenceforward, however, prospects brightening somewhat in the Deccan, the stress in Bombay and the mortality declined to December.

Other details are the following :—The proportion of residents and strangers who died during 1877, is clearly indicative of the extraneous mass of mortality intruded through immigration ; thus, whilst in 1876 strangers contributed less than one-half the total deaths, in 1877 their casualties were as 20,000 to 12,000 of residents ; and it is said upwards of 6,000 strangers died in Bombay during the dates April to June, when 'fever' was at its height. Of the total number of deaths in 1877 as compared with 1876, no less than eleven-twelfths were contributed by new-comers arriving within the famine period.

It was clearly shown that the excess of fever-deaths occurred almost solely, in those quarters of the native town where the famine-immigrants lodged ; and this quite irrespectively of local malarious proclivities, as displayed in previous annual returns.

It was also proved that the high fever mortality prevailed chiefly, amongst the classes of people forming the mass of immigrants, and the worst lodged ; these were Hindoos in 1877. Thus, amongst Hindoos (of the Maratta race mostly) the normal fever death-rate being ten per mille and tolerably uniform, in January 1877 the estimate rose to 15·50, and in succeeding months to 18·41, 26·89, and 40·34 per mille ; amongst Hindoo outcastes the normal rate of 6·63 per mille similarly increased to 18·24, 28·30, 33·17, and in May to 44·78 per mille. On the other hand, amongst the Mussulmans the contemporary rise was from normal 10·20 per mille to only 21·91 ; amongst Parsis from 3·53 per mille to 11·79 ; and amongst Europeans the monthly death-rate from fever continued to be regulated by one, two, or no deaths. Such race differences are unprecedented in normal years, and their connection with corresponding proportions of newcomers seems undoubted ; for not only were they most marked when immigration had reached its height, but in detail they are equivalent to the components of superadded population ; the great majority of strangers being Hindoos and outcastes from the Deccan, yet not a few Mussulmans chiefly from Northern India, and the rest from Persia and adjoining countries.

A further notable feature of the year was the increased mortality amongst women and children, in accordance with the larger number of families who this year accompanied the male bread-winner in his search for work, being driven by want from their home. Infants from seven to twelve months died from 'fever' at the estimated rate of 300 per mille, adults from twenty to thirty years at the rate of 11·81 males and 16·57 females ; these proportions also being quite exceptional. An important datum which cannot be elicited from the mortuary Returns, concerns the amount of illness short of death, which was due to fever ; or, in other words, the mortality rate of that disease. As indicated by brief experience at the Relief-camp, this rate was about 9 per cent. ; which, assuming that 5,000 of the 7,000 excessive fever-deaths in Bombay were due to famine-fever, would give a total of more than 500,000 cases of illness in 1877 from this one cause. The total death-rate of the town, as estimated on the census of 1872, amounted to 52·0 per mille, as contrasted with 32·25 per mille in the previous year of 1876 ; the actual number

of casualties registered was 33,511, and of these 12,832 were due to 'fever,' no other heading being nearly so predominant.

In connection with famine-diarrhoea, which was shown to be so prevalent in the Presidency town of Madras (holding not unlike relations to its own country districts), I would add that the municipal report indicates a similar affection may have prevailed here, especially amongst destitute immigrants; the absolute number of deaths from cholera (so interpreted) and diarrhoea in excess of the normal mean was, however, inconsiderable as compared with the augmented fever-deaths.

1878.—Though famine immigration from the Deccan greatly diminished, yet the fever mortality did not subside in proportion; and from hospital data, I am able to state that now the resident poor of the town suffered more than in the previous year. The total deaths exceeded those of 1876, the latest normal year, by 6,116; and of this surplus 4,007, or two-thirds, were due to 'fever;' *vide* the Chart. As in 1877, but rarely in normal years, the fever mortality augmented in May and declined in June, when it is the custom of country labourers to return to their homes; in general features too, of caste, sex and locality, the experience of the great famine year was partly repeated. The fever sickness here seemed to be kept up by localised epidemics, the chief of which, according to my hospital data, occurred among weavers and other Mussulmans; and on testing these data of 'relapsing fever' with those of fatal 'remittents' shown in the Municipal Returns, I find the clinical experience may have been (as was not unlikely) a sample of larger facts; thus, during 1878 fever-deaths in the town amongst Mussulmans, so far from declining distinctly, as did those amongst Hindoos, are found to be rather in excess of the previous years, viz. as 2,477 to 2,422; and 375 weavers in the town died, against 186 in the previous year. A second sub-epidemic outbreak was detected amongst low-caste immigrants from Kattiawar, whose numbers brought to hospital aided in the May predominance of spirillum fever shown in the Chart, Column E.

1879.—Fever mortality but slowly declined, the deaths being 8,445, or more than 50 per cent. in excess of the previous normal mean; its monthly distribution is shown in the Chart. Experience at the G. T. Hospital showed best the frequency of relapsing fever when carefully sought for, there being four or five times as many admissions there as in the larger Native hospital (alone referred to in the Chart); and I had the clearest evidence of its spread by contagion. Deaths amongst Mussulmans still predominated in the town; and equally in my wards, cases of relapsing fever amongst them. There occurred this year another minor famine immigration of ryots from Kattiawar, and several cases of specific fever from this group were admitted into the G. T. Hospital; two died (father and daughter), and I note that here 'remittent' fever deaths were registered outside. By caste these people were Jains, and the municipal returns show an augmentation of fever mortality in this sect; the significance of the datum, like that of contemporary high Brahmin mortality, being not obscure to medical practitioners acquainted with the local history and contagious properties of the new disease.

1880 and 1881.—Traces of the late epidemic still remain, notwithstanding subsidence of the main cause giving origin to it in 1876-77;

and in this respect, as in so many others, experience at Bombay has resembled that of some large cities of Europe.

MEMORANDUM ON THE CONTEMPORARY STATE OF PROVINCES IN DIRECT COMMUNICATION WITH BOMBAY.—In nearest and fullest association were the famine districts of the Deccan, whose state I have already described and whose connection with the local epidemic was immediate and indisputable. Also within Presidency limits is the Kattiawar peninsula of Gujerat, where prevailed famine and fever sickness, almost as early and as marked as in the Deccan itself ; and whence, too, was much immigration at similar and later dates to the Presidency. Due prominence being accorded to these instances, I would next allude to more distant countries.

As a wealthy commercial town uninvolved in the prevalent distress, Bombay became more than ever the centre towards which the needy turned when compelled to leave their homes ; and with railways commonly accessible, distant parts freely furnished their quota to the sick in the town. As regards Madras, where famine in 1877 was so severe, there is not much evidence of migration hither, whilst from Central India, Oude, Rajputana, and the North-West Provinces there is much concurrent testimony of large migrations to Bombay, in consequence of scarcity, from 1876 onwards ; and it is notorious that with and after the dearth in those parts, there ensued a fever mortality of appalling extent. Regarding the nature of that fever I have sought in vain for adequate information ; it seems Europeans were not spared. From many towns in the above-named provinces (a long list is before me, and Azimgurh perhaps oftenest entered) immigrant Mussulmans (weavers chiefly) and Hindoos (ryots and low-caste) early began to flock southward, the journey by rail from Allahabad not occupying more than four or five days ; some walked long distances. On arrival they lodged in parts of the native town less crowded by the Marattas, but as soon (1876) and afterwards as often as these (1878) became inmates of the fever wards of the hospitals in Bombay. I am unable to say (the people being unusually timid and prevaricating) if these immigrants brought fever with them into the town ; but many displayed the specific infection in typical form, and that very soon after their arrival. Their resident fellow-caste men were contemporaneously affected ; and, in sum, the evidence regarding the distant famine area of Northern India is similar to that of the Deccan itself. See p. 27.

That in Persia and China dearth and disease were almost contemporary with the events in Western India is well known, and fever persisted after the famine. Its true character is unknown to me, for the few cases admitted into the G. T. Hospital of pilgrims and others coming in native craft from the Persian Gulf, Jeddah, and Aden, were not at a stage or in a condition to furnish decisive evidence. This might be looked for, even had the fever been specific. According to my late experience, future accounts of fever epidemics in the East may be radically defective, if they do not include adequate information as to the state of the blood during pyrexia. This remark is capable of general application.

Interpretation of Events at Bombay.—In the Municipal mortuary-returns the term 'fever' is held to signify mainly malarious 'remittents,' with or without local complications; a very small proportion, viz. not more than one-twelfth part, of the mortality under this heading being attributed to ague, simple continued fever, and enteric.

Normal years (Chart 1, Col. C, 1875-76).—During the decennium prior to 1877 the fever-deaths were distributed throughout the year almost uniformly, there being rather the fewest in the third quarter, *i.e.* 23.7 per cent., and rather the most in the first quarter, *i.e.* 26 per cent. Such a disposal does not favour the view of their being due solely to malarious influence; nor does the fact of there being rather more 'fever' deaths in April and May (hot season) than in October and November, or immediately after the heavy rains, when miasm proper might be expected to abound. While it is allowed that in this island conditions of malaria-production should be present for a considerable portion of the year, yet they cannot be supposed to obtain in the crowded native town, where the foul state of the surface soil, including the foreshores, and the habitual overcrowding preferably claim attention. The actual additions to population here from October to May, due to immigration of healthy male labourers, have not been precisely estimated; but it is probable that they aid in the maximum fever mortality of December and January (cold months), which amounts to 17 per cent. beyond the monthly mean, and apparently is not due wholly to local complications. There is, however, a distinct tendency in ordinary years to increase of the fever-deaths during the hot month of May; and both the epidemics of 1864-66 and 1877-78 reached their acme in this month. With the rains in June, the fever mortality has always promptly declined. From these and other hospital data, I infer that the 'remittents' of Bombay are not purely malarious; and they may include modified forms of 'continued' fevers (*i.e.* typhus and enteric), due to causes similar to those recognised in European cities.

Abnormal years (Col. C, 1877-8-9).—No change of name being made in the fever registration of 1877, the immense augmentation of deaths at that crisis is recorded under the head of 'remittent' fever, yet a reference to my Chart should, I think, at once dispel the idea of such augmentation being due to malaria; and if in ordinary years this eminently seasonal influence does not seem of primary importance at Bombay, during the late augmented sickness it must have become greatly modified to permit of its application as the immediate and adequate cause of mortality. No such modification, however, was shown to occur, nor could the adoption of the name 'remittent fever with spirillum' stand in place of demonstration; such a nomenclature serving only to increase a confusion already embarrassing enough. It was not supposed that malarious influence abounded more than usual during 1877-78; and had this been the case, there would remain the difficulty of accounting for the epidemic spread of the new fever. In sum, I find no valid evidence in favour of the view that the late excessive fever mortality was due solely to malarious influence.

No addition to precise knowledge accrues from attributing the sickness simply to intensified mal-hygienic conditions, for filth, overcrowding, and hardship of life operate only by favouring the extension of specific disease.

Lastly, as regards other general agencies ; on consideration, I perceive no essential connection of the late disease with local climatic changes of temperature, moisture and wind, or with geodic relations of soil, elevation and ground-water.

Analogical Inference.—On comparing the late experience in Bombay with that of European cities under similar conditions, it became in the highest degree probable that a form of contagious typhus would arise, the presence of which would serve adequately to explain all the phenomena ; and that a virulent type of relapsing fever did, in fact, make its appearance the following data will show.

Direct Evidence.—This came from two sources,—chiefly from the hospitals, where sick emigrants and residents were examined, with positive results afterwards stated ; but earliest from the Relief Camp mentioned above.

Here, as member of a visiting committee, I attended for three weeks, on April 8, 1877, seeing the following instance :—

CASE V.—G. D., adult male, robust, porter at a railway-station where many famine-immigrants from the Deccan left the trains daily arriving ; admitted with his family April 3, being ill with fever, and two others had been attacked before. His axillary temperature was $105^{\circ}5$, rising to $106^{\circ}2$ next morning ; on the seventh evening it was 104° , and then I found the blood to be full of active spirilla ; there was a marked crisis the same night, the temperature next day sinking to $95^{\circ}4$, and the blood-parasite being no longer visible. The man remained free from fever for seven days, and then (fourteenth day of illness) underwent a relapse (spirilla again found), which lasted six days, ending abruptly with a fall in temperature of 9° ; he became extremely ill during the febrile stages, having typhus symptoms with delirium ; and was much reduced afterwards. Two of his young children acquired the same fever while in camp.

Amongst other instances in which the blood was scrutinised, was that of a pregnant woman who aborted at the crisis of her first attack ; here the blood-parasite had been detected previously, and as it was found in three other fever cases, the demonstration appeared sufficient ; seeing that the general symptoms in all were so usually alike, and that when the state of the blood gave negative testimony, an adequate explanation was always forthcoming.

The committee had notes taken, with charts, of 48 unselected sick persons : males 31, females 17 ; more than half under 20 years of age, youngest $1\frac{1}{2}$ year, the oldest 70 years ; 10 were residents, 35 immigrants, and 3 of unknown home. The general symptoms, personal history and social condition were generally characteristic of relapsing fever, and sometimes typical ; the chief forms of this disease also were here illustrated, and I now perceive that the enquiry really concerned a kind of epitome of the town fever. At first 25 per cent. of the cases were recognised as being certainly, or probably, examples of spirillum fever : afterwards, on reperusing the original notes with fuller practical knowledge of the disease, I concluded that nearer 75 per cent. may have been such. Contagion was strongly indicated from the first ; thus, the number of sick increased in camp : two out of five hospital assistants were attacked with severe fever, and one died with typhus symptoms ; a cook and another person were seized with similar fever, and 14 of the patients examined were well before coming hither, being affected at periods of six days and upwards after their arrival ; some of the new attacks occurred

in the temporary hospital itself—members of a family living together were thus implicated, and in two such instances noted the children showed the blood-spirillum. The mortality in camp was less than obtained in the J. J. Hospital at this time, because the cases were often milder, and such as the friends would have kept at home; but two houseless and friendless relicts of the camp, on its dispersal before the rains, died in the above hospital with demonstrated spirillar infection. According to this summary, it may be affirmed that much, at least, of the excessive fever sickness in Bombay, at the height of the epidemic, was due to relapsing fever; and it would be difficult to conceive of better testimony than the above, of a practical kind.

Hospital Statistics.—The value of these data rests on the admitted fact that sickness in the town and under treatment, was alike as regards subjects affected, locality, degree, date, and character. The total medical and surgical accommodation in the two Bombay hospitals amounting to one bed per 1,000 inhabitants, it could not be expected that the medical wards would afford more than a dim reflex of the sickness outside; yet that the image was a true one I entertain no doubt whatever, from both local knowledge and direct comparison of disease in members of one family, part of whom remained at home and part came under treatment. See also below.

J. J. Hospital (Chart 1, Col. D).¹—In normal years the seasonal increase of remittent fever is clearly indicated; thus, in the five years previous to 1876, the mean monthly admissions were the highest just after the rains, viz. 30·8 in November, and least at end of hot season or beginning of monsoon, viz. in May 9. No notable departure from these means occurred until 1876, or the year immediately preceding the epidemic, when from March onwards there was a decided and continuous increase of admissions, as follows:—February, mean of the five previous years, 17·6, actual in 1876 the same; but afterwards an excess by months in order of 7, 6, 7, 10, 12, 11, 10, 12, 8, 9. The deaths from remittent fever were 30 per cent. of admissions in 1876, against the mean of 31·3 per cent., a difference which shows that febrile affections, if commoner, were not more severe than previously. These numbers could not be displayed in the Chart, and they are here mentioned because 'relapsing' or 'famine' fever, if not first beginning about March 1876, yet was first noticed at this date (see below); and the rise in hospital admissions may have corresponded to the initiation of the new fever. The town mortality, obviously, might not furnish so early indication of an incipient disease, less fatal than usual.

Abnormal Years.—The very first monthly admissions of 1877 were more than two-and-a-half times the mean, which is a more abrupt augmentation than elsewhere shown in the Chart. In March there

¹ These statistics are given on my sole authority, official returns not including relapsing fever; though numbers are small, yet they have a proportionate as well as direct value; limitation here being due to frequent overcrowding of the medical wards, which necessitated the rejection of fresh fever patients. During 1877 no fewer than 2,947 sick were refused admission, mostly suffering from fever.

Data from the smaller G. T. Hospital could not be shown in the Chart; so far as I am responsible, they correspond with the above: fifty cases of demonstrated spirillum fever being admitted in parts of April and May, and during parts of November and December twenty more; observation was then stopped on account of my contracting the fever, and temporarily leaving India.

occurred a further rise, which may be traced also in Cols. C and B ; and in May a greater advance to near the maximum (see the Col. E of this date), which corresponds to the actual maximum of fever-deaths in the town (Col. C) ; in and after July till October, hospital admissions remained steadily high, the medical wards being constantly full. I was absent from this hospital for the next five months ; the hospital admissions were fewer (see Cols. D and E) ; on resuming charge in April, 1878, there was a fresh exacerbation of so-called remittent fever, lasting till May (see also town deaths, Col. C), and then a decline took place to the end of the year, when I again left. Col. E very closely repeats these febrile movements : they are less evident in the town registers, which deal with fatal events only. In 1879, the J. J. Hospital entries approached the normal, yet with this quasi-normal state relapsing fever was present ; which is a point worthy of notice, as indicating how at the same institution the end of an epidemic comes to resemble the beginning. That the fever had not ceased was, however, fully proved by observation at the G. T. Hospital ; instances still occurring at the time of my second departure from India, early in 1880.

Analysis of the above Data.—Little is known of any period before April 1877 ;¹ from the following month of May until that of December 1878, no fewer than 1,468 cases of 'remittent' fever were admitted, or about four times the mean of the preceding years 1875-76. Of these 1,249 were specially noted : 910 had the blood examined, and in 509 (near 56 per cent.) the spirillum was seen ; in the remaining 401 it was not found ; there were 339 cases less acute or obviously complicated, which were not submitted to minute scrutiny ; total 740 cases, in which the blood-parasite was either not seen or not looked for. But these negative instances were admitted under conditions of date, race, home, lodging in the town, family, occupation, sex, age, and reputed illness, identical with those of the positive series ; and not seldom the history and symptoms of their fever were, also, highly presumptive of specific infection. In my judgment, therefore, it seemed likely that, altogether, at least three-fourths of the 1,468 admissions belonged to the famine-fever series ; *i.e.* a sum of 1,243, which is not far short of the actual excess (1,284) of admissions beyond the mean of two previous years ; and hence the inference that, in reality, ordinary remittents were not much increased at the height of the epidemic. As this remark will apply to sickness in the town, I should add that my opinion has been formed deliberately, and upon many grounds indicated in the following chapters of this work.

In detail, comparing these clinical data with the mortuary returns of the town, I note that the hospital gave earliest, fullest, and most continuous indication of the serious state of public health. Oscillations of the epidemic wave were necessarily less well indicated, yet the rise in May 1878 was concurrent. *Caste*.—Spirillum fever was much the commonest amongst Hindoos in 1877, and so fever in the town ; in 1878 it was more frequent amongst Mussulmans, and so fever-deaths in the town. *Residence*.—There is a remarkable coincidence between

¹ My appreciated predecessor in office, Dr. Henry Cook, informed me that, upon retrospection, he had found more than one instance of relapsing fever dating back to the autumn of 1876 ; and experience at the G. T. Hospital was equally explicit. See below.

localities showing most fever-deaths and those furnishing most admissions for relapsing fever: *e.g.* Dongri was crowded with famine-immigrants from the Deccan, numbers of whom showed sickness and were removed to the hospital near, and many more died at their residence; from personal visitation of the houses I know that the two classes of cases were identical, the sole disease being true famine-fever. For Mahomedans, there was the instance of Nagpada (Kamatipura) in 1878, where also I inspected houses of my patients, and found in them relatives and others unwilling to go to hospital (they had a dread of autopsies), who were sick with the same specific fever, as proved by the examination of blood-specimens taken on the spot. According to the municipal returns the fever-deaths in Kamatipura were as numerous in 1878 as in 1877, which was not so in other crowded quarters; and the clinical data here furnished a rational and adequate explanation of this exception. *Occupation.*—‘Labourers’ was the common term for relapsing-fever admissions and for town fever-deaths, in unusual proportion; the great variety of professions named by my patients showed further how widely the new fever had spread; *e.g.* noting in 1878 several instances of itinerant vendors of tea and sherbet, I found also in the municipal death-returns that fever, outside hospital, had been unusually fatal in this small community. *Age and Sex.*—Under these headings fever-deaths in the town were somewhat unusually distributed, and an accordance herewith was clearly noted in the hospital statistics of relapsing fever, which showed here, as in Europe, a predominance affected of male adults. *General Condition.*—The results of defective nutrition were frequently most apparent, especially in wandering immigrants and beggars; yet in hospital a larger proportion of better-conditioned residents were admitted than was indicated in the town deaths: this difference being due to residents naturally seeking their accustomed aid, whilst strangers were ignorant and more prejudiced. Since generally the worst or abandoned cases of illness were brought to hospital by friends or the police, the death-rate of the fever might be expected to rise beyond that in European cities; and such was the case, it being 19.5 per cent. of treated, as contrasted with 5 or 10 per cent.; here, again, was no real anomaly, but rather an indirect confirmation.¹

The above memoranda are offered for the information of those who had not the advantage of being on the spot, for it was never supposed by medical officers at Bombay that the hospital admissions for ‘fever’ were different from cases of ‘fever,’ generally, in the native town; and I conclude that the inference of identity of nature, which follows from all ascertained likenesses of the two groups (their comparative severity alone excepted), is as valid as it is natural. As samples only of the larger town statistics, hospital data might not quite tally in numerical proportions; but as regards character and distribution of the fever, they would be and are a good and sufficient test.

¹ From a return I had made, it was found the mean number of all cases brought to hospital in a moribund state in 1875-76 was 297 yearly; in 1877-78 the annual mean was 612, and the monthly maxima corresponded to dates of most admissions for ascertained relapsing fever. In normal years the death-rate of remittents at the hospital was (mean of five latest) 31.3 per cent.; in 1877 the deaths were 292 in 1,073 admissions, or, in spite of the terrible distress then prevailing, only 27.2 per cent.; in 1878 the rate was 28.7 per cent., and in 1879 it rose to 36.6 per cent.; compare these years in Col. D of the Chart.

The special value of hospital experience is further illustrated by the following analysis of 487 cases of demonstrated spirillum fever.

1877.—Hindoo Marattas were mostly affected, there being 206, with 34 deaths; whilst of Mussulmans of varied origin there were only 50 admissions, with 6 deaths. The Hindoos came from the Deccan famine districts (even the more distant), with a contingent from the Concan, doubtfully indigenous; herein is strict accordance with the town fever mortality.

1878.—These proportions were now reversed, for of 235 admissions, chiefly in April and May, only 54 (with 7 deaths) were Hindoos, mainly from Kattiawar; whilst 173 (with deaths 30) were Mahommedans, mostly from Northern India; here, too, is close accord. And, to conclude, all the particulars collected prove clearly the late epidemic in Bombay was of composite character, being connected with dearth in several countries through famine immigrations, which kept up the supply of susceptible material, in addition to that afforded by the resident pauper population. Hence, also, the sustained character shown in Column C of Chart 1.

As to type of fever, the Hindoo agriculturists in May 1877 showed the most acute or sthenic form; and the Mussulman weavers in 1878 a less pronounced, typhus-like and asthenic type.

SUMMARY OF THIS CHAPTER.

The late excessive fever sickness in the town was independent of local dearth; the circumstances of its first appearance and early course indicate its non-malarious character; upon analogical grounds it was likely to be true famine-fever; and, in fact, specific pyrexia characterised by the presence of the blood-spirillum, was detected in a large number of instances. This combined testimony points to but one conclusion, which is not contradicted by other facts, and which legitimately explains all the chief phenomena of the epidemic.

APPENDIX TO THIS SECTION.

Origin of the late Epidemic.—The little that is known may be usefully recorded, both for future guidance and as evidence that sickness began gradually and spread contingently, at Bombay as in European cities.

The earlier hospital records were seldom complete enough for use here, and even fuller data of the ordinary kind might not serve, unless the blood state were also ascertained.

It is open to conjecture that (*a*) the fever began *de novo* amongst the starving immigrants, but see below; (*b*) that sporadic cases have always been present in the town, without being recognised; (*c*) that sporadic cases were imported from the Deccan, Kattiawar, Northern India, or possibly from Persia by sea. Either of these last suppositions is probable; and, reckoning the epidemic to date from late in 1876, I find on analysing the hospital data of that year, amongst 35 fever cases,

8 like relapsing fever, 8 of doubtful character, and 19 of the usual remittent type. In March (*i.e.* eight months before the recognition of public sickness) the following example occurred at the G. T. Hospital:—

CASE VI.—A. D., adult, hospital assistant in the wards but sleeping at his home in the Kurruk (a notorious fever quarter in 1877), fell ill outside hospital, and was admitted March 22, 1876; there was then high fever of four days' standing, which terminated suddenly on the eighth day, the even. temp. sinking from $104^{\circ} \cdot 2$ (at midnight the *perturbatio critica*) to 97° and subsequently lower, next morning, great depression ensuing: again, on the estimated sixteenth day of disease, high fever returned, lasting six days and terminating critically; afterwards no rise of temperature. I have considered the original notes and chart, and taken the evidence of eye witnesses qualified by subsequent experience to give an opinion; and should infer that this case was indubitably one of relapsing fever acquired either in hospital or at home.

CASE VII.—An adult man, wandering mendicant, lately from Oude in Northern India, was admitted into the J. J. Hospital, April 16 1876, with fever of ten days; pains, diarrhoea, collapse and jaundice followed, and on sixteenth to nineteenth days a febrile relapse, with nervous symptoms; convalescence protracted. There was public scarcity and fever sickness in Northern India at this time. Here, too, the inference seemed clear. A later example was that of a young man from Wai (Deccan), destitute, one month in Bombay; had high fever with rheumatoid pains and other common symptoms; a sudden fall of temp. on ninth day from $105^{\circ} \cdot 5$ to $98^{\circ} \cdot 2$, and a typical relapse on nineteenth to twenty-third day of disease; thenceforward convalescent; date of admission November 27, 1876.

There are other like cases amongst individuals some weeks resident in Bombay, who doubtless acquired their disease in the town, about these dates; the town death-rate had not then risen, and only with advent of pauper crowds did the fever and mortality begin to abound.

NOTE ON A PREVIOUS FEVER SICKNESS.¹—During the years 1863-4-5, Bombay was visited by an epidemic even more severe than that under review; numerous cases were admitted into the J. J. Hospital, where the diagnosis first suggested was that of typhus, caused by large immigration of labourers, with consequent excessive overcrowding and defective food supply; actual famine nowhere prevailed, yet as at St. Petersburg about the same date (1864-65) and under similar conditions, a form of recurrent typhus might, I venture to think, have been present (see below). I have perused with care the available records of this era, and agree with the earlier diagnosis rather than with the later one adopted of 'remittent' fever; it was distinctly intimated that the fever was the same both in and out of hospital; and I find of the establishment in attendance on the sick, there died in 1864—female nurses, two; vernacular students, four; and ward boys, three; the remarkable experience of the years 1877-78 being thus, as it were, anticipated. In the town there were 12,953 deaths from fever termed

¹ The municipal records date back only to 1848, and doubtless were at first incomplete. There is no recollection, I found, of unusual sickness at that period; yet it was worth noting that during 1848-50, Dr. Morehead saw a number of cases of remittent fever, fatal with jaundice; and of those described, Nos. 54 and 56 (*loc. cit.* vol. i.) are, to my apprehension, strongly suggestive of the icteroid form of spirillum fever. Such suggestion is strengthened by the fact of all these ten cases occurring in indigent subjects; that the like were not seen at other dates (being extremely rare in hospital practice at Bombay), and that during 1877-78 specific *typhus biliosus* was regarded by high authority as identical with the so-called 'malignant bilious remittents.'

remittent, and during 1865 no fewer than 18,767 deaths, the previous equable normal mean being 6,684 yearly ; the maximum mortality during the worst year occurred in the hot weather months of March, April, and May (so in 1877) ; public sickness subsided rather abruptly in June 1866, that being the time when labourers commonly return to their country homes, and the rainy season begins. So few medical data were procurable that I must hesitate to express a decided opinion on the nature of this great calamity ; yet that it was not a malarious remittent may be safely assumed ; and if common typhus, the course and decline would be exceptional ; in a strict sense, but one inference remains—there being a single form of disease known which is adapted to explain the conditions stated. Whether or not infection could have been conveyed into the town was, it seems, not suggested.

CONCURRENT FEVER EPIDEMICS.—This subject has been ably discussed by the late Dr. J. L. Bryden in Chapter III. of his Statistical Report of 1876 (Calcutta, 1878) ; and whether or not opinion coincide in their interpretation, the copious data collected in this document claim a separate notice here, from the connection which some appear to have with sickness at Bombay.

Referring to the severe local epidemic of 1863-4-5 (see above), it is distinctly stated by the health officer of Bombay that the fever followed on immigration of labourers, seeking employment on the numerous works undertaken during that heyday of cotton prosperity. I do not learn whence the labourers came ; but if custom have not changed, working men, especially of the weaver class, must have arrived from the northern provinces of India ; and, assuming this to be the case, it becomes important to remark that, according to Dr. Bryden, in 1862-63 a great fever outbreak had occurred at Agra ; at Delhi, also, relapsing fever was present in 1864, and at Umballa, Lahore, Umritsur, and in Central India ; these being localities also named by fever patients under my care in 1877-78, who were likewise immigrants. The epidemic extended to the north of the Bombay Presidency, and is said to have been 'evidently yellow relapsing fever' (Rep. p. 193). Some details are contained in the Indian Annals, 1865, of this disease as seen in Western Malwa by Dr. H. C. Brodrick, and in the 'Madras Quart. Jo. Med. Sc.' v. ix. and x. there is an account of fever at Bangalore, to the south-east of Bombay, which had many aspects of relapsing fever ; date 1865. Further off, yet still in communication with the Western Presidency, viz. at Calcutta in 1864, typhus was reported, being probably connected with an epidemic affecting the villages of Lower Bengal. This 'typhus' fever was introduced into the Demerara emigration depôt, some only of the coolies affected being from Lower Bengal, the rest from Upper India where typhus was raging, and they were in the depôt only a few days before embarkation : it was also conveyed by emigrant labourers to Assam and to Réunion (*loc. cit.* p. 200). Respecting this transfer to the distant island of Réunion, particulars are available which tend to show the true nature of the imported disease ; Dr. H. Lacaze ('Union Médicale,' 1865, vol. ii.) stating that the fever was of a new form, distinctly contagious, and presenting many of the features of icteroid relapsing fever.

I am disposed to agree with him, seeing that jaundice is extremely rare in *typhus exanthematicus*, and that 'bilious remittents' are not contagious. Last'y, there is the history of a concurrent epidemic at the island of Mauritius, where the disease was popularly termed the 'Bombay fever,' from its having been introduced by immigrants from Bombay. In the Trans. of the Epidemiolog. Soc. of London, vol. 3, 1867, there are several descriptions of this outbreak of 'pernicious fever,' the general argument of which is that the type was that of an aggravated malarious 'remittent.' An Official surgeon, however, regarded the fever as 'true typhus' (p. 193). In the absence of detailed cases and particulars needful for strict independent judgment, I was unable to decide whether or not 'recurrent typhus' was ever indicated; but there are points named, which suggest this interpretation.

Fever Epidemics concurrent with that of Bombay during 1877-78. Whilst it is indubitable that the great bulk of local sickness prevailed amongst famine-immigrants from the Deccan, there was evidence of famine-fever being present in the town prior to 1877, and during 1878-79 it was commonest in subjects coming from Northern India. The reports of the provincial Sanitary Commissioners prove that drought and fever of a severe type were widely prevalent at this epoch. In the Punjaub fever was of continued character and not due to malaria, relapsing fever being distinctly recognised in 1876-77; here mortality was even more excessive in 1878, relapsing fever proving very fatal in the southern districts, especially in the towns and rural circles to the south of Delhi (whence many of my patients directly came); there was not then actual famine, but the poorer classes were much distressed for food and suffered from unusual hardships: that true typhus existed was clearly demonstrated in some parts. In the North-Western Provinces and Oude, fever was unusually prevalent and fatal during 1876; in 1877 there was much at Lucknow and other large towns, of continued type: typhus, relapsing and enteric fevers are expressly recognised: in 1878 the existence of contagious fever is also admitted. In Bengal during 1877, fever became epidemic in certain districts; at Midnapore the attacks seldom lasted more than three days, but relapses frequently occurred after a week or so, the disease being widely different from the epidemic of 1871-76; and at Serampore, with overcrowding and dearth of provisions, fever arose. In the Central Provinces nearer Bombay, mortality from this cause was high in 1876, also in 1877, and still more during 1878, when in the most affected district of Nimar, the presence of non-malarious relapsing fever seems to be clearly indicated; cases being seen not only amongst emaciated and starving subjects, but also prisoners admitted to gaols, and at all the dispensaries. Migration from this part to Bombay by rail is known to have taken place largely.

In sum, there is conclusive testimony of widespread and severe fever sickness in other districts than the Deccan, whence also the facilities of communication were considerable. The need of precise clinical details and diagnosis, may be keenly felt during the perusal of the official Reports; nor, must it be added, were the opinions of the writers uniform on even fundamental points.

GENERAL HISTORY OF RELAPSING FEVER.—A comparison of late events at Bombay with the Irish epidemics of 1846-50, naturally suggests itself: and on referring to original documents, I find much to indicate that with identity of certain conditions, there may have been a partial identity of disease. It is understood that the Irish fever was mainly of the relapsing kind.

In the late absence of famines in Europe, data still exist for comparison with the Bombay epidemic of 1863-4-5; *e.g.* the history, which I have also considered, of events at St. Petersburg during 1864-65, displays even a precise similarity of originating influences. Fever was said to be imported into the city, Moscow being mentioned in this connection; relapsing fever was at Odessa in 1863, at Riga in 1865, and, more noteworthy, it then prevailed in Kasan, the most easterly province of Russia in Europe (E. long. 60°, confines of India 70°). Information regarding countries further East, is necessarily wanting; yet, bearing these concordant dates in mind, an impression arose which must have occurred to others than myself, since Dr. A. Hirsch ('Handb. d. Hist. Geogr. Pathologie,' Stuttgart, 1881) alludes to the possibility of there being a central focus of relapsing fever in Asiatic provinces lying between Russia and India.¹ The existence of such a focus is not, however, essential to the comprehension of long conveyance of this disease.

Analogy here with the natural history of Cholera must be obvious; this transportable malady having pursued a course similar to the one suggesting itself for Spirillum fever.

There only remains to remark that, contrary to some early anticipations of physicians, the most strictly epidemic of human fevers, on the continent of Europe now displays a tendency to become endemic (to be 'naturalised,' as expressed by the late Prof. Lebert); and, supposing that relapsing fever were not already indigenous there, it remains to be seen if its endemicity be or be not established at Bombay. I possess the notes of cases extending from early in 1876 to the beginning of 1881.

For other remarks on the epidemiology of Spirillum fever, see SECTION III., *Chapter 3*.

¹ Dr. Morache (Recueil d. Mém. de Méd. milit. fevr. 1866) has briefly described a mild epidemic of typhus 'with cases of relapsing fever' observed at Pekin during 1864-5, or about the time when so much identical sickness broke out at St. Petersburg. The author insists upon these simultaneous dates, and states that the fevers might be traced eastward across Asia as far as Kiakhta on the Chinese frontier; the sanitary condition of Mongolia was not known, but in the North of China proper typhus and relapsing fever seem to have been recognised. There had been an inclement winter, and the people had suffered. Typhus and enteric are said to be common in Chinese towns and villages; the relapsing fever element was identified by Dr. Morache upon the receipt from Paris of medical intelligence respecting the new fever at St. Petersburg, and previously it had been considered as a form of typhus. Here may be remarked the utility of promptly disseminating fresh clinical knowledge; and especially noteworthy is this additional evidence of a great epidemic epoch dating about 1863-5.

EXPLANATION OF CHART No. 1.

COLUMN A.—The numbers of individuals on relief works is the total present in last week of each month : the decline in February was incidental ; the brief rise in September was due to apprehension of renewed want, from defective rainfall. In 1878, the daily maximum of relieved amounted to 19,544, and in 1879 to 17,656. No relief works prior to 1876.

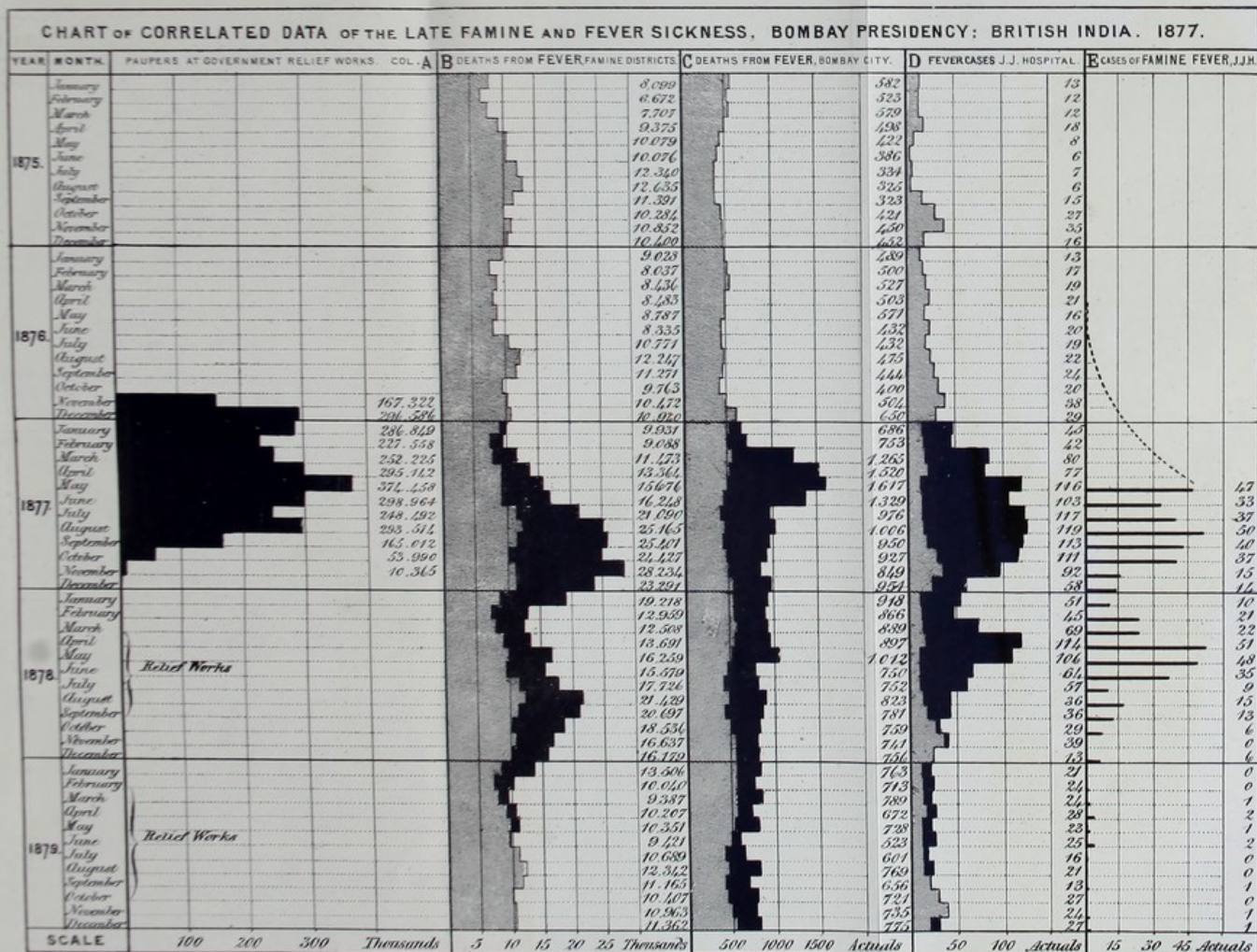
COLUMN C.—The light-tinted bars from 1877 onwards are the means of actuals in 1875 and 1876, being superadded to show better the actual *excess* of deaths (black-tinted bars) during 1877, 1878, and 1879. These means for the months in order are: January, 8,563, 7,354, 8,071, 8,929, 9,933, 9,205 ; July, 11,555, 12,441, 11,331, 10,023, 10,662, and 10,678 : sum 118,745. Yearly actuals—1875, 119,910 ; 1876, 116,570 ; 1877, 223,388 ; 1878, 201,418 ; 1879, 129,840. Augmented mortality is seen to have begun shortly after the recognition of public distress ; prior to June there was much emigration from these districts ; after June many returned : similar movements in 1878. In 1879 the epidemic seems to have become exhausted here.

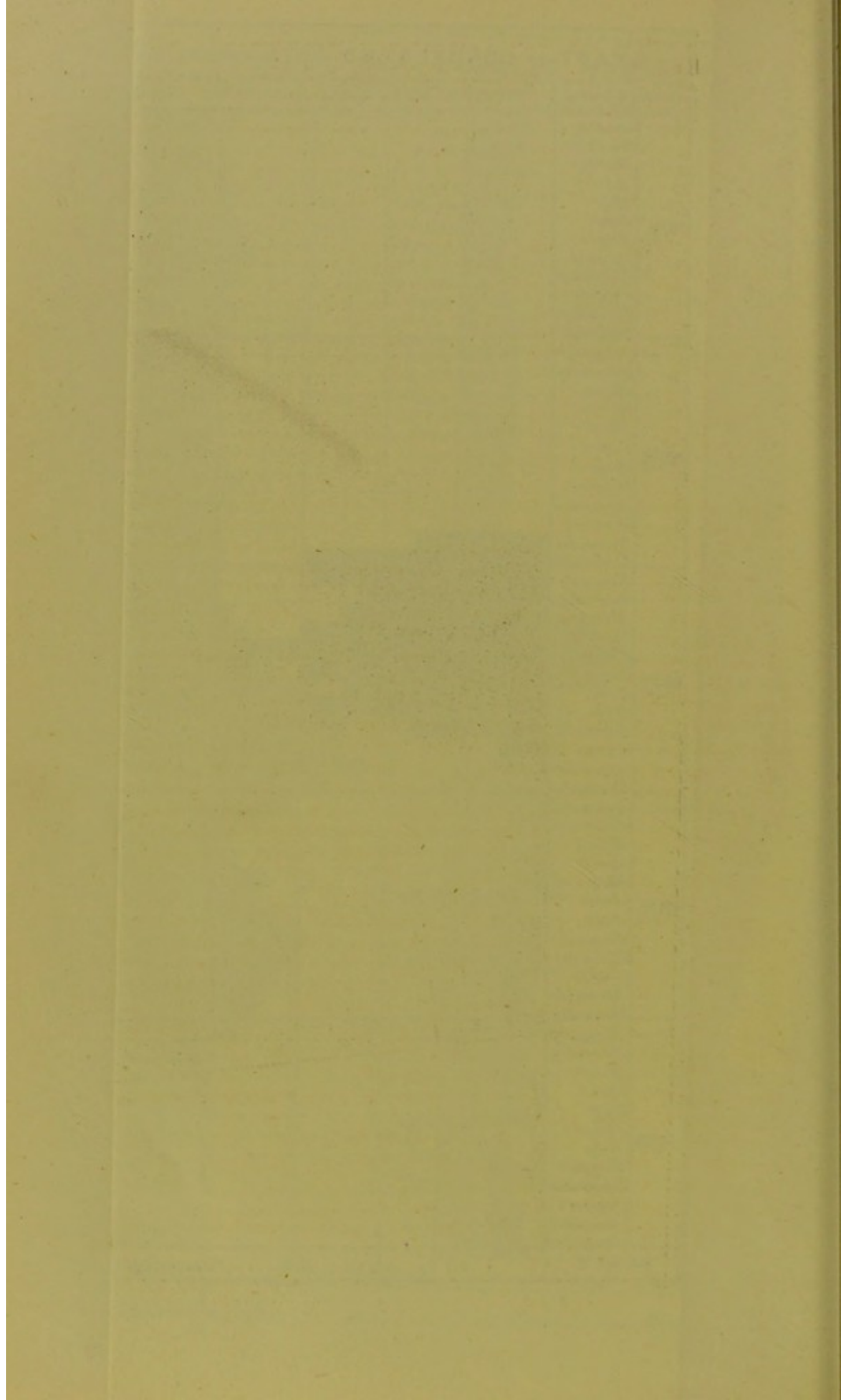
COLUMN C.—The pale bars for 1877 onwards are the means of actuals in 1875 and 1876, being superadded as in Col. B. These means in order are for January onwards, 535, 511, 523, 500, 466, 409 ; July, 382, 400, 383, 405, 481, and 550 : sum 5,545. Yearly actuals—1875, 5,244 ; 1876, 5,867 ; 1877, 12,832 ; 1878, 9,944, and 1879, 8,445. The augmented fever mortality is seen to have begun about the same time as distress in the Deccan, and to attain its maximum in the main famine year of 1877 at nearly the same time ; this being also the date of largest immigration from the affected districts. Slow decline of this excess is referable, partly to townspeople becoming implicated, and partly to immigration of paupers from other districts than those of the Deccan ; this cannot be shown here.

COLUMN D.—The admissions are those of so-called remittent fever, there being no recognised heading for famine-fever. The pale bars indicate as in Cols. B and C, the normal means for 1877 onwards being as follows—13, 14, 15, 19, 12, 13, 13, 14, 19, 28, 36, and 22 : sum 218. Yearly actuals—1875, 175 ; 1876, 267 ; 1877, 1,073 ; 1878, 639, and 1879, 273. The augmentation of fever admissions, in 1877, is contemporary with the recognition of public distress (Col. A) and with the excessive mortality of Cols. B and C ; its persistence and the rise, in 1878, are due to influences not shown in this Chart ; see Col. C and the text.

COLUMN E.—The earliest appearance of undoubted famine, relapsing (or spirillum) fever may be indicated by the dotted line : the maximum numbers, in 1877, correspond with fever admissions in Col. D—the earliest being contemporary also with *plus* fever-deaths in Bombay and *plus* public distress in the Deccan. In 1878 fresh races and castes furnished many cases of fever (*vide* Cols. C and D) which were now discriminated with care. The decline of specific fever at the J. J. Hospital in 1879, as here shown, requires to be supplemented by experience at the G. T. Hospital (*vide* the text), when it will be found to correspond better with fever-deaths in the town at same dates.

CHART No 1.





SECTION II.

CLINICAL HISTORY OF THE SPIRILLAR INFECTION.

CHAPTER I.

SOURCE AND SUM OF DATA.

By an excellent regulation the medical officer in charge of sick should enter daily, in printed forms, the state and progress of every patient under his care ; and I am bound to state that this injunction was faithfully carried out, under circumstances of unusual stress, at the hospitals under my superintendence. Considerable advantage, too, at the College Hospital was derived from the more elaborate records of the Clinical wards ; where under the eye of able and experienced teachers, many special details were elicited. Investigation by means of the microscope, as an extra-official work, had to be separately performed ; and in this I had the willing and valuable assistance of Mr. Succaram Arjoon and Dr. A. M. Kuntay. It was my practice to see personally every 'fever' case on admission, and then to examine the blood ; and had this not been done, many useful data would have been lost. Owing to the large amount of current duty arising from the excessive public sickness, scientific research was necessarily deferred ; and the prior object being to establish the diagnosis of this new disease upon a wide and adequate foundation, little time was left for particular enquiry : afterwards, attempts were made in this direction.

Sum of Data.—The materials collected at the J. J. Hospital between May 1877 and December 1878, comprise notes of 1,249 cases of remittent fever (so-called) ; in 339 of which the blood was not examined for want of opportunity or from the absence of defined symptoms (Class A) ; in 401 the blood-spirillum was not found upon microscopical scrutiny (Class B)—both these groups including, besides remittents, some instances of symptomatic pyrexias retained for the sake of comparison. The remaining cases, amounting to 509, all displayed the blood-parasite ; and it is upon this group (Class C) that the following clinical details are mainly founded.

At the G. T. Hospital, during the intervals of my tenure of office, 110 cases of Spirillum Fever were seen ; and also a large number of other cases, which have been employed as data for the section on Remittent Fever.

At both these hospitals the officers in charge, permanent or tem-

porary, likewise made special investigations ; with the result of confirming and continuing the present series of positive data.

It has been previously stated that at the Camp of Refuge opened in April 1877, several cases, and the earliest of all, of genuine famine-fever were made known.

Thus, the entire number of certified instances of specific fever of which the notes are available, amounts to 650 ; and in 624 of these the main particulars were entered personally, or under my own inspection.

General Character of Data.—Partly for future guidance, it will be useful to mention past experience under this heading. According to European standards, the hospital accommodation at Bombay is very inadequate (there being only 600 beds to more than half a million of people) ; but, owing to various prejudices, it is not the Native custom to resort early or freely to European medical aid ; and new comers from the interior were more than usually indisposed to seek for admission. Hence very few instances of relapsing fever were seen at the beginning of illness ; and almost always a week or so had elapsed before our help was sought. This circumstance was peculiarly unfortunate, since this period is about the mean duration of the first attack, and if the case were not promptly diagnosed, its nature might remain obscure for another week (till the relapse set in) ; or in case of early decease or absence of relapse, it might never be strictly verified. Commonly the last day or two alone of invasion were witnessed, when alarm at the rising symptoms induced the friends of the patient to seek for aid ; and sometimes it was the equally alarming state at critical fall which impelled them : many cases were not brought until it was evident that convalescence was being checked by accessory fever or local complication ; and, finally, there might be available for analysis only the history of a late febrile attack to account for anomalous symptoms or general prostration. These deficiencies were, however, to some extent made good by the many instances of contagion in hospital, which happened at intervals throughout the epidemic ; and which usually displayed the entire specific illness. The first apyretic interval and recurring attacks of fever were abundantly illustrated ; but the latest stages and ultimate effects of infection were less well displayed, as patients usually insisted on leaving hospital with returning convalescence ; this defect is not, however, entire ; and, generally, it is probable that all the chief modifications attending the late epidemic, did in course of time pass under observation.

Lastly, owing to prejudice of relatives, autopsies were sometimes impracticable when it was very desirable they should be made ; yet so many friendless subjects reached hospital, that nearly adequate information of structural changes became available. And after making due allowance for the local circumstances impeding full enquiry, there still remains a collection of evidence, which, I think, may fairly rank with most European data acquired with more adventitious aid. Every one of the records made use of in the present work is still open to inspection, and each has been carefully and repeatedly scrutinised ; for of all none has proved uninformative or useless for analysis, if only rightly used.

CHAPTER II.

DEFINITION AND CLINICAL SUMMARY. ILLUSTRATIVE CASES.

Definition and Designation.—Pathologically, as well as clinically, the spirillar disease should be regarded in its entirety ; and thus viewed, it presents both febrile and non-febrile phenomena.

Inevitably, the complex pyrexial state has attracted most attention ; and by it, the infection is currently known as a peculiar kind of 'fever.'

Yet in order of time, such fever is but the culminating event of a prior non-febrile period of blood contamination ; which, though offering comparatively few symptoms for clinical notice, is yet an essential part of the disease, and constitutes the so-called 'incubation-stage.'

The spirillum 'fever,' in man, is remarkable for its tendency to recur at tolerably regular intervals ; its successive febrile repetitions becoming gradually less sustained, and the intercurrent non-febrile stages more prolonged : in this way, whilst relapsing the disease spontaneously declines.

For convenience, the febrile events are in order of succession named the first or 'invasion' attack, the second attack or 'first relapse,' the third attack or 'second relapse,' the fourth attack or 'third relapse,' and so on. Of the antecedent non-febrile periods, the earliest, or that preceding invasion, does not commonly come under notice ; the second, or that intervening between invasion and first relapse, is here termed 'the first apyretic interval ;' the second, coming between relapses one and two, is the 'second apyretic interval ;' and the third, between relapses two and three, is the 'third apyretic interval.'

As to clinical variations of the disease, whilst the invasion is always pronounced, the relapses differ considerably in intensity ; and most so the later. Sometimes there is no palpable recurrence of fever after the first attack (which has then been termed 'abortive'), or only a slight periodic indication ('suppressed' or 'latent' relapse), a similar indication oftener, however, standing in place of later recurrences : commonly one distinct relapse takes place ; occasionally a second, and still more rarely a third or fourth.

All pronounced pyrexial events present well-defined limits and high range of temperature, a rapid pulse, severe body-pains, and especial implication of the viscera in the upper abdominal zone. The apyretic intervals represent a quasi-normal condition of the frame. Visible blood contamination by the spirillar organism is at first scanty, and becomes increasingly evident during fever ; until the acme of attack, when both blood infection and pyrexia abruptly cease.

In early and severe attacks, about this acme-epoch local accidents

(such as hæmorrhages) are apt to arise ; and other complications (as secondary fever and local inflammations) commonly date thence, or immediately afterwards. Death from fever is usually referable to the same time ; and with all these events, certain blood changes concur. Sequelæ are not very common.

So far as yet known, the spirillar disease is always acute. This infection is said to arise spontaneously (in a clinical sense) oftener than some other acute infections ; but, once developed, it is decidedly contagious. A close, if not absolute, relationship exists between presence of the spirillum and manifestation of constitutional symptoms.

Clinical Designation.—Having become assured by unprejudiced observation, that the late Bombay fever is not only a veritable species of disease, but also identical in character with the ‘relapsing’ or ‘famine-fever’ and the ‘recurrent typhus’ of Europe, I consider it desirable to adopt a more exact designation for this affection, on the following grounds :—Practically, a considerable proportion of surviving cases in man did not relapse ; and, as a rule, but one attack was produced by inoculation of the *Quadrumana* ; both these data pointing to the presence of a simple and, therefore, fundamental character of the infection in question. Moreover, according to my experience, most fatal cases do not attain the recurrent stage ; and had their diagnoses rested on such relapsing feature, it could not have been accurately made.

Next, by far the largest proportion of cases, could not be said to result from starvation. Further, the term ‘typhus’ has in England acquired a definite meaning it is not needful to disturb ; nor does the Continental expression convey a thorough generalisation. To continue to apply to this disease the name of ‘remittent fever’ would be, I may add, to ignore the progress of clinical medicine ; and to increase a confusion of nomenclature, already embarrassing in practice.

Lastly, through many early perplexities having learnt the value of one characteristic, the presence of which was a never-failing guide to diagnosis, prognosis and treatment, I was in a measure impelled to adopt, as a synonym of relapsing or famine-fever, the cognomen ‘spirillum fever,’ which, whilst not excluding the appropriate use of current terms, will be found to apply where they are unsuitable.

Thus used, the word ‘spirillum’ stands provisionally for an organism, not entirely answering to accepted generic definitions of either ‘spirillum’ or ‘spirochæte.’

Description of the Disease.—The following summary account is based on records made chiefly during the height of the epidemic ; it refers to the successive febrile and non-febrile phenomena as commonly witnessed, with their general features, variations, complications and sequelæ.

First or Invasion Attack.—Prodromata were seldom recognised with certainty : those named amount to a sense of malaise felt shortly before the onset of fever (see the next Chapter). The blood-spirillum is probably always present, though scantily, at this time.

First day of illness.—Has been seen almost exclusively amongst

servants, or patients who were seized with fever in hospital. Twelve such records are available for analysis, all of which contain the initial temperature and the pulse, but sometimes little more; because the true nature of the attack not being always recognised at first, brief evening notes alone were recorded.

Hour of invasion.—Twice this was not certain, on account of there being some premonitory mild or irregular pyrexia; of the rest, fever set in just before or commonly after sunset in eight, and in two only was it observed first at the morning visit.

In all cases the onset was defined, and in most abrupt; pyrexia may or may not be attended with the subjective symptoms of chilliness, not amounting to rigors; severe headache (usually frontal) and pains in the spine and limbs, with a sense of weakness and indisposition for exertion; thirst, a coated tongue, and dry mouth; loss of appetite, nausea, or vomiting; costive bowels and a dry skin speedily follow; the countenance may even now become dusky, and indicative of oppression. Soreness and tenderness of the upper zone of the abdomen have been observed at this time, and twice at least the liver was enlarged; the condition of the spleen, which it is more difficult to ascertain when changes are only beginning, was seldom accurately learnt; but once at least the organ was sufficiently enlarged to project beyond the costal margin; the epigastrium is affected with uneasiness.

Though pyrexia be considerable, patients are not necessarily prostrated; thus, a native student on this day was found writing in the wards, with a temperature of 103° and his blood charged with the spirillum; and in my own attacks, by dint of great effort, the strong feeling of oppression was overcome for a few days longer, subsequent depression being possibly augmented by such effort. Children, and especially infants, seem to suffer little at first; the latter seek the breast as usual.

Pyrexia.—The mean evening temperature was $103^{\circ}\cdot2$ when fever came on in the afternoon, and $104^{\circ}\cdot5$ when it came on in the morning; in the last-named instances the temperature at 7 A.M. was $103^{\circ}\cdot2$, and there was usually a history of fever in the previous night, which doubtless represented the true initiation of the attack; an abrupt ascent of 6° was noted in two out of six cases of afternoon initiation, and there is reason to believe that the rise at evening, which is practically invariable, may continue during the earlier part of the night. Considerable uniformity in these initial rises is noticeable in the mild typical cases under notice; the maximum morning temperature on the first day was $103^{\circ}\cdot8$, with 105° in the afternoon; the maximum initial evening temperature was $104^{\circ}\cdot2$. The minimum temperature noted on this day was 102° in an infant.

The absolute initial rise was, in the mean, $4^{\circ}\cdot7$ in the morning, and $4^{\circ}\cdot9$ in the evening onset, which shows how nearly alike the temperature is at 7 A.M. and 4 P.M. of the first day of attack.

In two fatal cases occurring amongst the hospital establishment, the precise date of onset was obscured by paroxysms of intermittent character which were not specific; and in two other instances out of thirteen there was a single aguish attack immediately before the commencement of persistent fever; such attacks have been noticed also ten, fourteen,

and twenty days prior to specific invasion—their significance is not clear.

Pulse.—Shows variations considerable, and seemingly dependent upon personal idiosyncrasy, sex, or age. The mean of initial morning temperature being $103^{\circ}\cdot1$, that of the pulse is 112, and initial evening attack 116, the temperature being also slightly higher. No fixed relation of pulse and temperature is noticeable; though on this day the evening pulse, like bodily heat, is usually higher, and never lower, than in the morning. The cases under review display an unusually rapid pulse in woman and child; and, on the other hand, a sluggish pulse in two men out of seven. For example, a child with initial evening temperature of 102° had a pulse of 124, and a woman with a similar morning temperature a pulse of 114; whilst a man with initial morning temperature of $103^{\circ}\cdot6$ had a pulse of only 96, and at evening (105°) of 106 only; and another man with an initial evening temperature of $104^{\circ}\cdot2$ had a pulse of 104, which contrasts greatly with a third who, under an evening temperature of $104^{\circ}\cdot6$, had a pulse of 136. Putting aside exceptions, the average pulse of adult men on this first day may be 120, and that of women and children slightly more, the temperature being about the same for all.

The quality of the pulse is expressible thus early as soft or compressible, even if rather full, and it is sometimes also small.

Second day.—The pyrexia is but slightly abated, the mean morning temperature in these same cases being $103^{\circ}\cdot1$, that of evening $103^{\circ}\cdot3$; in half the cases the morning temperature was slightly higher than that of evening, doubtless from exacerbation during the night. The extremes of morning temperature were $101^{\circ}\cdot4$ and 104° , the extremes of evening temperature $102^{\circ}\cdot4$ and $104^{\circ}\cdot4$, being steadiest as well as highest.

The pulse follows the temperature, and it shows even more strongly in its frequent morning excess, the disturbing influence of the febrile invasion; thus the morning pulse may be quickest even when the morning temperature is below the evening temperature; but I should add that the reverse obtains sometimes, and the inference from these observations would be that the pulse both rises more promptly and subsides more slowly than the bodily heat, as commonly measured, in response to the hidden pyretic influence of this form of fever. It is marked as small and feeble. The mean morning pulse was 124; that of evening about 118; and while admitting that the rapidity of the pulse is liable to many contingencies, still this statement has its import. In the bi-daily readings under analysis, evidence of minor pyrexial movements is necessarily absent, but such movements are known to occur.

The headache often increases, and may be intense, without regard to temperature and pulse; injection of the eyes is noted; the coated tongue is still white and moist, but in a case of typhus tendency it was dryish; the bowels are costive, thirst is considerable; the liver and spleen are full and tender; the pains continue; the skin was dry only twice; nausea and vomiting were sometimes present in the morning, in conjunction with severe headache; the sleep is disturbed; the urine was noted as scanty, specific gravity 1016, reaction acid, no albumen.

Third day.—The pyrexia is decided, but the morning remission is now constant, and perturbations due to the invasion disappearing, the

evening rise becomes invariable. The mean morning temperature is $102^{\circ}\cdot4$, that of evening temperature $103^{\circ}\cdot6$: the maxima respectively $104^{\circ}\cdot8$ and $103^{\circ}\cdot6$; the minima $101^{\circ}\cdot2$ and $101^{\circ}\cdot8$ (which last is exceptional).

The pulse has become slower, the morning mean being 112, the evening 116 ; it is less sensitive, yet the morning pulse is not very seldom quicker than might be anticipated from the consentaneous temperature ; instances of unusually slow and rapid pulse are noted.

The headache persists, but may abate, it seems to hold no fixed relation to the pyrexia ; the tongue is in the same state, a florid tip and edges is entered ; bowels require aperients ; thirst is great ; the liver is usually large and tender, three times in fifteen it alone was implicated, whilst the spleen, usually affected in the same way, was alone changed twice ; the uneasiness in both these organs is liable to be increased upon deep inspiration, or in the act of coughing, and the whole abdomen is often full and tender ; not excepting the hypogastrium, where especial uneasiness, without distension of bladder, may sometimes be felt ; the aching pains are great, particularly in the loins and lower limbs ; the state of the skin varies ; vomiting is noted, concurrent with febrile exacerbation ; appetite impaired ; sleep wanting, even if little or no headache ; cough (bronchitic) now appears in some instances, and jaundice is noted, with more frequent or marked hepatic congestion ; epistaxis happened once. The urine had a specific gravity of 1017, was acid in reaction, and contained no albumen.

Fourth day.—In about fifty cases submitted to analysis, the pyrexia offered several variations, which referred chiefly to depressions occurring in the early morning ; the most typical series was one of twenty-two cases, furnishing the following results :—Morning temperature—mean 103° , max. 104° , min. $101^{\circ}\cdot6$, range $2^{\circ}\cdot4$. Evening temperature—mean 104° , max. $105^{\circ}\cdot4$, min. $102^{\circ}\cdot6$, range 3° . With one exception the evening temperature was equal to or in excess (usually) of the morning temperature, so that now the prolonged effect of nocturnal exacerbations is rarely perceived next day. Pulse—at morning the mean was 113, max. 125, min. 94. At evening, 117, 130, and 108 ; so that the pulse responds freely to degrees of pyrexia, and it, too, was once only quickest in the morning.

In two other series of cases the mean temperature was somewhat lower, but not generally the pulse ; the relations of pulse and temperature, which on the whole are uniform, may be disturbed by exceptional instances, wherein the pulse is either sluggish or irritable ; its debilitated character was marked, with one exception where there probably co-existed hypertrophy of the heart.

In four out of twenty-two types, the critical subsidence of pyrexia took place upon this day ; they were all very mild cases.

The general symptoms undergo little modification, but some changes are noted ; thus, headache may be mitigated ; the tongue tends to dryness, with a brownish fur ; there may be diarrhoea ; thirst is very prominent ; the liver and spleen are persistently implicated ; the pains are about the same ; the skin is dry when fever is persistent ; vomiting is noted ; the appetite continues impaired, with an occasional exception, when hunger is expressed ; no sleep ; cough is more frequent

(congestive); the peculiar facies of the disease is well marked, and in general there is augmenting weakness, emaciation and distress. In the instances of crisis headache had subsided, the tongue was coated white or brown, there was vomiting, once epistaxis; the spleen continued large, there was exhaustion, a feeble pulse, and, in all, the body was covered with clammy sweats.

The urine had a specific gravity of 1010-18; chlorides varied from $\frac{1}{8}$ to $\frac{1}{4}$ the volume, and there was a trace of albumen once in three cases examined.

Fifth day.—In these ordinary examples the pyrexia remained constant, but there is a tendency to vary. The temperatures were as follows:—Morning—mean 103°, max. 105°·3, min. 101°·8, range 3°·5. Evening—mean 104°, max. 105°·4, min. 102°·4, range 3°·6; individual cases show a nearer approximation of temperature early and late in the day, so that the pyrexia appears more continuous in character; very seldom the morning temperature was slightly higher than the evening temperature. The pulse shows a decided increase in frequency, so that it may be said to rise with the progress of the fever; for morning the mean, maximum and minimum were 118, 147, 100; and for evening, 123, 142, 100; as the morning pulse may be augmented when the temperature is not so, it appears that its irritability is again increased, so that the influence of nocturnal pyrexia becomes perceptible once more.

In three of twenty-two cases the critical fall occurred this day, and then twice in the morning.

As to general symptoms, the headache may be increased, the tongue brown and dryish, thirst follows the temperature (very rarely it is lessened); the liver is not freshly implicated, but the splenic enlargement may be now first noted; pains are more frequent; partial sweats have been noticed; vomiting and epistaxis are rare; cough is not more frequent; jaundice may be present; sleep is disturbed, and a new symptom appears, namely delirium at night (two in twenty-two cases).

The urine has been noted as scanty, specific gravity 1015, chlorides deficient, albumen absent.

Sixth day.—The fever is somewhat higher and more irregular, being often disturbed in its more or less level and continuous course by approach of the crisis, when a *perturbatio critica* is observed. The temperatures were as follows: Morning—mean 102°·5, max. 104°·6, min. 100°·6, range 4°. Evening—mean 103°·8, max. 105°·8, min. 101°·8, range also 4°, with a tendency to morning remission; the rise at evening, always present, is often considerable (*i.e.* 2° or more), but vacillations are noted so often that the mean evening temperature is slightly below that of the fifth day. The pulse almost reverts to its condition on the second day, showing, namely, a tendency to excess in the morning, or to equality with the evening pulse, which, on the other hand, exceeds in frequency in barely half the cases; the means, maxima and minima, of the pulse at morning and in evening respectively, were as below: Morning, 118, 136, 100; evening, 117, 142, and 105.

General symptoms.—Headache may be less; the tongue tends to dryness, with a brownish fur; bowels vary; more thirst; liver and spleen full and tender, the left lobe of the liver may be especially affected; vomiting rare; epigastric uneasiness varies; aching pains are usual; skin is harsh, or

dry ; appetite has several times been noted as considerable at this time ; sleep disturbed ; pulmonic congestion not increased ; jaundice same ; epistaxis may occur, dilated pupils and depression ; during acme at this time, rigors, restlessness, and sweats may appear, or acute delirium, which is, however, rare. The state of the urine has varied somewhat ; the specific gravity from 1015 to 1018, scanty or free, high coloured, acid, rarely a trace of albumen ; chlorides and bile pigment are present, the former from $\frac{1}{4}$ upwards. Exhaustion is still partly hidden by the pyrexia, but is manifested in the pulse.

In one-fourth of a small series of types the fall took place this day oftenest in the morning.

Seventh day. - Amongst ordinary cases reaching to this day, the pyrexia generally is less marked than on the previous day ; whence it appears that the longer duration of the fever does not entail its greater intensity. The temperatures were as follows :—Morning—mean $102^{\circ}\cdot4$, max. $105^{\circ}\cdot4$, min. $99^{\circ}\cdot8$. Evening—mean $102^{\circ}\cdot3$, max. $105^{\circ}\cdot2$, min. $99^{\circ}\cdot8$. The range for both times was $5^{\circ}\cdot5$, but variations in individual cases are so considerable that the mean ratios are of limited value ; and that of evening temperature would be higher if the crisis had not sometimes already commenced ; in this series there is no rise of striking importance. The pulse also varies considerably, and that owing chiefly to the impending critical fall. Morning pulse—mean 108, max. 130, min. 96. Evening pulse—mean 111, max. 124, min. 94 ; there is little difference between the pulse of morning and evening, though the morning temperature is slightly in excess ; yet the morning pulse is rather less, showing that the pulse is more sensitive than temperature, and now rises and falls quicker than before ; it would seem that the fall of pulse precedes that of temperature as the period of crisis draws nigh ; dicrotism is noted.

General symptoms—headache may increase ; the tongue may be less dry, the mean temperature being lower ; thirst continues ; diarrhoea may set in ; the liver and spleen are not more affected, and enlargement of the latter has been noted as going down ; aches are less ; the condition of the skin varies with the movements of fever, sweats attending the remissions ; bilious vomiting is noted ; appetite mostly indifferent, or bad ; cough is not usual, but pulmonary congestion may end in some degree of basal consolidation ; jaundice comes on sometimes ; abdominal uneasiness may be severe. Delirium is commoner (one-fourth of cases), and the *perturbatio critica* at this period may be marked. The urine varies in amount ; its specific gravity is still low, 1010–18, no deposit, no albumen ; when depression is considerable it may be passed in bed.

In nearly one-half of the instances the crisis occurred on this day, and oftenest in the evening.

Eighth day.—In a series of ordinary cases known or estimated to have reached this date, one-third only had pyrexia, which was hardly as high as on previous days. There is a tendency to equability of morning and evening temperatures, partly due to decline of the latter, in anticipation, as it were, of the fall next day. The means were $103^{\circ}\cdot4$ (morning) and $103^{\circ}\cdot9$ (evening), with a range of 2° or 3° . The pulse is also rather slower, and its evening excess less marked ; it follows the

temperature when this is materially higher in the earlier part of the day, and its irritability seems to be lessening, possibly by cardiac exhaustion; morning, 109; evening, 113.

General symptoms.—On the whole there is a significant absence of sthenic or severe symptoms, proportionate to the high pyrexia which may obtain; headache persists, and the conjunctivæ are injected; tongue coated, not necessarily dry (most patients drink frequently); thirst is present; the liver and spleen are not more affected, or pains greater, except on movement; abdominal uneasiness may be considerable; delirium not more frequent, but depression and the facies marked; the *perturbatio critica* is not common.

In one-third of the instances under notice crisis took place in the morning of this day.

Ninth day.—A few selected cases now remain in which the duration of the fever is stated with precision to be nine days, or longer; such have not been witnessed amongst the instances of contagion in hospital, but they seem to occur. The mean temperatures are high and uniform, being 104° morning and 105° evening, and this close upon the critical fall. The pulse, on the other hand, tends to become slower, the means being 111 morning and 112 evening per minute; it is always soft and usually small.

The general symptoms were not exacerbated, and may be even less severe, though debility becomes marked; the liver and spleen are less tumid and painful; sweats are not uncommon; the *perturbatio critica* was not noticed in this small series; urine, specific gravity 1018, neutral; chlorides reduced ($\frac{1}{4}$), no albumen, quantity moderate.

Crises happening on this day were in the evening.

Tenth day.—As reported, in these rare instances the pyrexia was commonly abated, the mean temperatures being 102° morning and 103°·7 evening, and the pulse 101 and 110.

General symptoms.—In severe cases headache persisted, thirst was marked; sleep disturbed, pulmonic congestion was noted, also jaundice; the liver was tender and large, the spleen not so; a trace of albumen was noticed in the scanty, high-coloured urine, specific gravity 1010, with bile; partial sweats occur. Delirium, most marked at night, is usual, which is a noteworthy feature; debility considerable. Decided hunger was expressed when high fever persisted; aches were common.

Critical fall took place in the evening, but was not particularly marked; it was rather prolonged, showing a tendency to lysis.

Eleventh day.—In rare cases the crisis was deferred until the morning (usually) or evening of this day. The depression was then considerable, temperature being 96°·5 and pulse 79. The general symptoms had been well marked, and especially visceral complication, of the liver oftenest, which still persisted at the fall. Delirium was sometimes present, and headache continued. The urine was found to have a specific gravity of 1010, and to be free from albumen.

The Crisis.—In the evening or during the night of the sixth or seventh day, there usually happened a brief augmentation of all the symptoms, with the addition of some delirium (*perturbatio critica*); and immediately after a complete reversion of symptoms takes place at this time, in the course of six to twelve hours the condition of the patient becoming

totally changed. This typical crisis is almost invariably attended with copious perspiration, which, beginning before the decline of the pyrexia, may persist long afterwards. The fall of temperature amounts to 5° , 7° , or 10° F., and in the morning the body-heat is 97° , 96° , or less; defervescence is most rapid at first; it may end in a condition approaching to collapse. The pulse also now declines, but not so promptly as does the temperature; it becomes small and feeble, and may be intermittent; the number of respirations also diminishes, though not yet to the normal rate.

Relief is at once experienced by the patient, the headache and abdominal uneasiness subsiding; deep sleep follows, the tongue speedily becomes clean, and excepting debility, there may be little peculiar to note. Occasionally, however, with extreme muscular weakness, coldness of surface and cardiac exhaustion, there are indications of nervous derangement and even delirium, with no evidence of vascular cerebral excitement; the pupils are not contracted, and the skin remains cold and clammy; the urine is copious and pale, and, in short, there has ensued a condition which, in my experience, is almost pathognomonic when taken in connection with the previous history. Patients were not rarely brought to hospital in this state.

I have many times verified the statement that the blood parasite suddenly disappears from the blood at, or just prior to, the critical termination of fever.

The aspect of the patient in severe cases is one of sheer exhaustion; the features are shrunken and wan, and the voice feeble almost as in cholera. Crisis by diarrhoea was extremely rare, yet the fall was then pronounced; by hæmorrhages alone, it was never witnessed.

Commonly with cessation of fever and repose, a prompt reaction takes place, and the normal temperature is regained within the next twenty-four hours, or somewhat more slowly. The symptoms at defervescence by lysis will be presently described; they are usually severe, as seen at late stages of 'continued' fever proper.

Amongst the less known symptoms belonging to the late epidemic in Bombay was the occasional occurrence of a scanty but distinct eruption of pink spots, supervening usually at the close of the attack, and seated on the chest, abdomen and arms. The spots are small, and either quickly subside, or, darkening in hue, they present a staining of the skin with hæmatin, or they become distinctly hæmorrhagic and petechial, and then last for a week or ten days; successive crops may be noticed for two or three days, but not longer, and their outbreak may be deferred to the relapse. This eruption was not associated always with one type of the fever; it is not peculiar.

In general it might be said that two classes of cases were intermingled during the epidemic; one, namely, displaying a comparatively sthenic type, but, to judge from descriptions, never equal to the European standard in robustness; and another evincing rather an approach to typhus fever.

The preceding description applies to a well-defined series of phenomena beginning in health and ending in convalescence, and comprising, it may be, the entire attack of illness. There is no apparent difference in origin, symptoms, intensity, and duration between the single manifestation and the first of a recurrent series; and when death

supervenes it generally takes place in connection with this invasion period.

As to the forecast of a recurrence of the attack, I am unable to mention any conditions which would assist in this. From presumably the same source of infection, both a single and a recurrent attack may arise in different individuals (*e.g.* as happened in two servants attached to the fever ward of the G. T. Hospital); yet I have noticed that when a patient undergoes a second infection after a long interval, the form of fever will be the same on each occasion, and also that affected members of the same family will often show attacks of a similar form.

Supposing, as happened in about three-fourths of all patients surviving, that a 'recurrence,' or the so-called 'relapse,' takes place, I proceed briefly to describe the order of events; and it may be understood that one description applies to the first few days after the invasion-attack, whether the fever reappears or not.

First Apyretic Interval.—In the great majority of ordinary cases, which are also the milder, the predominant feature of this period is debility. This state unquestionably results from the previous febrile excitement, but there is not such a close correspondence between the two conditions as might, upon general grounds, be anticipated; and it is a remarkable feature of the spirillum fever that reaction and convalescence should often be so prompt and so early. Although he had been suffering from high fever for a week, yet the patient, if tolerably robust, may revert to his normal condition almost or quite as quickly as if he had undergone simply an attack of ague; it is true he will have lost more flesh, but with the crisis the disease has been as emphatically discarded. This is not the case with some other specific pyrexias of high intensity and brief duration.

Temperature and Pulse.—Commonly after the first day or two following the crisis, there commences a gradual rise of body heat to the normal level, which is attained just before the onset of the relapse, or somewhat earlier. Even when no recurrence takes place, the temperature is restored in the same gradual manner; and sometimes I have then noted at about the eighth day, a slight and brief perturbation (corresponding to a latent relapse) prior to the temperature becoming fixedly normal.

The pulse, on the other hand, not at the crisis wholly subsiding, gradually descends to the normal frequency, or below this, by about the eighth day; and there then may occur either a slight rise, as of temperature, with no further sign in the abortive cases, or a great and sudden rise, which appertains to the true relapse.

The usual daily ranges of pulse and temperature are, as a rule, maintained throughout; hence this non-spirillar interval is a quasi-normal state.

A remarkable variation, however, occurs which happened as often as one in six of all cases, could not be foretold, and commonly was not indicative of serious consequences. I refer to a sudden rise of temperature supervening promptly after the crisis, and sometimes so pronounced as to simulate a return of the attack. This event occurs equally often after the second attack (first relapse), and I have been accustomed to term it the 'rebound,' or 'secondary' fever. Its duration is brief, and the blood spirillum is invariably absent. The general symptoms

are those of fever, but less marked and peculiar than in the specific attack.

Other symptoms.—The following have been noted in marked cases : Headache or heaviness, much thirst, aches, moist skin, and much debility for a time; a tendency to dryness of the tongue and a brownish fur with florid sides and tip ; the bowels still costive, but appetite speedily regained, and sometimes good or even voracious at the beginning of this stage.

Sleeplessness is apt to continue, and in even mild cases slight delirium was not uncommon, now sometimes first appearing ; and this last remark is true of jaundice. Diarrhoea and vomiting have been seen just after the crisis. Tenderness and enlargement of the liver speedily subside, ceasing altogether in a few days. Splenic implication also promptly diminishes, but the size of this organ is usually not so soon reduced, and prior to the relapse it may again become enlarged. Pink spots were sometimes first seen a day or two after the crisis, passing into true petechial marks when associated with persistent typhus symptoms. Cough (mild bronchitis) is a symptom that may now first appear, or become more troublesome.

In a few days the headache and thirst quite subside ; the aching pains of the back and limbs (chiefly the lower, and much increased on exertion) may persist, and be the cause of much distress. There are temporary sweats, not said to be preceded by feverishness. The stools are bilious, and often unusually so ; deafness and neuralgia. The condition of the urine was not found to be abnormal in the great majority of cases ; once in twenty it contained on the second day a little albumen : mean specific gravity, 1015 ; this was temporary, and renal casts were never present ; generally the urine is rather copious, pale, and of low density. Oedema of the feet (without albuminuria) is sometimes noted in the first part of this interval.

In most hospital patients convalescence was gradual, in even the milder cases ; muscular and cardiac debility lasted longest, with deep-seated aches ; jaundice and cough but slowly subsided. Emaciation was sometimes pronounced, even after a single attack. Anæmia was not a usual sequela, nor were pre-existing local affections notably increased by the fever. Irregular aguish attacks seemed to be occasionally re-excited. These points will be discussed hereafter, with complications likely to arise at this stage.

When a relapse supervenes, this apyretic stage comes to its end in about seven days.

Second Attack: First 'Recurrence' or 'Relapse.'—This is a brief and variable repetition of the first attack ; and though for the time pronounced, its general severity, and still more that of succeeding relapses, is decidedly reduced.

As compared with the invasion, in fully developed first relapses the maximum temperature, particularly towards the close of the attack, is somewhat higher, and the pulse almost equally exceeds in frequency ; the common symptoms, generally speaking, are less marked, but they are characterised by greater debility. Much, however, depends upon the intensity of the relapse, which, contrary to the tolerably fixed character

of the invasion, is a very uncertain datum,—there being seen every grade of febrile disturbance from a slight rise of heat to a continuous paroxysm, almost equal to the first.

That the second attack is a curtailed copy of the first, is shown by its frequent resemblances as displayed in the temperature chart, with respect to deviations in course and general character, which were sometimes even faithfully reproduced; and there are no clinical features in the *replica*, which can be termed strictly new. It will, therefore, be sufficient to allude to the modifications of symptoms presented.

Prodromata.—It is well worthy of notice that the relapse not rarely sets in quite abruptly, and without any precedent symptoms apparent to ordinary observation. On many occasions the patient has expressed himself as continuing to improve, or even better than usual, until within a very few hours of the attack. As frequently, however, there was some headache or pains in the body, or there were distinct chills, indicating a preliminary paroxysm. Some more unusual signs will be mentioned below. The attack usually supervenes after noon, sometimes in the night, rarely in the morning.

Pyrexia.—As contrasted with the invasion, here the initial rise of temperature is not quite so abrupt and high (mean of first day, 102°F.); but before long the body-heat may attain a higher elevation than it had in the first attack, and a similar exacerbation may take place just before the crisis; there being often a depression of mean temperature before or at the middle of the attack, which may proceed so far as to become a cleft in the chart. The pulse seems commonly to attain its maximum (mean 118 per minute) more slowly and gradually. In all cases there occurs the usual morning decline and evening elevation of temperature, the mean daily range being greater than in the invasion, but only occasionally exceeding 2°F. ; and the pulse oscillates similarly, but somewhat less in proportion.

In selected cases under analysis, the pyrexia continued in the mean for about five days; never exceeding seven, and being generally briefer.

The crisis is equally abrupt as at first, being accomplished in a few hours, or slowly prolonged at the end; the mean fall was $7^{\circ}\cdot5$, the maximum $11^{\circ}\cdot8$, the absolute lowest temperature noted being $94^{\circ}\cdot8$; the normal line of $98^{\circ}\cdot5\text{ F.}$ was always reached, but very seldom did the fall cease there. Generally the crisis occurs during the night or early morning.

A brief 'rebound' not unusually now takes place, immediately after the crisis; the patient then rallies, and the second apyretic interval begins, which much oftener than not passes into permanent convalescence.

Other symptoms.—The initiatory chills of the first day may reappear on the second, indicating an exacerbation of the fever, which is not uncommon during the earlier part of the relapse. Perhaps the most prominent symptom is marked headache, most severe about the second day, and again after the middle, but less so towards the end. Thirst, often urgent, and early complained of, increases throughout. Dryness of the fauces was noted, and the tongue becomes rapidly coated with a white creamy covering, which afterwards turns to brownish; it is moist at first, and sometimes dryish and roughened at the close, the edge and

tip acquiring a florid hue and its volume shrinking. The skin remains dry, except during decided remissions, and when the critical fall is at hand ; it sometimes becomes pungently hot and harsh. Sleeplessness is a common symptom at the height of the attack, and some delirium at night may appear with or after a remission towards the close, but it is not common. Aching pains, generally distributed, increase in severity from first to last, and are very seldom absent ; they are much complained of, and are referred to the back, loins, larger joints and lower limbs, including both muscles and joints, and exacerbating with the pyrexia. The abdominal fulness, tension and tenderness, chiefly in the upper zone, may appear early but are most marked towards the end of the attack ; and the same is the case with splenic enlargement, which, although more frequent than at the invasion, is not always so considerable as to be detected without special care. The liver is less often enlarged, though even early there may be decided tenderness (elicited in movement or pressure), which also augments with the fever ; jaundice was rare, but a third of the prolonged cases showed it at last. Bilious vomiting, on the other hand, was rather commoner at first, and with the mid-exacerbation ; it may be attended with hiccup ; nausea is an entry at the time of final exacerbation. The appetite for solid food was commonly impaired, yet there were curious exceptions. Deafness is an entry coincident with midway delirium, and also epistaxis.

The urine is commonly plentiful and of density rather below the mean, until near the close of the attack, or with a mid-fall ; it was usually acid in reaction, and when examined, always in this series free of albumen or organised sediment. The bowels were costive, especially at first ; the stools were always bilious ; diarrhoea might supervene.

In general the symptoms are modified by daily changes, and by less regular movements of pyrexia, which may lead to a pseudo-crisis in the midst of the attack ; a tendency existing to remit, or even towards intermission, not observed in the first attack.

The blood-spirillum is always present, and may abound ; it precedes the beginning of the relapse, and disappears immediately before its close.

Second Non-Febrile Interval.—This stage in the interrupted sequences of the spirillum fever, becomes defined only in the event of a third attack, or second recurrence or 'relapse,' which was seldom seen in hospital practice.

But even when no distinct relapse occurs, sometimes a perturbation of bodily heat about the fifth or six day appears ; there being at the same time some signs of constitutional disturbance.

When the relapse takes place, this second interval commonly proves longer than the first, its mean duration being ten days. Rallying after the crisis of the preceding relapse is usually prompt, and unattended by the rebound ; the temperature and the pulse speedily regain the normal ; the general symptoms are mild, and chiefly point to debility ; headache seldom persists after the first day, but aching pains in the loins and lower limbs, back and upper limbs, are common, and may last for a time ; the tongue is dryish, and brown only at first, and in attacks of a low type ; the skin supple, and the state of the bowels varies, yet

constipation is not obstinate ; nausea and vomiting are rare, and the appetite soon returns. The spleen subsides, and so hepatic signs ; but jaundice may persist, and even now for the first time appear. The delirium of the second febrile stage seldom extends to this interval (one in eight cases), then being limited to the night-time, and declining in a day or two ; the patient sleeps. Cough may last, or now first come on, as was noted of the first apyretic interval. Commonly the patient continues to improve until the very day, perhaps hour, of succeeding relapse. When there happens a decided complication—feverishness, diarrhoea, or bronchitis will interrupt this level course ; but they were rare in the ordinary cases under review.

Second Relapse.—A third attack of the genuine spirillar type is not commonly seen, though some quasi-periodic febrile disturbance is apt to succeed the first relapse ; and the aid of the microscope may be needed to ascertain its real character.

Second recurrences differ from the earlier attacks by their briefer duration and less regular form ; they last from two to four or five days, and when shortest may consist of a single prolonged paroxysm extending little over twenty-four hours ; if lasting longer, the pyrexia may be sustained, but its tendency is to remit or intermit, and I have seen an entire day's interval of low temperature intervene (the spirillum being present) between the two or three paroxysms of ague form, which constituted this second relapse ; so marked an interruption was never seen in prior attacks. Owing to such liability of variation, no satisfactory mean type can be elicited ; of temperature and pulse some data are given below. Other symptoms are as follows : commencing abruptly morning or evening, chills or even rigors are not uncommon (which is noteworthy in connection with the intermitting tendency), and in milder degree these may be repeated with succeeding febrile accessions. Headache, thirst, severe aching pains, burning of the eyeballs or sockets, burning of the soles, have been noticed at beginning and end ; the tongue is coated, and usually remains moist, but with continuous and low fever it speedily becomes dry and brownish ; the liver is seldom implicated, the spleen oftener from the second day onward, at least one-half my cases showing a large and tender spleen on the fourth day ; the appetite is suspended, and thirst may be considerable ; the skin is frequently moist in association with remissions and intermissions of the fever ; the bowels may be costive or free ; diarrhoea has been noted at the end. Jaundice reappears here, as during the earlier recurrences, but delirium is unusual, it occurs at the fall ; bilious vomiting may be present in connection with the febrile exacerbations. Some critical perturbation is noticed in prolonged cases, and the fall is marked by sweats, pains, and great debility. Throughout, weakness is a prominent feature, yet there is no character peculiar to this stage, which is at most a mild repetition of the preceding febrile attacks ; in general, the symptoms vary more at morning and evening, according to hours of remission and exacerbation ; the blood-spirilla may abound.

Third Interval.—As applies to non-febrile periods previously named, this apyretic period becomes defined only when terminated by a distinct pyrexial recurrence, here called the third relapse or fourth attack,

reckoning from the first. Since hospital patients would seldom stay long enough under observation, opportunities of seeing this period throughout were not numerous ; its duration was ten to fourteen days.

Commonly the state immediately succeeding to the second relapse is a quasi-normal one, and offers no distinguishing characters, whether or not a third recurrence takes place ; only when this does occur, there are somewhat oftener signs of perturbation (*e.g.* a rise or fall of body heat) about midway of its course. Depression persisting after the last crisis, the body heat may remain subnormal for a few days longer, with a pulse rallying more slowly than the temperature.

The general symptoms are those of a depressed state gradually overcome ; thus, the tongue cleans, and appetite and sleep return ; a glazed tongue has been seen to persist ; the pains subside gradually, and headache is uncommon ; the pulse improves, and the abdominal viscera resume their normal condition. Variations here refer to a sub-latent febrile perturbation occasionally noted about the middle of this period, and manifested by headache, pains, giddiness, feverishness at evening or sweats at night, chills being rare ; the spleen may become enlarged and tender ; depression attends. Other signs were a burning sensation in the soles of the feet, sleeplessness, an irritable tongue, all lasting for a few hours only, but leaving an unfavourable impression on the system ; actual pyrexia may be overlooked. Then follows a return to the state of quasi-health, which continues until the day of relapse.

Third Relapse.—Considering that a third recurrence or fourth attack of spirillum fever, supervenes only after a month or more subsequent to the invasion, it will be readily understood that it was seldom seen amongst hospital patients, and may therefore be commoner than appears from the records. There were six instances entered of this event, but most cases were not seen throughout, and some reliance had to be placed upon the history as given by the patient. These late recurrences occurred mostly during the height of the epidemic and in young subjects ; they were not necessarily preceded by severe attacks, and in themselves were mild, varied and brief, being seldom regarded as more than ephemeral events, entailing only some moderate perturbations of health.

I have hardly a doubt that in the more pronounced of them the blood-spirillum reappears, and might be found upon special scrutiny ; but it happens that no opportunity occurred of applying such special tests, as the Albrecht method and staining process.

Duration of attack one to four days, wholly or in part.

The pyrexia commonly assumes the form of a single paroxysm more or less prolonged ; at the longest remitting, yet still acuminate ; and the temperature may reach 103° to 104° ; when briefer, the rise may be little above the normal ; the fall is comparatively slight, but was clearly indicated in even the mildest cases. The pulse rarely rises much above 100 per minute, and its decline is gradual. Other symptoms recorded are headache always, pains in the lower limbs, coated tongue, loss of appetite, constipation, vomiting at onset (not rare), jaundice, splenic uneasiness, though not hepatic ; brief recurring feverishness without chills or sweats, thirst, sleeplessness, undoubted depression of the system, and considerable perspiration at the end. The relief that follows,

is more decided than might be anticipated from the seemingly mild character of the attack. I may add that the fever-free state which ensues, has been clearly characterised by a subnormal temperature lasting for several days, liable to be marked by occasional brief depressions, and only gradually rising to the normal level ; the instances are too few for further comment.

Fourth Relapse, and Subsequent Recurrences.—There was available in the whole series of relapses seen, only one clear instance of fourth recurrence or fifth febrile attack. It occurred in a youth who in hospital had a well-marked first relapse (invasion attack prior to admission), which was followed by others mild in character and successively briefer, though rather more pronounced, the fifth event being represented by a prominent single paroxysm rising abruptly at evening and culminating the following morning at 104° ; the crisis followed in the evening of the second day, and next morning (third day) the temperature was $97^{\circ}4$, which is rather low for these later series. In this case, the apyretic intervals were level and not extremely long; the patient was discharged five days after the last attack, and nothing further was known of his illness.

Of *Fifth* relapses I have no definite information to offer.

Amongst the few cases of relapsing fever unusually detained in hospital, there might be seen minor derangements of temperature and health which showed a periodic tendency ; and in some general features a resemblance to the brief and abortive attacks regarded as standing in place of first, second, or third relapses. Such detained cases were very few in comparison with those early discharged ; and it is reasonable to suppose that some of the latter would have displayed similar marked perturbations. Cases were also sometimes admitted for debility or diarrhoea, with a history of late recurrent fever, and showing soon after entry two or three isolated paroxysms, which appeared at intervals fairly corresponding to those of known late relapses, and were open to interpretation as fifth or sixth recurrences. Little attention could commonly be given to these brief events, and ordinary microscopic observation commonly failed to detect the blood-parasite. Upon two or three occasions, however, the simpler search was successful during such isolated, ague-like attacks, and thus their character as late specific relapses became clear ; that they were not due to fresh contagion in hospital, I inferred from the fact of no primary attack of spirillum fever in man, ever presenting the form of a dissociated or single daily paroxysm.

General Features and Variations of Relapsing Fever.—Severity of the disease.—Cases are mild or severe according to the intensity of pyrexia, and the presence or absence of local complications ; the influence of personal destitution and starvation was not very apparent, but it could seldom be accurately gauged ; and unquestionably the majority of patients brought to hospital in dying state, bore the signs of extreme indigence. During the febrile stages pyrexia is often excessively high, surpassing the range in other continued fevers ; yet this consideration becomes of less importance in spirillum fever than in typhus, since usually the attacks do not last long enough to thoroughly exhaust the sufferer or induce permanent local lesion ; and the same remark applies to comparison with severe remittents. A liability to complications always

exists. Notwithstanding its pronounced character, this fever is decidedly less fatal than the others just named ; and this feature appears to be due not only to the mild character of the infection, but also to the relief afforded by the apyretic intervals, during which the patient approaches more or less towards convalescence. Were the whole series of two or three febrile periods to be accumulated into a continuous pyrexial state, I doubt not the death-rate of relapsing fever would at once be raised. The extremes of age add to risk.

Many casualties take place in connection with the first attack, from high fever and its immediate attendants ; others at subsequent early date and a few later on, from local complications. I did not learn that pre-existing organic disease is necessarily aggravated, or that a tendency to chronic lesions is often set up by the spirillar infection.

General Course.—Why some attacks of fever should be single (in a practical sense) and others composite, remains unknown : when multiple, the successive events are dissociated except as regards the ensuing feeble state of the patient. The disease is severest at first, and it abates by abbreviation and deferring of the pyrexial recurrences : thus, the attacks which were at first continuous, next assume the remitting and finally the intermittent type. The liability to acute complications also diminishes in order of time. Whilst in general apyretic intervals present the aspect of a quasi-convalescence, yet they are truly incubation-periods of successive illnesses.

Duration.—Regarding an infected state of the body and the disease as one and the same, the following averages are met with :—Duration of an abortive attack of spirillum fever 14 days, of which 7 febrile : the ordinary attack with one relapse lasts 26 days, of which 12 febrile : the rarer attack with two relapses lasts 39 days, of which 15 febrile. Pre-febrile or incubation periods last 7, 7 and 10 days, in all 24 : the successive febrile manifestations occupy 7, 5 and 3 days, in all 15. By common reckoning, the first incubation-period or that of the invasion attack, is not included in the duration of illness ; yet it is as much a part of it as the succeeding pre-febrile stages, which are not so liable to be overlooked. The data on which these statements are founded will be furnished in the following Chapter IV. ; considerable deviations are met with.

Stages.—The correct way of viewing the succession of febrile and non-febrile phenomena which compose the stages of an ordinary illness, would be to regard them not as parts of an indivisible whole, but rather as repetitions (varying in number) of similar clinical elements. An entire attack of spirillum fever may consist of one incubation-stage and one febrile manifestation, a considerable number of cases practically showing no more. The first incubative period is usually disregarded, and only the culminating pyrexia attracts attention : in recurrences the period of latent infection is known as the apyretic interval, and looked on as a stage of the disease, though mostly void of symptoms. As variations the apyretic period may be lengthened (perhaps by the suppression of a relapse) or shortened ; or it may be occupied in part or whole by secondary or superadded febrile phenomena, such as a rebound or fever symptomatic of local lesion, and so may become almost obliterated. There is reason to suppose that both malarious and ordinary continued

fevers sometimes co-exist with spirillum fever, and under ordinary inspection they equally obscure its peculiar features ; but I have invariably found that the true interval is preserved, inasmuch as then the blood-parasite disappears (whatever the temperature), to reappear in due course.

The pyrexial stage itself may, after the first attack, be abbreviated in every degree : it is never lengthened, except in the very rare cases where a redoubled relapse may be suspected. Even in the midst of febrile complications it may with due care be recognised at the appropriate time, through means of its attendant spirillar blood-infection : naturally it tends to become shorter in the relapses, and in any one of them may be so much abbreviated as to be practically non-existent.

The type of the fever has been alluded to as regards the first and succeeding attacks. Only general terms apply here, for after much labour spent in comparing and analysing the ordinary temperature charts of the several periodic events, I have arrived at the conclusion that the spirillum fever is more variable in its course than any of the other so-called continued fevers, and almost as variable as the malarious. The first attack is probably never intermittent ; the second may be so when the series is about to be closed : I have occasionally seen a tendency to repetition of type in the whole series of three attacks, but no rule obtains after the first onset. The change of type at successive febrile manifestations, accords with their progressive abbreviation and mitigation in degree ; and when the order from continued to intermittent is checked or reversed, the indication is one of increasing severity : this is an extremely rare event.

The height of the fever ordinarily bears a relation to its duration, thus the pyrexia is most pronounced, as a whole, at the invasion : it may, however, for a brief period be equally or more elevated in a first recurrence ; if high subsequently, its duration is still more limited : commonly it is not nearly so marked.

Form of Fever.—Practically there are two forms of spirillum fever, namely the abortive and the relapsing ; or, preferably, the single and the repeated. Of recurrences the number varies considerably ; and as in successive attacks the type of fever becomes modified, it may be said that this disease merits the distinction of being elevated into a *genus*, as much as does malarious fever ; having equally a specific cause, and many modifications worthy to be ranked as *species*.

In a strict sense, the spirillar infection probably always reproduces itself once, at least, in the body ; but not always in a palpable manner : when febrile disturbance is excited thereby, the manifestation is apt to vary exceedingly ; it being understood that the last pyrexial attack is always less pronounced than the one preceding. The difference, therefore, between single and repeated attacks is, that in the former there is no evident recurrence ; whilst in the latter the repetitions, one or more, are sufficiently marked to attract attention.

Complications.—These occasional attendants upon the disease may be regarded as either belonging to the febrile stages, or as incidental to them ; probably none being absolutely peculiar. The state of the patient previous to infection is an important item in determining their occurrence, and it was commonly one of debility amongst my hospital

patients; the other leading condition relates to the amount or quality of infection, and nothing is known of this except by its effects. Complications belonging to the fever, may be said to be concerned with the blood or the blood-vessels: of the former little that is definite could be learnt with the microscope alone, excepting as regards the blood-parasite and cell contents, though I feel sure there are other ascertainable conditions of the circulating medium, which would prove of the highest interest: of the latter, there were noted congestions, thrombi, embolisms and infarcts, with resulting hæmorrhages, sloughing and inflammation in different parts of the body. Visceral complications are also of a general character, and occur in all three cavities of the trunk; the more vascular organs probably suffering most, or oftenest. Commonly, however, the attack leaves no permanent lesion.

Sequelæ.—There are none special known, and but few were witnessed: the conditions of observation were not however very favourable.

The above account is in effect an amplification of the Definition first submitted, as regards the clinical history of spirillum fever. Each of the items named will next be discussed in detail, with separate chapters on Mortality, Diagnosis, and Prognosis. Under the heading of Pathology the outward conditions and essential nature of the disease will be treated of. The blood-parasite is described separately.

ILLUSTRATIVE CASES.

Single Febrile Attack.

CASE VIII.—M., 25, servant in a fever-ward, G. T. Hospital. First day—while on duty seized suddenly with fever and chills at 2 P.M., has frontal headache, pains in loins, no marked splenic or hepatic uneasiness, slight cough; t. 104° , p. 140: a few spirilla in the blood. Second day—m. t. 102° , p. 112, much headache, no thirst, hepatic and epigastric tenderness, lumbar pains, one stool; e. t. $103^{\circ} \cdot 6$, p. 120, splenic enlargement and tenderness, pains in limbs: spirilla very few. Third day—m. t. $103^{\circ} \cdot 4$, p. 130, resp. 44, has the typical aspect, no sweats, much headache, slight thirst, tongue white, moist, slight cough, and sputa scanty, abdomen tender and not distended, severe body-pains and cannot sit up; two watery, bilious stools; vomiting last night; spirilla few: e. t. $103^{\circ} \cdot 2$, p. 134, persistence of symptoms. Fourth day—m. t. 103° , p. 130, resp. 36, no sweats, much thirst, pains severe and a sense of distension in the joints of limbs; other symptoms persist; he is oppressed and sinks in his bed: e. t. $102^{\circ} \cdot 4$, p. 152, resp. 40: three stools; no sleep: spirilla several. Fifth day—m. t. 103° , p. 130, resp. 30: the symptoms persist: vomiting last night; he slept from chloral given; skin now moist; three stools: e. t. $103^{\circ} \cdot 8$, p. 136, resp. 44: no worse: spirilla several. Sixth day—m. t. $102^{\circ} \cdot 2$, p. 130, r. 40: cough increased, sputa frothy, no dullness or pain of chest, spleen no more enlarged: e. t. $102^{\circ} \cdot 2$, p. 126, r. 32: no change: spirilla many. Seventh day—crisis—5.30 A.M., t. 99° , p. 94, r. 32: perspiring freely, slight headache, slight thirst, cough and abdominal uneasiness diminished: vomiting in night and he slept a little (chloral repeated): 8 A.M., t. $94^{\circ} \cdot 6$; 4 P.M., t. $95^{\circ} \cdot 6$, p. 100, feeble, r. 34, skin clammy. Eighth day—m. t. 96° , p. 92, r. 32: depression continues, pains in joints, tenderness in lower part of abdomen, one stool; he eats: e. t. 97° , p. 96. Ninth day—m. t. $97^{\circ} \cdot 8$, p. 86, pains increased, no abdominal tenderness: e. t. $98^{\circ} \cdot 4$, p. 90. Convalescence progressed slowly on account of the arthritic pains, but seemed uninterrupted: on the fourteenth day the m. t. was 100° , p. 108, and the signs of pyrexia slightly increased; next day similar and slight chills; again on the following day, when some abdominal tenderness reappeared; after this, return to normal temp.: the blood was examined twice daily from 12th to 17th days inclusive, and without clear positive results, fully-formed spirilla, at least, not being seen; possibly they were overlooked. Pains in the shoulders and some epigastric tenderness persisted

for a fortnight; the man was much reduced in flesh. His fellow-servant was attacked the day before him and suffered more, undergoing a marked relapse; and so another attendant in the same ward.

Fever with One Relapse.

CASE IX.—F., æt. 35. Hindu from the Deccan famine-districts, one month in Bombay; resides in a large house with many countrymen and relatives from the same village, several of whom have been admitted into this hospital with spirillum fever. A small, thin subject, at reputed eighth day of illness. A.M. temp. $104^{\circ}\cdot2$, pulse 132: frontal headache and pains of joints, slight thirst, hepatic and splenic enlargement and tenderness: tongue coated, but moist. P.M. t. 105° , p. 136 full and soft, respirations 44: skin moist; marked tenderness in upper abdominal zone, spleen $2\frac{1}{2}$ inches below cost. cart., liver not enlarged upwards but downwards 2 inches, epigastrium not disturbed, but tender; abdomen moderately full; no tenderness elsewhere: bowels open to-day. Heart's sounds clear, the second being accentuated, even at apex. Some bronchial râles heard in the chest; her breathing is hurried and general aspect distressed. The blood is full of spirilla.

Ninth day of disease.—A.M. t. $104^{\circ}\cdot8$, p. 148. P.M. t. $104^{\circ}\cdot4$, p. 140, resp. 44. Fever slightly less, the acme being passed; no sweating yet; hypochondriac and epigastric tenderness less, abdominal fulness the same; has appetite and some thirst, but dislikes cold water as likely to bring on chills; temporal headache only on coughing; pains in the elbows and knees, without swelling. Heart's sounds weak, but clear; an impulse is perceptible which extends to the xyphoid cartilage and even to the right side of it; second sound accentuated at the base. The liver and spleen are but slightly reduced in size. The night's urine was 13 ozs. high-coloured, quite clear, sp. gr. 1010, acid, albumen $\frac{1}{8}$, chlorides doubtful (on the addition of nitrate of silver the colour becomes deep purple and there is cloudiness, but no deposit on standing for two days): urea, 61·2 grs.: the day's urine 16 ozs., very high-coloured, clear, sp. gr. 1010, albumen none, chlorides doubtful (colour changes to yellow-brown as above), urea 70·4 grs. The blood full of spirilla.

Tenth day.—Crisis. A.M. t. $97^{\circ}\cdot2$, p. 90, resp. 26. Subsidence of fever with copious sweats at 3° A.M.: the distress is gone; no collapse, though she cannot stand from pains in knees and weakness: liver and spleen much less tender and projecting; epigastric tenderness also less: some thirst; skin now dry (7 A.M.): troublesome cough since the fall began. Night's urine, 35 ozs., high-coloured, clouded but without sediment, reaction acid, sp. gr. 1006, albumen a decided trace, chlorides undetermined; urea 123·2 grs. 4 P.M. t. 96° , p. 78, resp. 26; 6 P.M. t. $96^{\circ}\cdot6$, p. 72; had sweats during the day, headache quite subsided, pains in knees and ankles continue; spleen felt $1\frac{1}{2}$ in. below the cost. cart. but not tender; some tenderness on firm pressure over epigastrium, and more over lower edge of liver which reaches to 1 in. below the cost. cart. in nipple line. No exhaustion: heart's systole fairly heard (no murmur) though the pulse is decidedly feeble and small. Day's urine 22 ozs., rather high-coloured, clear, sp. gr. 1010, acid, albumen none, chlorides none, bile-pigment much, urea 96·8 grs.

Eleventh day.—A.M. t. $96^{\circ}\cdot4$, p. 80, resp. 28. P.M. t. $96^{\circ}\cdot6$, p. 84, resp. 34. Looks much pulled down, but is not exhausted; troublesome cough at night; additional pains in shoulders and elbows; no swelling. Night's urine 11 ozs., high-coloured, clouded, no sediment, sp. gr. 1015, chlorides $\frac{1}{12}$, urea 111·3 grs.: day's urine 13 ozs., very pale, rather clouded, no sediment, sp. gr. 1003, acid, chlorides $\frac{1}{18}$ (no tint on addition of nitrate of silver), urea 30·3 grs.

Twelfth day.—A.M. t. $96^{\circ}\cdot4$, p. 78, resp. 28, rallies very slowly, pulse is weaker and first sound of heart less distinct: skin dry; pains in upper limbs more, preventing sleep: no swelling of the stiff and bent shoulders and elbows; extension increases the pain: she is crippled by pains in the lower limbs, which are referred to the upper margin of the patella and insertion of the ligamentum patellæ, and are much increased upon exertion. Appetite good, but still some epigastric tenderness; volume of liver and spleen much reduced, and tenderness less; abdomen flattened. P.M. t. $98^{\circ}\cdot2$, p. 80, resp. 30: chief complaint is pain and stiffness of the right shoulder (the deltoid muscle being stiff and tense) and elbow, so that she cannot move these joints; the hands are quite free; the knees are bent and stiff, and she cannot stand

without assistance. Urine: night, 30 ozs., rather high-coloured and clouded, no sediment, sp. gr. 1006, chlorides $\frac{1}{8}$, urea 158.4 grs.: day, 12 ozs., pale, clear, sp. gr. 1006, chlorides $\frac{1}{8}$, urea about 30 grs.

Thirteenth day.—T. 97°·6, p. 70, resp. 26. Cough less, pains in r. shoulder and the knees also diminished, and she can now walk a little and raise the arm; no wasting visible: the appetite has now become voracious. Spleen not to be felt below the cost. cart., it is probably measurable but the parts are now very lax and the organ very mobile: liver has resumed its normal dimensions; some epigastric tenderness remains, no pain after eating, no vomiting. Pulse has improved, and heart's first sound is better heard; it is of rather booming character over mid-cardiac region. P.M. t. 98°·4, p. 72, resp. 26, the pains are less than in the morning. Urine: night, 40 ozs., pale and tolerably clear, sp. gr. 1004, acid, chlorides $\frac{1}{8}$: day, 12 ozs., pale, clear, 1006, chlorides $\frac{1}{8}$; total urea 102.9 grs.

Fourteenth day.—A.M. t. 97°·6, p. 64, resp. 26. Some cough again, pains no worse, still some epigastric tenderness on firm pressure but no tumefaction; pulse is very feeble, though the heart's impulse is perceptible. P.M. t. 98°, p. 64. Urine: night, 18 ozs., pale, clear, 1006, chlorides $\frac{1}{8}$: day, 16 ozs., pale, acid, 1004, chlorides $\frac{1}{8}$: combined urines, urea, 89.7 grs.

Fifteenth day.—Sixth day of apyretic interval, and two days before the relapse; A.M. t. 97°·4, p. 60, resp. 22: cough at night; pains in r. shoulder and knees felt only when she moves about. Epigastric tenderness less; spleen just felt under the lowest ribs, not tender; heart's action firmer, impulse and first sound distinct, second sound accentuated at base only; pulse moderately firm. P.M. t. 98°, p. 60, resp. 24. Urine: night, 24 ozs., pale, clear, 1004, chlorides $\frac{1}{8}$: day, 22 ozs., pale, tolerably clear, no sediment, sp. gr. 1005, acid, chlorides $\frac{1}{4}$, urea in mixed urines 85.1 grs. Microscopic examinations of blood—first specimen taken at 9 A.M.: the appearances indicate the presence of spirillum: tri-hourly records now begun of temperature, pulse and state of skin and blood.

Sixteenth day.—Day before beginning of the relapse; A.M. t. 98°, p. 60, resp. 22. No pain in the shoulder, and in the knees only at rising; still slight tenderness of epigastrium on firm pressure; cough less; slept: spleen can be felt and its anterior border is probably a little tender: appetite good. P.M. t. 98°·4, p. 58 (the minimum velocity) and small; resp. 24: she moves about the ward free of pain, no increased thirst; heart's sounds feeble, the second pronounced and impulse not felt; liver and epigastrium unchanged, spleen barely felt and the uneasiness which is experienced on pressure seems to be over the costal cartilages at their junction with the 7th and 8th ribs, and not over the spleen itself; no headache, no pains. Weight of body 76 lbs. Urine: night, 36 ozs., pale, clear, acid, 1008, chlorides $\frac{1}{8}$: day, 18 ozs., acid, 1006, chlorides $\frac{1}{16}$, mixed urines, urea 76 grs. Blood examinations: the spirilla are distinct and not very few in some specimens taken.

Seventeenth day.—First day of relapse. A.M. t. 99°·4, p. 70, resp. 24; skin dry, no headache, no thirst, no loss of appetite, no giddiness, no pains in the joints or limbs; no tenderness in epigastrium or hepatic region (no enlargement there) or in the left hypochondrium, though the spleen is somewhat increased in volume since yesterday and is felt 1 in. below the 9th and 10th costal cartilages: second sound of the heart pronounced, the first is attended with an indistinct murmur over the mid-cardiac region, at apex of sternum and to the right side, but not upwards. There is no distress, but she lies down and is timid. Night's urine 24 ozs., pale, clear, no sediment, sp. gr. 1008, acid, albumen none, chlorides $\frac{1}{8}$, urea 73.9 grs. P.M. t. 105°·6, p. 108, rather full, very soft, resp. 40: skin of forehead and chest moist, that of limbs dry: she had chills at 12.30 noon (t. already over 102°) and some sweats (limited as above) at 4.30 P.M.; now (5.15) skin again drying: no pains in limbs; spleen certainly not larger than in the morning (perhaps a little smaller), not at all tender; no hepatic enlargement or tenderness, but the localised spot at apex of sternum still tender on pressure: cardiac impulse less forcible and the first sound barely heard anywhere, no murmur in recumbent posture, but a cooing sound at once heard in sitting posture, impulse then very faint. Thirst a little more, appetite not impaired and has eaten pulse and rice; looks a little harassed; no giddiness on standing and no pains in loins: has drunk only about 8 ozs. of water and yet urine abundant: a little cough. Urine of day: 42 ozs., pale, clear, acid, 1006, albumen none, chlorides $\frac{1}{8}$, urea 100.3 grs. Rather more spirilla in blood.

Eighteenth.—Second day of recurrent fever : A.M. t. $103^{\circ}2$, p. 108, rather firmer and less full, resp. 36. Skin soft : temple headache ; much thirst at night, less now ; no pains in limbs ; some tenderness in epigastrium, not over liver and spleen ; occasional cough ; appetite fair. Tongue pale, little coated, moist, large ; has drunk water freely. Heart's action feeble, a soft murmur heard in mid-cardiac region only (? in muscular substance) not prolonged, second cardiac sound pronounced at apex and base ; r. side of heart over-distended (?) : cough bronchitic. Spleen decidedly enlarged, but not nearly so much so as at close of invasion-attack, moveable and quite free from tenderness on even firm pressure : liver not enlarged downward and reaching hardly so far as costal margin, not at all tender, so far as accessible to pressure : the epigastric tenderness is rather more marked (?), no fulness but some tension of parietes here ; no pain, flatus, or eructation ; no nausea or vomiting after food ; no jaundice : some tenderness at edge of ribs themselves on left side, as noted before. I found the skin dry, shrivelled, an hour later and the patient with a pale, harassed look ; sleeps ; wants meat. Urine of night : 38 ozs., rather pale and clouded, no sediment, acid, 1010, no albumen, chlorides $\frac{1}{2}$, urea 127 grs. 4 P.M. t. $106^{\circ}4$, p. 124, less full and soft ; resp. 46 : skin dry : temple headache ; shooting pains in calves when she attempts to walk, not in joints ; no giddiness ; more thirst but drinks little ; no appetite, a bitter taste in mouth after eating ; no vomiting ; tenderness in umbilical and epigastric regions ; she sweated twice in the day, namely at noon and 3 P.M. : no delirium. Spleen projects considerably, slightly tender ; liver proportionately less enlarged and not tender ; umbilical uneasiness on pressure may be nervous. rest of abdomen free. H.'s second sound more pronounced, impulse feeble and murmur inaudible in recumbent posture ; some distress of countenance ; slight bronchitis. Urine of day : 22 ozs., high-coloured, very clear, no sediment, sp. gr. 1010, acid, albumen none, chlorides $\frac{1}{10}$ (urine again becomes dark brown colour and very turbid on addition of nitrate of silver solution, hence the deposit, also tinted, may not be all chlorides), urea 86.8 grs. Blood—spirilla many and sometimes clustered.

Nineteenth.—Third day of relapse. A.M. t. $105^{\circ}8$, p. 136, soft, small and feeble ; resp. 42 : skin dry ; tongue pale, shrunk, slightly coated and dryish : has diffused headache ; thirst (drank only 8 ozs. of water in night) : giddiness when walking ; no pains of arms, but shooting pains in calves and knees when moving : no vomiting ; no appetite ; griping about the navel ; some tenderness over hepatic, splenic and umbilical regions, and much over the epigastrium ; bowels freely moved once this morning, stool said to be semi-consistent ; gastric uneasiness after eating : slight cough occasionally, no sputa. My notes add—distress, pallor, weakness, eyes yellowish ; heart's action feeble, second sound pronounced, no murmur, no impulse seen : abdomen retracted rather than full ; spleen more projecting, yet hardly tender ; liver not more projecting and not tender ; epigastrium not distended, yet tender ; umbilical termina at brief intervals and some tenderness on pressure ; vomited some bile when cleaning her mouth with her fingers (in native fashion), complains of 'pît' (biliousness) : the hands and feet feel cold, whilst the trunk feels very warm (a thermometer held in the fist stood at $102^{\circ}6$) : some moisture of skin in the early morning ; now dryness and shrinking of integument. Urine of night, 20 ozs., high-coloured, cloudy, no sediment, acid, 1010, no albumen, chlorides $\frac{1}{2}$ (dark purplish-brown tint as before) ; urea 88 grs. Great numbers of spirilla in blood.

4 P.M. t. $106^{\circ}2$, p. 136, resp. 44 ; skin moist, had sweats at 1 P.M., and again now, about the head, trunk and upper extremities ; much frontal and temporal headache ; nausea, but no vomiting ; no giddiness ; pains in knees and calves on movement only ; three stools, semi-consistent ; no griping, but tenderness in epigastrium ; hepatic tenderness, not splenic ; little appetite, everything except water and meat causing a bitter taste ; feels as if the belly were puffed, whereas it is neither distended nor flaccid : has thirst but is unwilling to rise to drink, has taken only 4 ozs. of water with meal ; tongue pale, dryish, but almost clean and not shrunken : the patient looks haggard, thin and distressed ; no delirium, no dreams ; cough not increased. Upon measurement the left lobe of the liver was found to be more enlarged and tender ; whilst the spleen had not increased in volume. Urine of day : 11 ozs. only, high-coloured, rather cloudy, no sediment, sp. gr. 1012, acid, no albumen, chlorides $\frac{1}{2}$ (colour as before) ; urea 65.3 grs. Much bile-pigment. Temp. of hand $104^{\circ}6$. Blood—many spirilla, but seemingly fewer with this exacerbation.

20th day of disease, 4th of this relapse.—A.M. t. $104^{\circ}8$, p. 126, very small and

feeble, respiration 42. Skin dry: tongue pale, little coated, not shrunken, dryish; slept with the chloral: has much frontal and temple headache, no giddiness yet cannot walk from muscular debility and trepidation; heart's action feeble, no murmur or impulse, second sound all over pronounced. Spontaneous vomiting last night, contents of stomach ejected, no blood; nausea preceded vomiting; no history of fever-crisis there. Two stools: feculent and yellow: no griping; hepatic, umbilical and especially epigastric tenderness considerable, no splenic tenderness though the viscus is larger than yesterday; liver rather larger and particularly the left lobe; no abdominal distension, but some rigidity over epigastrium due to muscular tension and, possibly, to presence of left hepatic lobe; no right iliac gurgling. (Memo. on the case.—The gastric and duodenal tenderness are here marked, the latter being felt on deep pressure only; the ileum and colon being more superficially placed, would be more readily reached). Pains in calves and knees on movement; no appetite and aversion to meat food which she craved for two days since; much thirst but dare not drink water freely (8 ozs. taken) lest the belly should become more distended: eyes yellowish, pupils dilated; facies less harassed (she had slept last night); no delirium: more cough, bronchitic, sputa copious, fine frothy, no dullness of chest anywhere. Urine of night: 16 ozs., high-coloured, clouded, no sediment, sp. gr. 1010, acid, no albumen, chlorides $\frac{1}{20}$ (deep purple, clouded hue); urea 105.6 grs. Temp. of hand said to be 98° only, or 6°·8 less than the axillary. Blood—spirilla many.

4 P.M. t. 105°·6, p. 138, resp. 40; skin dry; tongue coated and dry; no headache or vomiting; much thirst; one pale, scanty yet feculent stool; hepatic and epigastric uneasiness, not umbilical; pains in calves and knees as before; no giddiness; has appetite, but food distasteful and in spite of thirst she refrains from drinking cold water, lest it should cause chills and toothache. There was slight sweating at 1 P.M. on the upper part of the body. Being very low, 2 ozs. of rum were administered which induced some rallying: spleen very tense, yet only a little tender on firm pressure, liver less full, abdomen more retracted. Urine of day: 14 ozs., high-coloured, cloudy, acid, 1012, albumen $\frac{1}{12}$, chlorides not certain (a dark grey, scanty deposit, from the dark green, opaque solution, on addition of nitrate of silver), urea 102.8 grs.: a scanty sediment furnishing no definite structures.

21st, fifth and last day of relapse.—T. 105°, p. 132, feeble, running; resp. 34: skin dry, lips pale, tongue tends to dryness, a little coated; some sweats at 1 A.M.; slept well (chloral), no dreams; much thirst, drank luke-warm water about 15 ozs. in the night; no headache, or giddiness or vomiting; no stool; pain on pressure over hepatic, epigastric, and inner half of splenic regions; abdomen not retracted, visceral enlargement not more and spleen rather less tense; left lobe of liver, over the stomach, is tender. Pains in knees and calves, and also in shoulders and elbows, so that cannot lift right arm without help of the other. No appetite. Heart's action seems more excited, yet feebler; second sound pronounced, first sound attended with a murmur in mid-cardiac region only: both sounds heard at apex of sternum and to the right of it; murmur not more distinct there.

There is a scanty eruption of small, pink, raised spots on the left arm; some seem not quite recent, others may be of earlier date. There is no sign of the typhoid state, but rather pallor and weakness.

Urine of night: 12 ozs., very high-coloured (reddish), clear, no sediment; acid, sp. gr. 1013, albumen doubtful (cloudiness only), chlorides doubtful (a scanty deposit in the dark green solution), urea 88.1 grs. Microscopic examination, negative results.

Blood—many spirilla, and some large, nucleated cells.

3 P.M. Severe rigors coming on, t. 108°·6, p. 150; very copious sweating followed half-an-hour after the chills; frontal and occipital headache came on with the rigor and she was much agitated, not recognising 'day and darkness:' t. became dry and it is stated that the spleen at the same time was more swollen and tender. This was doubtless a genuine *perturbatio critica*.

4 P.M. t. 104°·2, p. 150, resp. 40: skin dry, no giddiness or vomiting; no stool, abdomen full; pains of limbs the same; hepatic, epigastric and splenic tenderness remain; also thirst, has drunk 8 ozs. of water.

6 P.M. The excitement has passed away, skin dry and hot (an hour later 103°·2), heart's action moderated, p. 128 (an hour after 116), soft, first sound free from

murmur; spleen larger and still tender, epigastrium very tender and the liver also to some extent, but there is little change here since morning: the woman has acute pain shooting from the front to the back of the head, body aches, but she is not more distressed or depressed than earlier in the day: no signs of the crisis yet.

Urine of the day 16 ozs., very high-coloured, clouded, no sediment, acid, 1013, albumen a trace?, chlorides not known (colour changed to light brown with cloudiness only), urea 124.6 grs. Blood, 4 P.M.—The spirillum has disappeared since 1 P.M., when the numbers were already declining: many large, nucleated cells are present.

22nd day, end of relapse by critical fall.—A.M. t. 96°·6, p. 84, feeble, resp. 28; skin now dry; tongue coated, dryish; slight diffused headache; some pain on movement in right shoulder and elbow; knees and calves still painful on movement: hepatic, epigastric (especially) and splenic tenderness: no appetite: thirstless, drank about 4 ozs. of tepid water in the night; cough troublesome in the night, sputum scanty, frothy; slept fairly (no chloral), and probably wandered a little, being under the impression of being beaten: no active delirium: no stool for two days. Her face is pale and pinched; no collapse, yet she cannot stand from weakness and pains in lower limbs; heart's action much feebler, no impulse felt and even the second sound is faint. Spleen is one inch less than yesterday, yet still prominent and a little tender; liver less and not tender; epigastrium not distended and still tender: the abdomen is moderately full: jaundiced aspect less. The main descent of temperature amounted to 8°, and the reduction of pulse-rate to 66, as shown in the chart; there were no sweats in the night, but only some moistness of the skin at 1 A.M. (report).

Urine of night: 12 ozs., very pale, clear, no sediment, sp. gr. 1003, acid, albumen none, chlorides none? (on addition of nitrate of silver solution a deep brown tint, but no cloudiness on standing); urea 23.7 grs.

Blood—no trace of the parasite: a few large pale cells.

4 P.M. t. 97°, p. 82, small and feeble; resp. 28; skin moist; tongue pale, coated, dryish; no headache; is deaf in both ears since morning; no giddiness; pupils dilated; no vomiting; pains of r. arm and lower limbs less, abdomen somewhat distended; still some hepatic and splenic tenderness, and more epigastric; one stool of natural amount and consistence, very pale hue; slept in the middle of the day; free sweats at 2 P.M., and again at 4: no thirst; cough the same. Dreamt in her sleep, but is not delirious; she is too weak to stand and can only sit up. On being weighed in a similar dress this evening, it was found that she had lost 10 lbs. avoird. since the 16th day, or more than $\frac{1}{3}$ of body-weight during this relapse not yet quite finished, her present weight being 66 lbs. Urine of day 19 ozs., high-coloured, clouded, no sediment, acid, 1013, no albumen (or cloudiness only), chlorides $\frac{2}{5}$ (colour of mixture light brown but clear and the deposit of chloride of silver large and well defined), bile-pigment much; urea 156.3 grs.; the aspect of the urine had become febrile again, a great contrast to its morning state; the woman had drunk only 4 ozs. of water during the day.

23rd, second day of critical fall.—A.M. t. 96°, p. 80 feeble, resp. 24: skin dry, tongue pale, moist, coated in middle; slept well, dreamt once; there were copious and general sweats at 10 P.M. (t. sank to 95°·8, *vide* chart), and twice afterwards; one stool scanty, natural, contained a lumbricus; appetite good and eats heartily: deafness still, and sense of heaviness in the head, no giddiness, pupils contracted, conjunctivæ still yellowish; some tenderness remains in the upper abdominal zone, particularly in epigastrium; the spleen is now 2 in. beyond the costal cartilages instead of 4 in. and not so hard as before; has still pains in knees and calves, less so in the arm: cough not increased. Heart's sounds very faint, even the second. She drank 10 ozs. of tepid water in the night. Urine of night 15 ozs., very high-coloured, clear, acid, 1013, albumen none, chlorides $\frac{1}{2}$.

4 P.M. t. 97°·6, p. 86, resp. 26: skin moist, tongue pale, repeated perspirations in the day, is slowly rallying, being able to move off the bed: complains most of pains in the lower limbs and weakness: the deafness remains and some local abdominal tenderness; the spleen has gone down $\frac{1}{2}$ in. since morning: had hiccup and gaping, before and after taking food; no pain after eating, or nausea, but only some over-fulness of the stomach felt: a natural stool, probably containing some of the urine. Urine of day 11 ozs., high-coloured, quite clear, chlorides $\frac{1}{2}$: total urea of mixed urines 226.5 grs.

24th day, prolonged crisis.—A.M. t. $96^{\circ}4$, p. 74, resp. 24, skin dry, tongue coated, increased cough at night preventing sleep; free sweating at 10 P.M., has temple headache, no giddiness, left ear still deaf, hiccup and one stool in the night, abdominal uneasiness subsiding, still some epigastric tenderness not fulness or tension; spleen at $\frac{3}{4}$ in. and softer; liver nearly normal. Heart's sounds clear and stronger, the second still much predominating: stool solid and yellow, eyes still tinged; she can now walk a few yards and has gained 1 lb. in weight (67 lbs. body weight).

Urine of night 20 ozs. (probably some passed with stool), pale, clear, no sediment, sp. gr. 1006, acid, albumen none, chlorides $\frac{1}{8}$, urea 63.3 grs.: this reversion to the character of urine at main crisis was concurrent with a slight decline of temperature last night. P.M. t. 98° , p. 80, resp. 26, skin dry: no sweating; pains in r. shoulder and knees the same, no pain in the calves; some epigastric tenderness; the spleen is now at the edge of the thorax: she is hungry yet has no taste for food, is languid and indisposed for exertion; the rallying is slow and intermittent.

Urine of day 14 ozs., high-coloured, clouded, sediment?, acid, 1012, albumen none, chlorides near $\frac{1}{4}$ (colour pale and precipitate defined): urea 88.7 grs.

25th. A.M. t. $96^{\circ}6$, p. 68, feeble, resp. 22, skin dry, tongue coated at root, slept well, sweating in the night on head and trunk, thirst none, yet she has drunk 15 ozs. of water, there being a slight rise of t. at 10 P.M., no headache, pains in knees less, cough less, still epigastric tenderness, one natural stool; second sound of the heart pronounced, yet feeble. She is very weak and pallid, yet not giddy, and can walk with help. Urine of day 44 ozs., pale, clear, 1006, acid, chlorides $\frac{1}{8}$.

P.M. t. $98^{\circ}2$, p. 76, resp. 24, no sweats, knees still painful and stomach rather tender. Urine of day 20 ozs., pale, clear, 1004, acid, chlorides $\frac{1}{8}$: total urea of mixed urines 197.1 grs.

26th. A.M. t. $97^{\circ}8$, p. 70, better volume, resp. 24, tongue clean, no sweats in night, no deafness, some pains in r. knee when walking, cough this morning; heart's sounds distinct, the first now heard, but second predominating; spleen reduced so as to impart only a sensation of fulness and resistance in left hypochondrium: liver normal: some epigastric tenderness remains: appetite improving but she does not drink as much as in health: weight of body 67 lbs., or not more than that of two days ago. Urine of night 34 ozs., pale, clear, 1006, acid, chlorides $\frac{1}{8}$.

4 P.M. t. $98^{\circ}8$, p. 76, resp. 24: drank 8 or 10 ozs. of water, appetite fair.

Urine of day 18 ozs., pale, clear, 1006, acid, chlorides $\frac{1}{8}$: urea of mixed urines 130.4 grs.

27th. A.M. t. $97^{\circ}4$, p. 70, resp. 22, some cough this morning, some sweats at night, no pains in knees, still epigastric tenderness; appetite good, not voracious; stools brown and consistent.

Urine of night 28 ozs., pale, clear, 1008, acid, chlorides $\frac{1}{8}$, urea 104.7 grs.

P.M. t. $98^{\circ}8$, p. 74, resp. 24, skin moist, still epigastric uneasiness.

Urine of day 36 ozs., pale, clear, 1004, acid, chlorides $\frac{1}{16}$, urea 50.7 grs.

28th. A.M. t. $97^{\circ}8$, p. 62, resp. 22, no fresh symptoms. Urine of night 39 ozs., pale, clouded, 1008, acid, chlorides $\frac{1}{16}$.

P.M. t. $98^{\circ}8$, p. 68, resp. 24, still some epigastric uneasiness on firm pressure.

Urine of day 33 ozs., very pale, clear, 1004, neutral, chlorides $\frac{1}{16}$; combined urea 183.7 grs.

29th. A.M. t. $97^{\circ}4$, p. 76, slight sweats in night; urine of night 49 ozs., pale, clouded, and ammoniacal from early decomposition (microscopic examination showed no abnormal particles), 1007, albumen none, chlorides $\frac{1}{16}$.

P.M. $98^{\circ}4$, p. 68, re-p. 24; still slight epigastric tenderness. Urine of day 31 ozs., pale, clear, 1007, chlorides $\frac{1}{16}$, urea of mixed urines 158.4 grs.

30th. A.M. t. $98^{\circ}4$, p. 60, still feeble and small, resp. 22; improvement gradual, the spleen is barely to be felt on manipulation upon turning the patient on her r. side.

Urine of night 29 ozs., acid, 1007, chlorides $\frac{1}{16}$.

P.M. t. $98^{\circ}8$, p. 64, resp. 24, no splenic uneasiness and hardly any epigastric.

Urine of day 34 ozs., pale, clear, 1006, acid, chlorides $\frac{1}{16}$, mixed urea 110.8 grs.

Blood—contains no abnormal ingredient.

31st. A.M. t. 98, p. 62, resp. 22, no headache or pains, no spleen, or liver, change, stools natural; no signs of relapse. Pulse at 4 A.M. only 58; sp. gr. of urine risen. Weight of body 68 lbs., a gain of 2 lbs. only since the fall.

Urine of night 44 ozs., pale, clear, sp. gr. 1010, acid, no albumen, chlorides $\frac{1}{10}$.
 P.M. t. 98°·8, p. 60, resp. 22, spleen distinctly felt when patient turns on right side, not tender: no other sign of relapse. Urine of day 52 ozs., pale, clear, 1003, neutral, chlorides $\frac{1}{10}$, mixed ureas 157·6 grs.

32nd. A.M. t. 97°, p. 60, resp. 22; the spleen is the same, not tender. Urine of night 33 ozs., pale, tolerably clear, acid, 1010, chlorides $\frac{1}{5}$.

4 P.M. t. 98°·4, p. 56, resp. 24; free sweating at 3.30 P.M., no epigastric uneasiness.

Urine of day 39 ozs., pale, clear, 1004, slightly acid, chlorides $\frac{1}{10}$, combined ureas 183·7 grs.

33rd. A.M. t. 98°·6, p. 54, resp. 20, spleen just perceptible, not tender, no pains anywhere.

Urine of night 47 ozs., pale, clear, acid, 1006, chlorides $\frac{1}{5}$.

P.M. t. 98°·6, p. 56, resp. 22, no complaint.

Urine of day 37 ozs., clear, pale, acid, 1008, chlorides $\frac{1}{10}$, urea of mixed urines 166·3 grs.

34th. A.M. t. 97°·8, p. 54, resp. 24, no abnormal symptoms.

Urine of night 51 ozs., clear, pale, acid, 1006, chlorides $\frac{1}{15}$. Second relapse indicated.

P.M. t. 99°·2, p. 58, resp. 24, skin dry; tongue clean, the spleen is not changed.

Urine of day 40 ozs., pale, clear, 1006, acid reaction, chlorides $\frac{1}{10}$, mixed urea 228·2 grs.

35th. A.M. t. 98°·2, p. 56, resp. 24, free sweating in the night, no discomfort of any kind, did not drink more liquid than usual (36 ozs.) Body weight 74 lbs., a gain of 6 lbs. Urine of night 66 ozs., pale, clear, neutral reaction, 1004, chlorides $\frac{1}{12}$.

P.M. t. 99°·6, p. 60, resp. 24, spleen unaffected.

Urine of day 21 ozs., very pale, clear, acid, 1005, chlorides $\frac{1}{10}$, combined urea 153·1 grs.

36th. A.M. t. 98°·6, p. 62, resp. 24, some sweats in the night, spleen not enlarged.

Urine of night 47 ozs., very pale, clear, acid, 1003, chlorides $\frac{1}{10}$, urea 82·3 grs.

P.M. t. 100°, p. 66, resp. 26, some sweating at 3.30 P.M., no headache, tongue coated at the root; spleen not enlarged downwards.

Urine of day 36 ozs., pale, clear, acid, 1008, chlorides $\frac{1}{12}$, urea 60·1 grs.

37th. A.M. t. 99°, p. 54, resp. 24, some sweating at midnight, complains of aching in the back on both sides the spine, spleen unchanged.

Urine of night 52 ozs., pale, rather clouded, 1004, neutral, chlorides $\frac{1}{15}$.

P.M. t. 100°·2, p. 68, resp. 26, looks a little pulled down and weak, yet body weight 79 lbs., and therefore still gains, no enlargement of spleen.

Urine of day 35 ozs., pale, clear, acid, 1008, chlorides $\frac{1}{5}$, urea of combined urines 172·2 grs.

38th. A.M. t. 100°·4, p. 72, resp. 26, slept well, no headache, thirst, giddiness, or pains in limbs, the aching in the back felt only in the neck now, slight epigastric tenderness only, the spleen is distinctly enlarged and firm. She is feverish at day-time as well as night.

Urine of night 50 ozs., pale, clear, acid, 1006, chlorides $\frac{1}{15}$.

P.M. t. 103°·6, p. 88, resp. 32, skin dry, tongue moist, coated at the back, some headache, had chills at 1 P.M., pains in elbows on moving, no giddiness, or thirst; still some epigastric uneasiness only, spleen enlarged $\frac{1}{2}$ inch downwards, not tender, appetite good.

Urine of day 37 ozs., pale, clear, acid, 1006, chlorides $\frac{1}{10}$, urea in united urines 172·2 grs.

Blood—examined each of the last three days without decisive results, but it is possible that immature spirilla were present during this mild second relapse.

39th. A.M. t. 98°, p. 72, resp. 24, general perspiration in the night (except soles of feet), and great relief with decline of temperature, spleen still felt, no abdominal uneasiness.

Urine of night 42 ozs., pale, clear, acid, 1006, chlorides $\frac{1}{10}$.

P.M. t. 99°·2, p. 80, resp. 28, skin dry.

Urine of day 30 ozs., normal aspect, acid, 1006, albumen none, chlorides $\frac{1}{16}$, combined urea 177.4 grs.

40th. A.M. t. $98^{\circ}2$, p. 64, resp. 24, no general or local uneasiness. Urine of night 44 ozs., pale, clear, acid, 1006, chlorides $\frac{1}{16}$, urea 129.7 grs.

P.M. t. $98^{\circ}6$, p. 66, resp. 22, skin dry.

41st. A.M. t. 98° , p. 64, resp. 22, no abnormal symptoms. P.M. t. $98^{\circ}8$, p. 64.

42nd. A.M. t. $98^{\circ}4$, p. 68, improving. P.M. t. $99^{\circ}4$, p. 70.

43rd. A.M. t. $98^{\circ}4$, p. 66, body weight 84 lbs. P.M. t. $98^{\circ}8$, p. 66.

44th day of disease. A.M. t. $97^{\circ}8$, p. 64, discharged at her own request; convalescent in aspect.

The concurrent data of First Relapse in this case are furnished in a separate Chart 3, in Section III., Chapter I., 'On the State of the Blood in Spirillum Fever.'

Another example is furnished in Chart 2, Plate IV. at the end.

Fever with Two Relapses.

CASE X.—H. A., æt. 36, male, Mussulman, petty shop-keeper, six months in Bombay, resident in a well-known fever locality; home Azimghur (N. India); admitted May 27, 1878, with fever of seven days duration. Morn. t. 103° , p. 110, soft; a heavy distressed look, pupils contracted, mind perplexed; tongue much coated, white at sides, dry and brownish in centre, with narrow fluid edges and tip; lips parched: slight jaundice, frontal headache, pains in loins and lower limbs (bones), no pains in hands: liver unchanged, spleen distinctly enlarged, not tender: skin soft, moist; no appetite. Heart's sounds clear, the first not prolonged, but rather shortened and weakened, even when the man sits up and the impulse so rendered perceptible. The fever is said to be continuous and more severe of late; has had a dry cough for five or six days. He is a spare, pallid, feeble subject: intelligent.

Blood-plasma clouded, but fibrillation distinct: little free protoplasm: red discs piled; very numerous active spirilla, commonly in rings. Ordered diaphoretics and stimulants.

Vesp. t. $104^{\circ}4$, p. 120, undulating: liver, spleen and epigastrium full or enlarged, and tender; bowels said to be regular; much thirst, can eat; skin dry, no eruption; no iliac gurgling.

30th. 7 A.M. t. $103^{\circ}6$, p. 124, full, but soft; no stool; tongue dryish: was slightly delirious last night, jaundice more marked, skin dry; there was some sweating in forehead at 8 P.M.: severe headache and pains in back and limbs; much thirst: urine said to be passed frequently in small quantities, high-coloured, sp. gr. 1010, acid: shows only a few granules under microscope. No eruption: pupils small: lips dry: systolic sound of heart faint: some bronchitic cough. Splenic dullness in axillary line measures 5 inches, from lower border of 8th rib to 1 inch below costal margin, and reaches to 3 inches from the median line: hepatic dullness from lower border of 5th rib to $1\frac{1}{4}$ inch below costal margin, in nipple line.

Vesp. 4 P.M. t. $104^{\circ}2$, p. 120, full, but soft: tongue dry in middle; jaundice deeper; skin dry, no sweats during the day: one semi-solid stool of yellowish-green colour: aspect anxious, countenance dusky; the man wanders in speech; pupils rather contracted. N.B. Acme of Invasion-attack about this time.

31st. A.M. t. $99^{\circ}2$, p. 100, smaller, soft; hepatic dullness now from upper border of 6th rib to costal margin, being therefore reduced: splenic dullness from lower border of 8th rib to 1 inch below costal margin, and $3\frac{1}{4}$ from the median line, being also reduced, but less so; the fulness and tenderness over hepatic, splenic and epigastric regions is less marked: one liquid, yellowish-green stool: tongue still dryish, and lips. No sleep in the night, being very restless and wandering in speech. Conjunctivæ less yellow, headache and pains less, thirst continues. Began to sweat freely at 6 A.M., skin now soft, forehead now wet; no eruption, or iliac gurgling: heart's systole seems stronger: complains of uneasiness in throat, congestion of pharynx and fauces present. He looks worn and harassed with features pinched, and is still delirious, being under the influence of fear: during the night he tried to run away: no account of increased fever then: the temp. has fallen 5° , and the sweating seems inconsiderable: no diarrhoea.

Vesp. t. $99^{\circ}6$, p. 108, soft, regular: tongue and lips still dryish: skin soft, one liquid stool containing blood and mucus: urine scanty, high-coloured, sp. gr. 1015 (with correction), no albumen, no sugar, chlorides, bile-acids and pigment in small quantity:

there is cough but no sputum or thoracic dullness; one bilious vomiting of greenish liquid: still some pains in head, trunk and limbs; no eruption; there was some sweating on forehead and chest at noon. Liver-dullness the same, no tenderness: spleen reaches only $\frac{1}{2}$ inch below costal margin, still tenderness here and over the epigastrium. (N.B. Crisis ended at noon, the temp. having declined to 98° ; and a rebound promptly follows.) Treatment sustaining.

June 1. A.M. t. $101^{\circ}8$, p. 112, full, soft. Hepatic dullness reaches as high as upper border of 7th rib, as low as $\frac{1}{2}$ inch above the costal margin: spleen reaches below only as far as the costal margin. No eruption, tongue moist, clearing; lips dry; 4 liquid stools, yellowish-green, with much dark blood and mucus: abdomen retracted, tender; straining and griping at stool. Slept for an hour, but was restless and wandering at night: headache and pains less: much thirst; skin soft, there was some sweating about midnight. Urine of night 20 ozs., sp. gr. 1022, colour high, acid, no albumen, bile-acids and pigment abundant, and chlorides $\frac{1}{2}$ vol. (i.e. much increased). No more vomiting; he looks haggard and pinched, is drowsy, pupils natural and act. Sedatives ordered.

Vesp. t. $102^{\circ}4$, p. 116, full, but soft; hepatic dullness the same, a little tenderness: splenic dullness reaches to upper border of 9th rib, below to costal margin and inwards to $3\frac{3}{4}$ inches from median line; still tenderness here. No eruption: tongue cleaning, lips dry; 3 semi-solid stools, highly bilious, with streaks of blood and mucus, strains at stool: slight body-pains continue: skin moist, there being some sweating during the day; thirst considerable: urine lighter-tinted, acid, 1020, no albumen, bile-elements and chlorides still plentiful. He has the same haggard aspect, eyes sunken, yellow, countenance dusky; pupils normal. N.B. this secondary fever was probably connected with the dysenteric symptoms. Weight 99 lbs.

June 2. A.M. t. $100^{\circ}6$, p. 100, full, soft: hepatic dullness the same, still some tenderness: splenic dullness reaches from upper border of 9th rib as far as costal margin, some tenderness here also: 3 semi-solid, yellowish stools with mucus, but no blood, still straining; jaundice diminishing: tongue moist, lips dry; face haggard, the cheek bones very prominent; forehead damp, less thirst, still headache and general pains: urine at night 24 ozs., acid, 1015, no albumen or sugar, chlorides $\frac{1}{2}$, bile-acids and pigment present. Uvula and pharynx still injected. Heart's first sound still feeble.

Vesp. t. 100° , p. 102, full, soft; aching pains in loins and in bones and joints of limbs (not the smaller joints), increased on movement. There is redness of soft palate and fauces, pain in deglutition limited to pharynx: no sign of paralysis here, but he swallows liquids slowly and with pain, irritation of larynx and hawking cough following. One scanty feculent stool: still tenesmus. Hepatic and splenic dullness the same. Weight 99 lbs. (N.B. The pharyngitis is a complication here, supervening with the rebound of temperature shown in the chart.) The man looks very low and weak, lips dry, eyes sunken, malar bone prominent; the sounds of the heart are feeble, short and clear, the second much predominating.

3rd mane. t. $97^{\circ}2$, p. 92, weak but regular, tongue coated white, with a tendency to dryness, lips moist, has throbbing headache, the pains in the joints is of a gnawing character, thirst less: limit of the liver upwards the upper margin of the 6th rib, downwards to within $\frac{3}{4}$ in. of the costal margin, the hepatic region is still rather full and tender: limit of the spleen upwards the lower border of 9th rib, downwards $\frac{1}{2}$ in. below the costal margin, still fulness and tenderness here, and also over the epigastrium: two stools feculent and bilious, without blood or mucus, and passed with a little griping and straining. With this fall there was some sweating at 6^o A.M. He looks more exhausted, the nose sharp, the cheeks hollow, and has a troublesome hawking in the throat; nothing wrong in the chest, the fauces are still reddened; he slept; no eruption. First sound of the heart limited to the apex.

Vesp. t. 98° , p. 88, still headache and pains: the hepatic dullness reaches to $\frac{1}{2}$ in. from the costal margin, the splenic has the same limits as before; skin moist, conjunctivæ clearing, no stool, urine free, sp. gr. 1020, chlorides $\frac{1}{2}$ vol.

4th mane. t. $98^{\circ}4$, p. 88, more full, regular; tongue clean and moist, little thirst, the headache and pains in the back much complained of, those in the limbs and joints are less: liver-dullness from upper margin of 6th rib to about $\frac{1}{2}$ in. from costal margin: splenic dullness unchanged: one feculent, bilious stool, passed without griping or straining, urine free, slight cough, no sweats, slept well.

Vesp. t. 98°, p. 80, headache less, no pains in joints, more pain in the back : liver and spleen of near normal dimensions, tongue cleaning, the pupillæ little prominent.

5th. m. t. 97°·8, p. 76, very weak, heart's action very feeble, first sound almost inaudible ; much general depression and languor ; the throat is much easier ; skin moist, tongue clean and smooth, still pains in the head, back and joints ; liver and spleen unaltered, one natural stool. Bark and ammonia, and alcoholic stimulants still administered.

Vesp. t. 98°·4, p. 84, much lumbar pain, less headache, and pains in the knees only : no stool : tongue clean ; still a little cough : 20½ ozs. of urine, with chlorides $\frac{1}{8}$ vol.

6th. m. t. 98°, p. 80, soft and regular, lumbar pain, some headache and pain in the knees, two stools, slight thirst, liver and spleen normal, urine free, tongue clean and smooth.

E. t. 98°·4, p. 84, weak and regular, pains the same, thirst less, no stool, tongue clean. N.B.—He was now becoming convalescent, seven days since the crisis of first attack.

7th June. Relapse : m. t. 99°·6, p. 104, full, bounding, soft : thirst, pains in head, back and knees are increased, he has a sense of burning in both feet : tongue slightly coated, tending to dryness in centre ; pupils normal : conjunctivæ yellowish. Fever came on at 10 last night, without chills, some sweating three hours later ; skin now dry. Urine 32 ozs. at night, acid, 1010, clouded, chlorides $\frac{1}{8}$ vol., no sediment. The spleen recedes in the lying posture and is then barely felt ; but when the man turns on his r. side, this organ descends 2 in. below the costal margin and is firm, not tender. Weight of body 99 lbs., or the same as on the 1st inst., despite the pharyngitis and dysenteric diarrhœa : yet no gain, as usually is acquired. Stimulants and support continued.

Vesp. t. 103°·8, p. 120, full, soft, regular ; tongue coated dryish, lips dry, skin dry, no stool, conjunctivæ yellowish, pupils normal ; pain in back severe, much headache, also pain in the knees and least so in small joints of hands and feet. Liver-dullness upwards to the upper border of 6th rib, downwards to the costal margin. Spleen-dullness upwards to upper border of 9th rib, downwards about 1½ in. below costal margin, and inwards 3 ins. from the median line ; there is fulness and tenderness over the entire upper abdominal zone. Urine in the day 30 ozs., pale, clear, 1007, no albumen. Much thirst (drinks water freely) : some pink spots on l. side of abdomen ; the man is oppressed, has a slight cough, throat easier ; there were sweats on the forehead about noon : he has good appetite. First sound of the heart short, but distinct. Blood-plasma clear, coagulation slow ; a peculiarity is the many floating granules in the plasma, active, rounded ; they seem to join together and two will sometimes appear connected by an invisible band : there are very few fully formed spirilla, intermediate shapes not seen.

8th. m. t. 102°·4, p. 116, full, soft ; dorsum of tongue smooth and dryish, lips dry, skin dry but not harsh, two consistent bilious stools in the night ; jaundice more marked ; pupils normal ; much pain in the back, the head, knees, legs, burning sensation in the feet : in the upper extremities pain said to be less ; slept ; fever exacerbated at midnight without chills, no sweats since, much thirst : liver-dullness as yesterday : splenic enlargement increased downwards $\frac{1}{2}$ in. and inwards $\frac{1}{4}$ in. : some fulness and tenderness over the entire upper abdominal zone : urine in night 30 ozs., pale, clouded, no sediment on standing, 1007 ; no eruption : his face is pinched and anxious, skin sallow ; no cough, the throat is less painful : has a hungry craving : first sound of the heart rather booming in the lying posture. Blood clear, coagulation slow, fibrillation distinct, there is free protoplasm of different sizes, a few granules and the spirillum rare.

E. t. 105°, p. 120, bounding, compressible, regular ; tongue coated, moist ; no stool : pupils normal, great thirst, pains as before, no eruption ; the liver now reaches $\frac{1}{4}$ in. below the costal margin : the spleen as in the morning : urine 26 ozs., acid, 1012, no albumen, chlorides $\frac{1}{8}$ vol., no sediment.

9th. m. t. 103°·6, p. 120, full, soft ; tongue white and moist at sides and tip, smooth and dry in middle, lips dry, one natural stool in the night, eyes yellow, pupils normal, much thirst ; much pain in the back, head, knees, burning in the feet, no pains in upper limbs, or in throat, no eruption : hunger felt ; liver about $\frac{1}{4}$ in. smaller

than yesterday; spleen also slightly reduced: urine high-coloured, cloudy, 1012, no albumen or sugar, chlorides $\frac{1}{8}$ vol., acid, no sediment; no abdominal uneasiness: no sweats, slept, is drowsy and exhausted. Weight 98 lbs.

Vesp. t. 103°·6, p. 124, full, soft; no stool in day; pains as before; urine 26 ozs., acid, 1017, cloudy, high-coloured, no sediment, no albumen, chlorides $\frac{1}{8}$ vol., bile-acids and pigment present; no abdominal uneasiness, slight sweating in the day; liver and spleen of unchanged dimensions.

10th. m. t. 104°·6, p. 128, full, bounding, compressible; tongue as before, lips and skin dry, no sweats during the night, the patient not sleeping well, but being restless and somewhat delirious: jaundice marked, much thirst and hunger: two semi-consistent yellowish green stools: pains very severe; throat better; no eruption: urine in the night 28 ozs., high-coloured, acid, 1015, cloudy, no albumen or sugar, bile-acids a trace, bile-pigment present, chlorides $\frac{1}{8}$ vol.: no abdominal uneasiness. Liver limits from upper border of 6th rib to costal margin: spleen from upper border of 9th rib to $1\frac{1}{4}$ in. below costal margin, and 3 ins. from median line: visage anxious and dusky.

Vesp. t. 105°·2, p. 136, full, soft; tongue glazed and dry in centre, lips parched, slight sweats on the forehead at 3; he is delirious, muttering to himself and tossing about the bed: eyes deep yellow; much thirst and craves for food; one bilious stool: pains as before; no eruption: urine of day 29 ozs., high-coloured, acid, clouded, 1010, chlorides $\frac{1}{8}$ vol. Weight of body 97 $\frac{1}{4}$ lbs.

11th. m. t. 103°·6, p. 120, full, regular, soft; tongue coated, dryish, lips dry, slight sweats on forehead at 1: no sleep, he being very restless, not so much delirious; two highly bilious stools; there is a sense of heat in the abdomen, no tenderness or fullness, much thirst and hunger: great pain in back, heads, knees and ankles, burning in the feet; no pains in the upper limbs; no eruption: urine of night 26 ozs., pale, clouded, acid, 1015, no albumen, bile-acids and pigment present, chlorides $\frac{1}{8}$ vol. Liver reduced. Spleen extends from the upper border of 9th rib to $1\frac{1}{2}$ in. below the costal margin, and $3\frac{1}{4}$ in. from the median line.

Vesp. t. 105°·6, p. 132, bounding, compressible; tongue brown on dorsum, moist, lips moist, there has been some sweating on the forehead; one bilious stool, a sense of heat in abdomen, no fulness or tenderness, much thirst and appetite, pains as before, no eruption; he looks very haggard and sallow. Liver-dullness from lower border of 6th rib to within $\frac{1}{2}$ in. of costal margin, where its edge can barely be felt. Spleen-dullness from upper border of 9th rib to $1\frac{1}{4}$ in. below costal margin, and $3\frac{1}{4}$ in. from median line. Urine of day 36 ozs., acid, pale, clouded, 1010, no albumen, chlorides $\frac{1}{8}$ vol. Weight 96 $\frac{3}{4}$ lbs.

12th. m. t. 104°·4, p. 144, full, soft; tongue coated black except in the smooth middle, dry all over; lips parched and cracked: no sweats in the night; skin now dry; one highly bilious stool; sense of abdominal heat is less, no tenderness or fullness here; much thirst and hunger; pains the same with burning in the feet; no pains in upper limbs; no eruption: his countenance is expressive of exhaustion and suffering. Urine of the night 30 ozs., high-coloured, clouded, no sediment on standing, chlorides $\frac{1}{8}$, 1015, acid, no albumen: liver and spleen unchanged. At 3.30 P.M. the t. was 105°, p. 132.

Vesp. (4) t. 99°, p. 100, small, feeble, tongue dark, moist, lips dry, skin covered with sweats now coming out, being preceded by rigors (*perturbatio critica*), eyes deeply jaundiced, pains continue, no burning sensation in the belly, no abdominal uneasiness, much thirst and hunger, no eruption: urine of day 39 ozs., acid, 1003, chlorides $\frac{1}{12}$ vol., no albumen, one bilious stool, liver and spleen unchanged since morning. Weight 93 $\frac{3}{4}$ lbs.

13th. m. t. 99°·2, p. 100, very feeble, regular: tongue brown, moist, lips moist, much sweating at midnight for an hour; skin soft, jaundice still, one stool, there is again burning sensation only in the abdomen, much thirst and craving for food, vomiting after ingestion; the pains continue, no eruption; urine of the night 28 ozs., pale, clouded, acid, 1015, chlorides $\frac{1}{8}$ vol., no albumen: sudamina in the groins, pupils rather contracted, abdomen collapsed, the liver reduced and retracted, the spleen extends from 7th rib to costal margin, and $3\frac{1}{4}$ ins. from median line.

Vesp. t. 99°·8, p. 112, very weak, regular; tongue cleaning, moist, lips moist, skin soft, two semi-solid stools, thirst less, appetite much, no abdominal uneasiness, no eruption; liver dullness unchanged, splenic-dullness from the 9th rib to $\frac{1}{2}$ in.

below costal margin and $3\frac{3}{4}$ in. from median line. Urine of the day 10 ozs., acid, pale, clear, no sediment, 1017, no albumen, chlorides $\frac{1}{4}$ vol. 8 P.M. skin cold, he is very weak and pulse feeble (stimulants and warm clothing), temperature not taken. Weight 90 $\frac{1}{2}$ lbs. this day.

14th. m. t. 99°·2, p. 112, feeble (said to be compressed by 150 grammes), tongue coated white, moist, no thirst, pains in back, head and limbs the same, no stool, conjunctivæ less jaundiced, pupils slightly contracted, no abdominal uneasiness, no eruption, liver unchanged: spleen of unaltered dimensions: urine of night 28 ozs., chlorides $\frac{1}{3}$ vol. Heart's action weak, impulse imperceptible, rhythm regular.

Vesp. t. 98°·8, p. 108, better volume (200 grammes borne), tongue clean, moist, a little thirst, pains less, no stool, conjunctivæ anæmic, pupils normal, no eruption. Liver—in recumbent posture the anterior border does not project below the costal margin, but on relaxing the abdominal walls and the patient taking a deep inspiration, this edge may be felt on upward pressure, and its notch between the right and left lobes can be detected; dullness in nipple line to upper border of 6th rib, and on deep percussion as high as the 4th rib. Spleen—dullness from upper border of 9th rib to costal margin and $3\frac{3}{4}$ in. from median line. Urine of day 18 ozs., pale, clear, no albumen, chlorides $\frac{1}{8}$, acid, 1028. There was slight sweating at 3 P.M., no chills; the t. descended to 97°·2, and this probably represented the end of a crisis delayed for some reason.

15th. m. t. 99°, p. 96, weak, tongue coated, moist, slight thirst, pains in head and upper limbs less, but in loins and extending down the legs very severe; some œdema of the feet, conjunctivæ pale, pupils normal, no eruption, no stool, no appetite. Liver—normal; spleen extends from upper border of 8th rib to costal margin, and 4 in. from median line. Urine of night 17 ozs., acid, chlorides $\frac{1}{6}$, no albumen, 1017.

Vesp. t. 97°·6, p. 92, better volume, tongue coated, dryish in centre, no headache, pains in loins and legs severe, no thirst, no stool, pupils a little contracted; liver unchanged, spleen the same, no urine since morning (ordered aperient). Body-weight 89 lbs., being a reduction of 10 lbs. in seven days.

16th. m. t. 98°·4, p. 100, feeble, tongue clean, some headache and pains in arms, much pain in loins and legs, no thirst, no eruption, one dark-coloured stool, conjunctivæ pale; liver unchanged, spleen reaches from 9th rib to costal margin and 4 ins. from median line. Urine of night 18 ozs., acid, no albumen, high-coloured, cloudy, 1022, no sediment, chlorides $\frac{1}{3}$ vol., skin soft, slept.

Vesp. t. 97°·6, p. 96, better volume (350 grammes needed), tongue coated, lumbar and lower limb pains severe, headache and pains in large joints of upper limbs slight, conjunctivæ pale and yellowish; no sweats; is weak and giddy; liver and spleen unchanged; urine of day 6 ozs., acid, pal., clear, 1020, chlorides $\frac{1}{3}$ vol., no albumen, bile-acids and pigment present.

17th. m. t. 97°·6, p. 92, tongue moist, glazed in centre, no sweats, one stool; urine 14 ozs., acid, high-coloured, 1020, no albumen, chlorides $\frac{2}{3}$ vol.

Vesp. t. 37°·4, p. 92, he is depressed, pupils a little dilated, much lumbar pain, no appetite, no stool.

18th. m. t. 98°·6, p. 88, weak, one stool, pains in thighs and legs considerable, and prevented sleep; urine 17 ozs., acid, 1017, clear, no albumen, chlorides $\frac{2}{3}$ vol.; the spleen is said to extend below the costal margin. E. t. 97°·8, p. 96, no stool; he is giddy, pupils a little dilated, the pains continue.

19th. m. t. 97°·9, p. 80, two stools, pains in legs less, and he can walk a little unaided: urine 22 ozs., acid, 1009, chlorides $\frac{1}{8}$ vol., no albumen, no bile-acids or pigment.

Vesp. t. 98°·4, p. 82, tongue coated, some sweating about the neck.

20th. m. t. 98°, p. 75, better strength, two stools, sleep disturbed by the pains, appetite improving; urine 21 ozs., acid, 1009, chlorides $\frac{1}{8}$ vol. E. t. 98°, p. 72.

21st. m. t. 98°, p. 72, one consistent palish stool; urine 24 ozs., acid, 1015, chlorides $\frac{1}{7}$ vol. E. t. 98°·4, p. 72.

22nd. m. t. 98°·4, p. 72, slow convalescence, appetite capricious, pains less; urine 20 ozs., 1015, acid, chlorides $\frac{1}{8}$ vol. E. t. 99°, p. 80; improving.

23rd. m. t. 98°, p. 64, slept, two stools, slight headache (frontal) and pains elsewhere. E. t. 98°·4, p. 76.

24th. m. t. 98°, p. 72, no muscular pains, giddiness, appetite increasing; urine 18 ozs., acid, 1012, chlorides $\frac{1}{6}$. E. t. 98°·4, p. 96, soft, one stool.

25th. m. t. $98^{\circ}4$, p. 76, weak, tongue slightly coated, still headache and giddiness, the muscular pains have returned to-day; urine pale, 1012, no albumen, chlorides $\frac{1}{8}$. Blood not visibly changed, a few granules are seen but no spirillum in the fresh blood. Second relapse.

2 P.M. t. $103^{\circ}8$, p. 82, weak (150 grammes), resp. 32: fever came on an hour ago, with chills which are still felt, though less sensibly: skin dry, tongue moist but glazed, some thirst, frontal headache and burning sensation in the eyes since morning, pains in limbs the same as before; the spleen seems unaltered, and the liver is not changed, one consistent stool.

4 P.M. t. $104^{\circ}2$, p. 92, skin dry, thirst moderate, there is headache and pain in the lower limbs. Blood—plasma clear, a few free granules, no spirillum (fresh blood).

26th. m. t. 101° , p. 96, soft, tongue slightly coated, one stool, no appetite, thirst moderate, muscular pains increased, headache and giddiness increased, less sensation of cold in the feet, skin moist, conjunctivæ sallow; urine 14 ozs., 1007, pale, no albumen, no bile-acids or pigment, chlorides $\frac{1}{8}$ vol. Blood—plasma clear; there are spirilla few but active, also some short active rods and free moving granules.

4 P.M. Fever exacerbated with chills at 3, t. $104^{\circ}4$, p. 100, soft: the pains continue the same, burning in the eyes, headache and giddiness; the liver and spleen do not seem to be changed, no tenderness of abdomen.

27th. m. t. $103^{\circ}4$, p. 110, weak, tongue coated, moist; no stool; no appetite; burning in the eyes, conjunctivæ sallow; the pains are much increased; he is depressed and can hardly answer questions; liver unchanged, spleen extends from upper border of 9th rib to 1 in. below the costal margin; no tenderness. Urine pale, 1009, no albumen, chlorides $\frac{1}{8}$ vol. Blood—clear, coagulation slow, much free protoplasm large and small, many white cells, no spirillum seen.

4 P.M. t. $96^{\circ}2$, p. 76, he is pallid and depressed; profuse critical sweating since 1 P.M., headache less, pains in limbs the same; he is too weak to sit up (ordered stimulants).

28th. t. $98^{\circ}2$, p. 88, small, one stool, tongue cleaner, headache and the pains are less, thirst moderate, appetite improved, skin soft: lungs and liver normal, spleen extends from upper border of 9th rib to 1 in. below the costal margin. Urine 20 ozs., pale, 1009, albumen none, chlorides $\frac{1}{8}$ vol. Blood—clear, coagulation slow, much protoplasm, many white cells and free granules, no spirillum seen: the free protoplasmic masses are both large and small in size.

Vesp. t. $96^{\circ}2$, p. 76, countenance pallid and expressive of exhaustion; profuse sweats since 1 P.M., skin now bathed in perspiration: headache less: still pains in the limbs; complains of inability from weakness to sit up.

28th m. t. $98^{\circ}2$, p. 88, small, soft, one stool in the night, tongue moist and cleaning, giddiness, slight headache, the muscular pains much diminished, thirst moderate, appetite improved, skin soft; lungs and liver are normal, spleen extends from upper border of 9th rib to about $\frac{1}{2}$ in. below costal margin. Urine acid, 1012, albumen and sugar none, chlorides $\frac{1}{8}$ vol.: 14 ozs. saved.

Vesp. t. $96^{\circ}4$, p. 80, no stool, pains the same.

29th. m. t. $97^{\circ}3$, p. 68, small, weak, one stool in the night, tongue coated, appetite increased, thirst moderate, slight headache and sense of burning in the eyes, pains in the limbs less, sleeps. Urine 16 ozs., acid, orange-tinted, 1016, no albumen, chlorides $\frac{1}{8}$ vol. Vesp. t. $97^{\circ}5$, p. 72.

30th t. $96^{\circ}4$, p. 62, bowels regular, slept well. E. t. $97^{\circ}2$, p. 64.

July 1st. m. t. $96^{\circ}2$, p. 60, feeble, regular, slight frontal headache, no pains in the limbs, the legs feel very weak; spleen now barely projects beyond the costal margin. Urine saved 20 ozs., pale, acid, 1016, no albumen, chlorides $\frac{1}{8}$ vol. E. t. $97^{\circ}2$, p. 64.

2nd. m. t. 97° , p. 60, weak, two stools, natural, tongue moist, cleaner, appetite good. Urine saved 15 ozs., sp. gr. 1005, neutral. E. t. $96^{\circ}4$, p. 68; tongue moist, slightly glazed, no pains, skin soft, the spleen has receded to the costal margin; urine pale, acid, 1007, albumen none, bile-pigment none, bile-acids present. Weight $96\frac{1}{2}$ lbs., so that he has gained $7\frac{1}{2}$ lbs. since the first relapse, notwithstanding the intervention of a brief second recurrence, and this persistent depression of frame.

3rd. m. t. $96^{\circ}5$, p. 56, weak, skin soft, giddiness and some headache, tongue glazed, moist, appetite good, sleeps well. Urine 26 ozs., 1012, acid, albumen none.

Vesp. t. $97^{\circ}5$, p. 68, soft, no stool; urine 6 ozs., 1016, acid.

4th. m. t. 97° , p. 64, one natural stool, only complaint is weakness. Urine 34 ozs., pale, acid, 1010, no albumen, chlorides $\frac{1}{4}$ vol. E. t. 98° , p. 72, of better volume.

5th. m. t. $97^{\circ}5$, p. 68, no complaint but weakness, liver and spleen seem normal; urine 35 ozs., pale, 1012, chlorides $\frac{1}{2}$ vol. E. t. 98° , p. 72.

6th. m. t. $97^{\circ}5$, p. 68, better strength, tongue slightly coated, one stool, slight giddiness and headache, did not sleep well owing to pains in the loins and knees; urine 36 ozs., pale, cloudy, faintly acid, 1012, chlorides $\frac{1}{2}$ vol., albumen, bile-acids and pigment absent. E. t. $98^{\circ}6$, p. 80, he feels a little feverish (? lateral relapse).

7th. m. t. $97^{\circ}4$, p. 64, weak, no stool, tongue slightly coated, some headache and pains in loins and knees; liver of normal dimensions; spleen extends $1\frac{1}{2}$ in. below the costal margin (*i.e.* has become enlarged). Urine 36 ozs., neutral, 1014, chlorides $\frac{1}{2}$, no albumen. E. t. $98^{\circ}6$, p. 68.

8th. m. t. $97^{\circ}7$, p. 54, small, weak, tongue slightly coated, still some headache and pain in the knees: is giddy and too weak to walk; one stool: appetite good. Urine 42 ozs., pale, faintly acid, 1009, no albumen, chlorides $\frac{1}{2}$ vol. E. t. $98^{\circ}6$, p. 74, better volume.

9th. m. t. 98° , p. 48, small, one stool, slight pains in lower limbs much increased on pressure, headache and giddiness. Urine 40 ozs., pale, 1010, bile-acids and pigment a trace, chlorides $\frac{1}{4}$. E. t. $98^{\circ}6$, p. 61, soft, fuller.

10th. m. t. 98° , p. 48, headache and giddiness on sitting up, lower limbs tremulous when extended, pains slight, one stool, tongue coated, breath offensive; urine 37 ozs., alkaline from ammonia, readily decomposing, 1015, no albumen, chlorides $\frac{1}{4}$ vol. E. t. normal, p. 64.

11th. m. t. 98° , p. 70, better strength: the spleen still extends below the costal margin; liver normal. Urine 32 ozs., pale, 1012, chlorides $\frac{1}{4}$ vol. Weight 101 lbs.: he gains. E. t. $98^{\circ}4$, p. 72.

12th. m. t. $98^{\circ}4$, p. 68, sleep disturbed from headache, giddiness and muscular pains complained of: the spleen seems to be a little larger again. Urine 36 ozs., pale, 1013, chlorides $\frac{1}{4}$ vol. E. t. 98° , p. 86, stronger.

13th. m. t. $98^{\circ}2$, p. 68, weak, tongue coated a little; still pains: appetite good; urine 36 ozs., pale, 1012, acid, no albumen. E. t. $98^{\circ}6$, p. 84.

14th. m. t. $98^{\circ}2$, p. 64, small; two watery, scanty stools, tongue coated, still headache; urine 38 ozs., pale, 1014, chlorides $\frac{1}{2}$ vol. E. t. $98^{\circ}4$, p. 68.

15th. m. t. 98° , p. 64, feels better; spleen still enlarged, extending from upper border of 9th rib to near an inch below the costal margin; urine 36 ozs., higher coloured, 1015, acid, no albumen, chlorides $\frac{1}{2}$ vol. Weight $101\frac{1}{2}$ lbs. E. t. $98^{\circ}4$, p. 72.

16th. m. t. $97^{\circ}6$, p. 68, weak; muscular pains have increased; the giddiness is less. Urine 36 ozs., 1012, acid, chlorides $\frac{1}{2}$ vol. E. t. $98^{\circ}6$, p. 68.

17th. m. t. 98° , p. 68, soft, pains less, still some headache: the spleen is enlarged as before. Urine 36 ozs., paler, acid, 1012, chlorides $\frac{1}{2}$ vol., no albumen. E. t. $98^{\circ}5$, p. 72.

18th. m. t. 98° , p. 64: improvement slow. Urine 36 ozs., 1012, acid. E. t. $98^{\circ}5$, p. 68.

19th. m. t. $98^{\circ}4$, p. 64, the pains are less; liver of normal dimensions; spleen still projects slightly below the costal margin. Urine 34 ozs., acid, 1012, no albumen, chlorides $\frac{1}{2}$ vol. He wishes to leave, and is discharged for better air. The chart is appended in Plate IV., No. 3, at the end: pulse tracings were taken.

Attack with Three Relapses.

CASE XI.—M., 30, Mussulman weaver, from N. India: admitted with fever of 7 days duration: he is so weak as to be unable to sit up; has diarrhoea; headache, thirst, pains, tongue white and dry, abdomen slightly full, hepatic tenderness: m. t. 101° , p. 120; e. t. $103^{\circ}4$, p. 126; spirilla in the blood. 8th day—m. t. 102° , p. 126, diarrhoea ceased, has splenic pain; e. t. 102° , p. 120. 9th day—m. t. 100° , p. 122, was delirious in the night, tongue brown in centre, hiccup, abdominal pain not more; spirilla present but fewer, and several moving particles seen: e. t. 96° , p. 90, this was the crisis. There followed a smart rebound of temperature (secondary fever), which lasted three days, the

hiccup and delirium persisting, with vomiting, injection of the conjunctivæ, prominent fungiform papillæ of the tongue, dryness of skin; treatment of the symptoms and support; on the fourth day the temp. became normal, and for three days longer he remained apyretic, the appetite being considerable. The first relapse then set in, being preceded by a mild paroxysm; it lasted five days and was of nearly continuous type: max. t. $104^{\circ}5$, p. 132, about the middle of the attack: the spleen was then enlarged but not tender, abdomen tense, stomach irritable, soreness of the throat, severe general pains: hiccup and delirium soon after re-appeared, the tongue became brown and dry, there was hepatic and splenic tenderness and bilious vomiting; crisis was moderate; e. t. 98° , p. 108, and not followed by relief; sweating moderate, much depression and persistence of delirium, vomiting and dryness of the tongue, some jaundice appeared. Secondary fever again followed, though less pronounced than after the invasion: he was prostrated, the heart's impulse and first sound being imperceptible in the lying posture: he complained of pains in the limbs and wandered in speech; the tongue was shrunk, dry and brown all over; and I noted the contrast between his state and that of a patient in a neighbouring cot, who had depression without these typhous symptoms. The man now rallied; there was some pulmonic congestion at first, and gastric irritability, with dry tongue, persisted for a few days longer: the appetite then returned, in spite of the vomiting; he expectorated a little blood: finally there was no complaint but pains in the calves of the legs. Fourteen days after crisis of first relapse the fever returned suddenly, about noon, without chills; it lasted three or four days, and was pronounced; bilious vomiting and slight jaundice returned, with increased pains: no delirium or hiccup: abdominal tenderness hardly marked: the crisis of this second relapse was pronounced, and not followed by a rebound. During the fever the urine was clear, pale, acid and free from albumen, sp. gr. 1012. Weakness, pains, cough lasted for a few days, and then convalescence progressed: there was a burning sensation in the soles of the feet at night, and the knees still ached. Seventeen days after the last crisis slight pyrexia re-appeared, without chills; there was gastric irritability and a red tongue, some headache, lumbar pains and thirst: these phenomena constituted the third relapse of two days' duration. Soon afterwards, the patient could not be persuaded to stay longer in hospital.

In its general features this case resembles No. 3: the Chart belonging to it is No. 4, Plate IV.

Fatal Cases.—Instances of spirillum fever ending in death were nearly as often as not complicated with pneumonia, hæmorrhages, and other lesions, giving rise to special symptoms of their own (*vide* the Chapter on Complications). As regards uncomplicated fatal cases, the majority of patients died about the time of febrile acme of the first attack; and the symptoms during early days of illness, were not peculiar or strikingly different from those of ordinary marked infection. Fever was seldom unusually high or sustained; but, on the contrary, often tended to be low or irregular, and in consequence the acuter fever symptoms less prominent: when pyrexia persisted to the last, the symptoms were those of the Acme of fever (*vide* that sub-section below); and when the blood spirillum continued to be visible with a declining or low temperature (as happened in about one-third of all casualty admissions of over a week's date), the symptoms seemed to me like those attending defervescence by Lysis (*vide* that sub-section below). It is remarkable that deaths at the stage of completed critical fall, at either invasion or relapse, were extremely rare; although it might have been anticipated that the very great depression then suddenly taking place, would of itself tend to a fatal result. Lastly, whilst it was not observed that other individual signs and symptoms were peculiarly marked in fatal cases, yet there was commonly apparent an early depression of the

system and tendency to the typhoid state, with decided abdominal suffering, dyspnoea and cardiac exhaustion : (*vide* Chapter on Prognosis).

Having thus pointed out several sources of information respecting this complex subject, it will suffice to add that in the Chapter on Mortality will be found other details needful for its entire comprehension. The following cases have been selected for general illustration.

Spirillar Infection ending in unexpected Death.

CASE XII.—M., 20, one of a party of famine immigrants from Azimgurh (N. India), others of whom were seen and some had died, was admitted on asserted 10th day of illness—t. 100°, p. 114, small, soft, regular; tongue pale, coated in middle, moist; the face pallid, complexion being muddy; abdomen full, being distended in upper zone, liver much enlarged and tender, spleen very large (reaching $2\frac{1}{2}$ ins. below costal margin), less tender; no stool for 9 days; no eruption; skin supple, sudamina in the axillæ. Heart's action tumultuous, first sound not prolonged but attended with decided murmur; second sound clear, impulse perceptible at scrobiculum cordis: pupils normal; has throbbing frontal headache, aching pains all over body, much thirst, occasional vomiting, a bronchitic cough: the blood is very thin, pale brown in hue and swarms with spirilla. No previous history of splenic enlargement, but the lad presents the aspect of malarious cachexia: body-weight 100 lbs. Vesp. t. 103°·2, p. 132, full but soft; liver reaches to lower border of 5th rib and below to costal margin, its region is tender; spleen reaches to upper border of 9th rib, and below 2 ins. below costal margin and 3 ins. from the median line, some tenderness here also; and over the somewhat distended epigastrium: skin soft, sudamina beneath the skin; lips dry, tongue still moist; less headache and pains in limbs, more pain in back, conjunctivæ sallow. There was some sweating at 11 A.M.: urine free, acid, clear, light-coloured, 1012, chlorides $\frac{1}{3}$ vol., no albumen or sugar, bile constituents present.

Next day, m. t. 101°, p. 124, full, firmer, tongue tending to be dry, lips dry: liver-dullness not increased vertically, still tenderness; the spleen seems somewhat pushed up; the epigastrium is mainly occupied by the right and left lobes of the liver, and is tender; 2 stools during the night (after saline aperient), of bilious brown hue; he slept, has slight headache and pain in the back and limbs, much thirst, some appetite; eyes yellowish; no sweats in night; urine 16 ozs., very pale and clear, chlorides reduced ($\frac{1}{10}$ vol.), no albumen, bile-acids and pigments a trace: no vomiting, pupils rather dilated. Vesp. t. 102°·4, p. 120, full, firm, regular; respirations 40 per minute; tongue coated with a yellowish fur, but moist; lips dry; liver-dullness extends from upper border of 6th rib to 2 ins. below costal margin; splenic dullness from upper border of 8th rib to $1\frac{1}{2}$ in. below costal margin, and $3\frac{1}{4}$ ins. from median line, epigastrium occupied by the hepatic lobes; all parts here tender on pressure: no eruption or fresh sudamina; one semi-solid bilious stool; no headache; pains in loins and limbs; conjunctivæ yellowish; pupils slightly dilated: skin dry but not harsh, much thirst, eats his food; urine of the day plentiful, pale and clear, 1012, acid.

It is said he slept the early part of the night, then awaking asked for water, skin being warm; did not rise; then he became delirious and gradually sank at 3 A.M. 7 hours after death I found extensive granular and fatty degeneration of liver, spleen and kidneys; the heart was dilated and hypertrophied (weight 12 ozs., substance pale but firm): the blood contained large granule-cells (endothelial or splenic) with fat globules in them; also a few languid spirilla.

Fatal Attack with Irregularly Developed Pyrexia.

CASE XIII.—F., 30, hospital-matron in good health previously and on active duty, acquired fever in her ward (where part of an infected family was lodged and where the clinical clerk at this time probably caught his fatal infection). Second day, m. t. 103°·2, p. 114, small, the fever began with chills at 10 A.M. yesterday, and she perspired freely; 4 hours later, chest sounds normal; abdomen free from fulness or

tenderness, tongue moist, almost clean; bowels free, no appetite, has headache, thirst and pains in loins and limbs, no jaundice: menses regular. Vesp. t. $98^{\circ}6$, p. 108, fever subsided, but at 8 P.M. it returned with headache. Third day—m. t. $99^{\circ}4$, p. 104, bowels opened, has vomited twice, still headache: e. t. $98^{\circ}8$, p. 104, as yet there were no symptoms to attract the special attention of the experienced native graduate in charge. Fourth day—m. t. $98^{\circ}6$, p. 106, purged thrice and vomited twice, evacuations bilious: e. t. 101° , p. 104: still no special symptoms. Fifth day—m. t. $101^{\circ}8$, p. 108, passed a quiet night, purged 4 times and vomited 4 times, no abdominal pain, grinds her teeth during sleep; tongue moist, thinly furred white, has headache and pains in loins and limbs as before, not much thirst; the belly seems puffed; compound sanctorine powder ordered, and cold to the head. Vesp. t. $101^{\circ}6$, p. 104. I found the woman restless and much distressed—her blood contained many spirilla. Sixth day—m. t. 99° , p. 108, free purging, no worms passed, no hepatic or splenic enlargement detected: she slept 4 hours (chloral given) pains of loins and limbs less, and less mental disturbance: skin clammy and she is low. At 8.30 the t. was 101° : vesp. t. 99° , p. 114, she is much oppressed but rational, pupils rather contracted, there are two red spots on right forearm, none on the back, a doubtful one on the left forearm: tongue now dry, brown on dorsum and rough, red at sides and tip, skin soft. Seventh day—m. t. 101° , p. 110, feeble and small, no sleep (in spite of chloral) and was delirious, one stool, conjunctivæ yellowish, tongue brown but less dry, pains of limbs and back, no abdominal uneasiness, some crepitus scantily heard in right infra-scapular region of chest, heart's impulse and first sound very feeble; skin perspiring, the headache is less and she sits up: is restless but tractable, eyes injected and heavy-looking, pupils decidedly contracted, grinds her teeth and wanders in speech at times: a few more petechial spots on front of left shoulder. Vesp. t. $101^{\circ}4$, p. 128, full but very compressible; she lies on back, often screaming and grinding the teeth, will not open the mouth, but tongue felt to be dry, head not hot, skin moist, no stool, urine passed in bed; abdominal fulness, tension and tenderness; she seems hysterical but doubtless the brain is affected, pupils dilated somewhat, very sluggish, the head turns to left side and possibly some facial paralysis, but no spasms seen: enema, cold to head, and sedatives; food. Later on the temperature did not rise; skin moist, no change, bowels moved, swallows with facility. Eighth day—m. t. $101^{\circ}2$, p. 124, feeble: little change, but pupils contracted, tongue dry brown and shrunk, no fresh spots, no sleep and still moans, yet is not unconscious, urine and stools passed in bed; a little sweating in the night; abdomen full, tense and tender; heart's first sound audible, though faint. Vesp. t. 103° , p. 140, small, regular, skin moist, some fresh spots on the chest, abdomen tympanitic and not tender, splenic or hepatic enlargement not detected, lungs seemingly unaffected. Pupils still contracted, she is nearly insensible, but can be roused; breathing 50 per minute, thoracic and jerking, no spasms; the typhoid state has not yet ensued. Later on the t. rose to 104° and p. 150, skin dry, pupils much contracted, (the pin-hole pupil) and much conjunctival injection, with ecchymosis at upper half of right eyeball, just beyond the normal edge, spasms of face and limbs, restlessness and tossing of arms, but not active delirium or entire unconsciousness: 10 ozs. of high-coloured, clear urine drawn off; procidentia uteri: symptoms of coma towards midnight, and death at 2 A.M. Autopsy 6 hours afterwards—Brain and membranes congested, with small petechial extravasations at vertex on left parietal and right occipital convolutions, and in right Sylvian fissure; brain substance soft, convolutions shrunken, and the moist membranes easily removed: mucous membrane of stomach showed many petechiæ along the lesser curvature, and was smeared over with viscid mucus, streaked black, which also extended into the œsophagus; there were petechiæ in the small and large intestine, common bile duct free; uterine mucosa pale: there were many sub-peritoneal extravasations of small size: liver firm, very large and very pale, a group of blood spots in left lobe over the stomach, bile abundant; spleen very large, dark, firm; kidneys rather large, flabby, mottled, both contained petechial extravasation. There were one or two hæmorrhagic spots beneath the parietal layer of pericardium; muscular substance of heart pale and friable; lungs inflated, there were sub-pleural and sub-mucous petechial spots.

CHAPTER III.

GENERAL PHENOMENA : PRODROMATA, PHYSIOGNOMY, BODY-WEIGHT ; ACME, CRISIS AND LYSIS. SPECIAL SYMPTOMS : HEADACHE, PAINS, THIRST, APPETITE, VOMITING, SWEATS, SUDAMINA ; THE TONGUE AND STATE OF BOWELS. HEART, PULSE, LUNGS ; LIVER, SPLEEN ; URINE.

Prodromata.—Particular interest attaches to the state of the patient 1, 2 or 3 days prior to the onset of fever, because on these dates the spirillum first appears in the blood, and some contemporary constitutional disturbance might be anticipated.

Precursory symptoms are such as appear *de novo* and spontaneously; they are mostly of subjective character, seldom pronounced and not always the same : as they may be overlooked or forgotten, it is possible they are never altogether absent. When present, they should be discriminated from signs of impaired health, so common in famine-fever patients prior to first attack ; and from the relics of previous paroxysms, in the instance of relapses. It seems likely that the more defined symptoms are truly initiatory; such are defective appetite, constipation, seldom diarrhoea, inability for sustained exertion of all kinds, much weariness, pains in the back and limbs, headache, flushing, chills, sweats (at night); a sense of burning in the eyes, or in the palms and soles, fixed neuralgic pain, hemicrania, vomiting, turgescence of the spleen (without pain); and the minuter changes of temperature, pulse and urine indicated below.

Such symptoms appear during the later specific incubation-period ; during the earlier part of this period when the blood is not visibly infected, they are unknown : though occurring when the blood-contamination becomes evident, they did not show any fixed relation to the new blood-state. If the latter be obscure or absent, the symptoms, too, may be almost wanting, as in the following instance :—H. V. C. was accidentally inoculated at an autopsy : no change noted till 4th morning, when awoke suddenly with headache and slight abdominal pain, not limited to the spleen, possibly some pyrexia, but an hour later t. $98^{\circ}2$, p. 72, and the specimen of blood taken did not show the spirillum : 5th day, dreams at night, some cough (? a cold in the head) ; 6th day, nothing definite and the late symptoms may have been incidental ; 7th day, a feeling of slight weakness, no spleen ; at duty as usual : 8th day, also out of sorts, but still to duty and had hopes that the risk was over ; blood free from contamination : at 3 P.M. headache and inability to work, no chills, fever beginning t. $100^{\circ}2$, p. 80. The spirillum was not found till next day,

but may have been overlooked by the patient : the ensuing attack was moderately severe ; no distinct relapse. For other quasi-negative examples at beginning of the relapse, see the cases above detailed.

In my experiments on the *Quadrumana*, the monkeys always seemed well until the access of fever ; though at the autopsy of three animals some slight organic changes were noted as already present during the specific incubation-period. In hospital, the more intelligent patients seldom (*e.g.* not one-half of them) made more than a vague mention of prodromata ; their statements are, however, recorded here for future use : at present, it is uncertain whether or not any of the premonitory symptoms named are common to several fevers, or peculiar to the spirillar ; their relation to preceding kind of infection and ensuing attack, and to the co-existing blood-state, have also to be accurately determined. The relationship to preliminary aguish attacks is considered elsewhere.

Perturbations of Temperature and Pulse as *premonitia*.—It might be supposed that precise objective data would be most likely to furnish definite information regarding pre-febrile changes ; and doubtless this opinion is correct. At present, however, such data are very scanty, the following being a summary of those available :—Invasion-attack : in my own case no changes of temp. were felt, nor did the pulse vary, so far as was ascertained. First Relapse—the ordinary chart of the woman Case IX. furnishes signs of a sinking of temp. on 2nd day before onset of fever, the pulse also declining ; and the 3-hour chart shows this depression very evident on the 1st day before, pulse still less frequent ; spirilla in the blood ; prodromata obscure : *vide* Plate 2. Plate 3 displays a longer 1st interval, with decided decline of t. and p. until near the relapse ; yet these changes do not seem connected with ascertained state of the blood, excepting that with permanent advent of the spirillum there occurred distinct perturbations : other signs obscure. A few other instances are available of ascertained pre-febrile infection, when only bi-daily observations were made, and consequently nothing precise learnt of temperature and pulse movements : here, too, the prodromes were not marked. For a slight depression before advent of a quasi-latent relapse see the chart of the woman K., Plate 1, Chart 9a. I have known the pulse alone rise from 76 to 100 for two days before relapse, when the spirillum was present : temp. at 98°–98°·4 F.

Comparative experiment has furnished useful data of temperature (other details being inaccessible) during the incubation-period of first attack ; and these accord with the human data in so far as showing, with much variability, a tendency to decline of body-heat just prior to febrile onset. An early analysis of 11 examples may be summarised as follows— inoculation being followed by a slight decline, there ensues a decided rise next day, or on the third, with a delayed night minimum, the blood at this time being free from visible contamination ; upon appearance of the spirillum, however, the temp. is found to subside below the mean, or to become more level, and then just prior to fever there occurs a decided fall, or else a preliminary rise : high pyrexia coming on quickly afterwards.

General Aspect and State during Fever.—Usually the patient takes

to bed on the first or second day of illness, and there lies supine until near the end of attack, being greatly indisposed to move ; but under the stimulus of necessity many individuals keep afoot, and according to their own account (which dates of fever confirmed) persisted at work or on long journeyings : whence it would appear the early prostration is not so absolute as in typhus proper. Such cases correspond to the 'ambulatory' forms of continued fever.

During the course of high fever, the physiognomy of patients was new to observers at Bombay, and so striking as sometimes to be recognisable at a glance. After two or three days the visage acquires a livid or bronzed hue, which is not like the effect of sun or dirt, or quite the flush of ordinary fever or of thoracic disease ; but is comparable rather to a combination of a dusky typhus hue, with the semi-translucent tint of native skins. It was seen best in Hindoo agriculturists and wandering mendicants, and less well in the more pallid Mussulman weavers and town residents : in black skins it was barely visible. Turgescence of the integuments was rare : the conjunctivæ were clouded and seldom injected, the eyes heavy rather than bright or suffused ; the pupils large rather than contracted.

More significant is the weary, haggard or hapless expression of the patient : features shrunk or drawn, with a slight frown and raising of the nostrils and upper lip indicative of distress, whilst the attention is, as it were, concentrated inwardly. Sometimes the expression was very stolid.

This remarkable 'facies' seemed most striking when the blood was charged with the parasite, and the abdominal symptoms pronounced. Even infants showed it : and all patients best during the severer invasion-attack : I do not know that it had a prognostic import. At relapse the face was often pallid and shrunken, and the aspect distressful, as if from pyrexia alone. The presence of complications modifies these appearances.

The posture of the sick may be supine, with the knees drawn up ; or lateral, with the legs and trunk bent so that the patient lies curled up ; he changes his position from side to side, unless either liver or spleen is pre-eminently tender.

The contrast here with ordinary typhus is noteworthy ; and I may add that inoculated monkeys during spirillum fever assumed a similar bent form, which was not presented by those animals suffering from other febrile infection.

When delirium supervenes the general aspect becomes that of direct blood-poisoning, and I have known this state closely simulate that of poisoning by Dhatura seeds : in the case referred to the man shortly died ; and without delirium, the distress may become mortal, as I have also witnessed. See 'Acme' and 'Crisis' below, for other details.

Body-weight.—This furnishes a ready test of the effects of specific fever. Robust individuals nearly regained their weight a few days after the end of first attack ; not so the weaker subjects, and in case of secondary fever at this time—thus the man whose case is detailed above (H. A., No. X.) weighed 99 lbs. av. at the beginning and end of the first apyretic interval, having gained nothing in weight during the ordinary

restorative period. Brief relapses leave their mark, one of a day's duration bringing down the weight 1 lb., which was not regained for two days more ; and longer ones, even when uncomplicated, produce a decided effect. Thus in the man H. A. the weight was 99 lbs. on first day, 98 lbs. on third day, $97\frac{1}{4}$ on the fourth, $96\frac{3}{4}$ on the fifth, there being a loss of $2\frac{1}{4}$ lbs. during four days' pyrexia ; promptly with main critical fall, sweats attending, the weight sank to $93\frac{3}{4}$ lbs., and the depression with sweats continuing, though not excessive, it was next day $90\frac{1}{4}$ and on third day of fall to the lowest point, it was 89 lbs., showing a loss of 10 lbs. or $\frac{1}{5}$ of body-weight. In the woman's case (F., No. IX.), the patient weighed 76 lbs. on evening before relapse began, and 66 lbs. at the fall, showing also a loss of 10 lbs., or at least $\frac{1}{5}$ of body-weight : the regain was hardly perceptible at first, and after a week at the rate of only $\frac{1}{4}$ lb. a day ; it attained to $1\frac{1}{4}$ lbs. daily at the end of a fortnight (body-weight 74 lbs.) and continued to advance. From these few data it appears that emaciation takes place chiefly at the end of the febrile attack, when copious sweats occur ; that during continuous pyrexia it may not be considerable ; and that recuperation after fever is but slow at first. When the infection has been severer than usual, rallying is delayed ; thus a young man weighing about 110 lbs. at beginning of illness, 11 days after relapse weighed only 95 lbs., showing a loss of 14 lbs. even then. When local inflammation concurs, doubtless the emaciation increases ; and wasting must be regulated by many conditions besides fever, which will be obvious upon reflection.

After the crisis when the patient begins to move about, his emaciated and weak state may remain hardly less characteristic : muscular debility is often considerable ; I have known dislocation of the lower jaw occur almost spontaneously. Convalescence was commonly slow at first, and it has been already stated that the pulse for a time declines in frequency after crisis.

Acme of Fever and the *Ferturbatio Critica*.—At or near the close of the attack, the febrile and general excitement commonly become augmented ; and fresh symptoms, more or less urgent, make their appearance. This final paroxysm may be so slight and brief as not to impress patient or attendant ; and when it occurs, as is usual, during absence of the medical officer, it is apt to be overlooked : judging from my last series of 60 cases, it is but rarely absent. There are, indeed, many degrees of this exacerbation, and the time of its occurrence varies much : in mildest form the body-heat and, less markedly, the pulse may almost alone be excited, but commonly there is increase of headache, thirst, body-pains and abdominal uneasiness, the tongue quickly becomes dry ; deafness, epistaxis, giddiness or delirium ; dyspnoea with a dusky or cyanotic aspect ; dysphagia, fulness and tenderness in the upper abdominal zone, hiccup, vomiting, a sense of distension ; and in addition much depression, will on different occasions be noted. Usually a remission of fever takes place just before the exacerbation, and it may be decided : this point is well worthy of notice. The succeeding rise of temperature, as ordinarily estimated, varies from 1° to 5° F., and it holds no fixed relation to the attendant symptoms, for I have seen the thermometer at 105° or 106° , with but little distress,

especially in relapses of both young and old : and, on the other hand, at 103° there may be much suffering : as a rare event, more than one exacerbation occurs, and then the final paroxysm is seen to be only the last of a series. At this time the pulse becomes very frequent, rather before and until after the acme of temperature ; it is very compressible and may be irregular, intermittent or dicrotic ; 140 or 160 pulsations per minute are not very rarely counted ; the heart's action is invariably weak : the respiration is much quickened, and in degree possibly peculiar to the spirillum fever ; yet with 50 inspirations per minute there may be but little distress, at least in youth. The abdominal signs vary not less than the rest ; spleen, epigastrium and liver, in different cases, being most complained of, and I could not detect a fixed ratio between the implication of spleen or liver, and the general suffering : the urine seems unaffected, or at most contains traces only of albumen. With the dyspnoea some degree of impaired resonance may be detected at base or apex of a lung, and incipient pneumonia may be suggested ; yet with the crisis complete relief will arrive. Epistaxis occasionally occurs at this juncture, heat of head (without headache) has been noted with it : the pupils are not affected necessarily ; the skin is dry ; body-aches may be so much increased, that the patient dares not move : and, in sum, his general aspect becomes striking—the harassed look, pinched features, hurried breathing, prostration ; often distress, restlessness and even delirium, forming a conjunction not seen at Bombay during late years, except in these cases of relapsing fever. When the attack ends gradually or by lysis, the acme becomes, as it were, anticipated, and the critical phenomena are mostly wanting.

In about one-fourth of cases under my care there was clear intimation of a *perturbatio critica*, ushered in by chills or pronounced rigors, of a few hours' duration, at most, and immediately preceding the crisis. At this time the above-named symptoms were very prominent, yet not different except in degree from those of ordinary acme ; and since the chills are not quite peculiar, I infer that this phenomenon naturally belongs to the category of events attending the sudden close of spirillar blood-infection. In addition to the example afforded by the woman's case quoted above—as No. IX.—another instance is here described, as I saw it ; and a third, copied from the J. J. Hospital records.

These phenomena have been seen at all ages beyond that of infancy ; and in both sexes. The illnesses in which they occurred were simple, but well defined ; the prolonged, low, typhus-like and complicated forms seldom displayed them. It happens that most of the attacks were relapses, and this preponderance may not be accidental.

Diagnosis.—For obvious reasons, it might be desirable to prepare for the acme and be made aware of its presence : as the patient himself may not be in a condition to furnish the subjective signs, his general state may then be considered with reference to history of the case and previous duration and course of illness ; a late remission not attended with persistent relief may presage the final exacerbation. Actual symptoms are almost characteristic, especially those of the *perturbatio critica* ; the temperature commonly rises high, but its height m. or e. is not an absolute guide to the stage of the case. The aspect of the blood is almost characteristic : see Sect. 3, Chap. I.

I might here quote for illustration my own late attack, during which I remained in my rooms for 4 days, and then in anticipation of the final rise was conveyed to quarters in the hospital; the same night, 5th-6th day of attack, great distress and delirium set in, the only relief attainable being from constant sponging with iced water (temp. after this $105^{\circ}2$).

The influence of local complications must be considered; it may tend to concealment of the acme; but the blood-condition would still be available as guide, and particular symptoms could be referred to their probable origin.

Prognosis.—Owing, doubtless, to the brief duration of stress, this is more favourable than might be anticipated; yet the truly critical character of the epoch is clearly shown by the fact of more than 54 per cent. of all deaths happening at this time of invasion-attack. It has seemed to me that a fatal result might occur in any pronounced case at this stage, and often it appears to be really contingent; nor do autopsic revelations always dispel this view. Upon comparing the symptoms of fatal and surviving instances, a similarity in certain respects is perceptible, as regards troubles of the circulation and respiration, and state of the abdominal viscera; with consequent general suffering. My data show that besides the depression, dyspnoea, moaning, restlessness and semi-consciousness, there soon follow before decease the more general changes pertaining to particular modes of death: nor are the above-named symptoms wholly peculiar in themselves. No more, therefore, than a general resemblance in the following examples could be looked for: in other instances enfeebled patients died exhausted before acme of attack, and some from cerebral hæmorrhage probably at this stage supervening; the symptoms in both series being modified accordingly. The common connecting link of all cases, seems to be formed by the state of the blood at acme of the fever.

Symptoms at acme of Specific Fever.

CASE XIV.—M., 35. 4 P.M. t. $105^{\circ}6$, p. 132, resp. 26; a little sweating, no headache, much thirst, pains in loins and neck, no delirium: an hour later I noted—there is much distress and he wanders in his talk, but is tractable: breathing quick and laboured, as if air entered the lungs with difficulty, the chest is fully expanded, the left lung seeming to encroach on the pericardium, pushing the heart over to the right, respiration is rather harsh, no dullness on percussion: the left side of the chest measures $\frac{1}{2}$ inch more than the right; is all this left predominance due to enlargement of the spleen? Heart-impulse feeble, but pulsations are quite perceptible at the tip of the sternum and rather to the r. side, as if the r. side of the heart were beating forcibly; the second sound is alone heard distinctly, there is no murmur with the first sound over the cardiac region; at the base, the second sound is alone heard; along the aorta there is a sound which might be called a murmur—a rumbling or friction sound which renders the second sound here indistinct, and suggests the idea of a feebly acting ventricle: it is not heard more to the l. side than to the right (*i.e.* is not produced in the pulmonary artery chiefly): area of cardiac dullness extends more to the right than usual and is elongated transversely; it blends with the liver-dullness. Spleen—is decidedly enlarged but it does not extend beyond the costal cartilages; tenderness here. Liver—is not much enlarged upon percussion, it may be pushed upwards by the distended stomach, tenderness here. Stomach—distended with air and tender: no gurgling or tenderness in r. iliac fossa. Urine of the day—18 ozs., high-

coloured, clouded, no sediment, acid, 1010, chloride $\frac{1}{4}$ vol., albumen none. The blood contained several spirilla at this time, which were also found 6 hours later, disappearing with the great fall of temperature which then ensued. Crisis began between 1 and 4 A.M.

The small monkeys of my inoculation experiments did not seem to suffer especially at the acme; yet here it may be observed that observation was defective; and it should be remembered that without unceasing attention the data requisite for absolute precision cannot be procured.

Uncomplicated Fatal Cases dying at close of Invasion-attack.

CASE XV.—F., 14, condition fair; the most urgent symptoms refer to the upper abdominal zone: drowsiness on the 7th (last) day: m. t. $103^{\circ}6$, p. 140: at 2.30 P.M. t. $104^{\circ}6$, p. 160, resp. 36, she was then nearly unconscious, pupils dilated and fixed, breathing shallow, pulse barely perceptible; there is great tenderness of the abdomen on pressure, and she screams on being touched there: death in four hours. No spirilla in the blood, but large granule cells, with endothelium; no coarse post-mortem lesion.

CASE XVI.—M., 22, clinical clerk, on estimated 6th day of invasion-attack m. t. $103^{\circ}4$, spirillum not seen by Mr. S. A.: e. t. $104^{\circ}4$: 7th and last day, m. t. $103^{\circ}4$, no spirillum; the pulse had risen from 130 to 140: in addition to the pyrexial symptoms were some jaundice, an eruption of pink spots, diarrhoea, cough; then less headache, anxiety of countenance, tendency to stupor; finally, skin moist, evacuations passed in bed—lips moist, tongue moist though brown, eyes bright but only at intervals intelligent, pupils slightly contracted; he occasionally turns on the side and moans; the chief feature is cardiac weakness, pulse hardly perceptible and uncountably frequent, both sounds of heart audible though rather muffled; no cough or dyspnoea, resp. 70, shallow, chiefly abdominal; abdomen full, tense and very tender over the spleen (projecting 2 inches) and liver (rather less enlarged downwards), stomach distended, much thirst, no hiccup or vomiting; the lad is not in the typhoid state' (MS. notes). The blood contained many white cells and an equal number of larger pale, granular cells; red-discs shrivelled and heaped. An hour and a half later sudden insensibility came on, with loud moaning, cooling of the limbs, skin of trunk hot and dry; death: at the autopsy, few palpable changes, the chief being spleen enlarged to 17 ozs., infarcts not noted.

The Phenomena at *Perturbatio Critica*.

CASE XVII.—M., 25. Admitted late in invasion-attack: symptoms pronounced; on last m. 6 A.M. t. $102^{\circ}4$, p. 110, much headache, thirst and giddiness; tongue white on dorsum, red at edges, moist; cough with scanty sputum, no pain in chest, yet respirations 70 per min. abdominal and shallow: fulness and tenderness over epigastrium and liver, not over the spleen, pains in loins, legs and arms, three stools; was delirious during the night, but tractable. Two hours later t. 105° and he was shivering strongly, the cot shaking with the rigors—sweating followed and at 4 P.M. t. $95^{\circ}2$, p. 62, no collapse and he is sitting up at comparative ease; drowsy, no dyspnoea. The succeeding relapse was very severe: the t. declined on 6th day, the typhoid state threatening and the spleen becoming much enlarged: 7th day at 5 A.M. t. $100^{\circ}6$; 6.20 A.M. t. 103° , p. 120, soft, regular; strong chills at 6, now much headache and distress, tongue brown and dry, stools and urine passed in bed; I saw him soon after and noted the great change for the worse since last evening—dyspnoea urgent, resp. 60 per min., breath cold, chest expansion imperfect, heart's action very feeble, pulse very rapid though regular, pupils dilated; he was barely conscious, and seemed dying from clots in the heart: blood free from the spirillum. 10.30 A.M. t. 99° : 11.30 t. $98^{\circ}4$, p. 106, resp. 46, some sweating, less distress, he looks drowsy; 2.30 P.M. t. $96^{\circ}4$, p. 90, resp. 40, skin moist: 5.30 P.M. t. $96^{\circ}2$, p. 88: next morning he had rallied further, t. $97^{\circ}4$, p. 80, resp. 36, no dyspnoea, still abdominal soreness, the spleen smaller and harder: he was greatly reduced in aspect yet promptly rallied.

The Crisis.—Amongst survivors, defervescence is thus abrupt in at least 90 per cent. of cases.

True crisis being limited to the terminal period of specific fever, all pseudo-crises or previous declensions of pyrexia, however considerable, will be found to be of different relationship. Critical symptoms may be somewhat alike at both true and spurious events ; but are much more pronounced when specific blood changes concur : amongst themselves, the several symptoms variously predominate. The general state of depression corresponds, generally, to degree of decline of pyrexia ; this is not, however, always so, as my cases show. The course and duration of crisis differed, even among instances otherwise not unlike ; yet I would specially mention that the general symptoms were almost invariably severe when the critical descent was slow to begin, and in its earliest course particularly—such prolongation of this stage approximating to fall by lysis. After the main subsidence is effected, depression may become somewhat intensified, or the lowest state be maintained for a day or more, with results either serious (as when active delirium ensues) or virtually mild ; and the restoration of strength and a quasi-normal state is attained with varying promptness, its general rapidity being one of the marvellous features of the spirillar infection.

It did not appear that towards the extremes of age the critical symptoms were more marked or otherwise diverse : older subjects did not suffer more, rather to my surprise : experience with infants was limited. Sex—females were not affected more than males at this stage of spirillum fever.

Weak subjects suffered most with the same degree of crisis ; the robust oftener showing extreme depression with comparatively slight constitutional exhaustion. The influence of caste and race, or of occupation and diet, was not apparent. As regards first and subsequent attacks, although the ranges of temperature at the close of relapses are somewhat greater than at end of invasion, yet in general the critical symptoms were not so pronounced ; especially when, as usual, the duration of the relapse was short : and whilst there is no essential difference to be noted in the character of these symptoms in early or late specific attacks, the sweats may be more copious and emaciation evident at the end of first relapses ; then, too, the critical changes are apt to be more sudden, and convalescence earlier. A minuter comparison could be made only between the longer relapse, and ordinary invasion ; for it seems that it is the duration of the fever in the spirillar infection, which (*cæteris paribus*) practically determines its clinical effects upon the frame. In even marked recurrences the general symptoms are seldom quite as severe as at first, notwithstanding the debilitating effects of a previous attack ; but the spleen may be more implicated, and the signs of simple depression as pronounced, even when relapse has lasted only a day or two.

Amongst Mohanedan weavers with prolonged, low fever and less pronounced changes of temperature, the critical symptoms often partook of a typhous character ; yet exceptions occurred when I least expected them : and whilst it may be surmised that the late epidemic in its course changed from the sthenic to the asthenic type, still in the last seen cases (1880) these symptoms were (like the blood-infection) hardly less marked than in 1877.

In a clinical sense, the phenomena of crisis may be regarded as composite in character ; there being present at this epoch, a certain degree of febrile exhaustion and a state approaching to that of collapse or shock. The first-named element not being peculiar, further comment is unnecessary ; but that the second is also of mixed nature is indicated by the fact of depression being sometimes considerable with only a moderate critical defervescence, whilst, on the other hand, with a marked crisis anything like collapse may be wanting. In fact, the crisis of spirillum fever is accompanied by remarkable changes in the blood, as well as by a sudden cessation of pyrexia.

As clinical varieties of this stage, there were noted numerous instances—the majority, indeed, in which there ensues prompt and almost complete relief from previous suffering ; or the shock symptoms will predominate, as in Cases XVIII. and XIX. quoted below ; or, lastly, with even considerable decline of temperature, some febrile phenomena may persist, resembling those of lytic defervescence. Analogous phenomena in other fevers will be alluded to under the head of Pathology.

As a summary of my notes on the terminal symptoms of spirillum fever, I may add that as probably peculiar to its crisis in their conjunction and comparative frequency, are the dilated pupils, persistent thirst, a tongue now becoming dry, still quick pulse and breathing, booming heart-sounds, supervening pulmonic congestion (bronchial or parenchymal), abdomen tender, with still enlarged viscera in its upper zone and sometimes retracted or distended, hypogastric uneasiness, retained fæces, retained or suppressed urine ; eyes injected, active delirium, a subjective sensation of abdominal fulness or internal heat, of tingling in the limbs, pains in locomotive organs ; an irregular distribution of body-heat, an eruption of pink spots, hunger, striking emaciation, œdema of the insteps or dorsum of the feet, epistaxis, melæna, and as early sequelæ pneumonia, parotitis, jaundice, diarrhœa or dysentery.

Common critical symptoms may be unusually pronounced, when the patient during fever has been weakened by diarrhœa, vomiting or sleeplessness.

The comparative predominance of usual and the less common symptoms is considered under the headings below ; and so the attendant state of blood ; and the relations of crisis to rebound or secondary fever, as well as to localised complications, in surviving and fatal cases.

Diagnosis.—In ordinary specific attacks, whether first or recurrent, there is seldom any difficulty in recognising the end of fever by both general symptoms and state of the blood ; the abrupt cessation of fever and disappearance of the spirillum being distinctive. But it is necessary to bear in mind that either of these phenomena may occur separately, and that the only proof of a true crisis is the non-existence of visible blood-contamination : thus, during specific fever the ordinary remissions may be so prolonged as to become intermissions with sweating and exhaustion, which could hardly be distinguished from a critical event except by remembering that invasion-attacks rarely last fewer than 4 or 5 days and are not liable to such intermission, whilst the relapses become increasingly intermitting in character. Moreover, irregularities

of pyrexia and other symptoms attend the close, as well as beginning and course of an attack ; thus, the *perturbatio critica* (or final paroxysm) may be so isolated from the previous sustained fever, as to become separated by an interval closely simulating the true crisis—such event being commonest in the relapses : or just after the true crisis there may arise an additional paroxysm, also resembling a part of the main attack—this event likewise being commonest in relapses, though I have seen it more than once after both primary and recurrent attacks : these rarer phenomena cannot be foreseen, and at first they were discriminated with difficulty. On the other hand, the true crisis may be not only recognised, but confidently foretold from the state of the blood ; for with absence of the spirillum, specific fever ceases for good.

That this fever may terminate without there being apparent any critical event, was several times noticed in attacks complicated with pneumonia, hepatitis, and some other acute inflammations. Here the symptomatic fever either co-existed with, or so promptly followed, the spirillar, that there was no visible interruption of continuous illness, or so little as to be seemingly insignificant ; and the microscope alone could determine the date on which specific blood-contamination ceased. I have elsewhere shown this confusion of febrile symptoms may obtain at the beginning of a relapse ; and it then becomes somewhat greater, for according to my experience the true crisis seldom fails to be indicated by some relief of febrile distress (however momentary or slight), even when symptomatic pyrexia runs high. It is very rare that a primary attack certainly lasts longer than 8 days (whatever time patients may name) or less than 4 or 5 ; relapses being much more variable closer attention may be needed to detect their critical termination. During irregular invasions and intermitting relapses, whether first or second, whilst the temperature-range is large, yet the pulse varies less ; general or local (even splenic) symptoms may be hardly characteristic, and a pseudo-crisis not be distinguishable from a true crisis without blood-scrutiny : and considering that the infection in these instances is seldom very abundant, the first requisite to accurate diagnosis is a thorough carrying out of such scrutiny.

Prognosis.—I could not but observe that all the many patients seen passing through extreme degrees of crisis recovered even promptly, and seemed to suffer no evil consequences from this wrench to the system : hence the inference that prognosis is not to be guided alone by appearances, however suggestive. The contrast here is great with the undoubted risks accruing at the acme of febrile attack. It is possible my large series of fatal cases is incomplete, yet, as it stands, there is hardly a death at end of marked crisis, or even at its beginning, unless some complication were present such as of itself would account for decease : therefore mere exhaustion must be rarely very serious in hospital, whatever result outside may follow, from the want of artificial warmth, stimulants and food. I note that amongst 109 recorded casualties there were only 9 instances of death at end of fever with symptoms approaching those of critical prostration amongst survivors : and since these include cases of decease during lysis and with local complications present, it results that no evidence is available of death from critical shock or collapse alone.

This circumstance indicates that the abrupt termination of spirillum fever is truly a relief-process tending to health.

To the following illustrations I have added a striking case of premature and repeated exhaustion, as a sample of clinical phenomena still imperfectly understood.

Cases illustrating the state at Critical Defervescence.

CASE XVIII.—M., 25, condition fair; admitted at mid-invasion period, fever high with remissions, crisis not so direct as usual, sweating scanty and irregular at first; 7th day m. t. 102° , p. 112, decline in progress, skin moist with sudamina in neck and axilla, giddiness, no headache or pains, urine free: e. t. $96^{\circ}4$, p. 80, the main fall with much sweating at noon. Next day m. t. $94^{\circ}4$, p. 72, feeble, regular, skin clammy, tongue dry, thinly coated white in middle, red at sides and tip, vomiting in night of bile, urine free, no stool, slept, deafness and giddiness, much debility and emaciation within the last 24 hours; e. t. $94^{\circ}2$, p. 84, feeble but regular, skin clammy, countenance dusky, oppressed, eyes sunken, voice low, has not taken food or medicine (stimulants) since morning, no thirst or headache, giddiness, no stool, urine free, vomited twice during daytime—a remarkable condition, abdomen collapsed and not tender, he looks like a corpse and his voice is barely audible, his complaint is that he cannot eat, the intellect is perfectly clear, but he seems hardly to live; no distress, the state being one of pure exhaustion, such as never before seen; heart's sounds clear, the first being very weak; there is irritability of the stomach, so that he cannot retain liquids (notes at bedside). A slow and halting advance to the normal state then began: the temp. of the trunk rose before that of the limbs, especially the lower; the pulse gained strength gradually rather than rapidly; no sweats; hiccup came on for a time and the urine was passed in bed once, vomiting continued; but no complication being present, in six days he had rallied: four days later a smart 'relapse' set in; at this date it was noted that desquamation of the whole body was taking place. He was much exhausted by the second attack, a few pink spots appeared at its close, and the spleen was much enlarged and tender: rallying was fair, the chief complaint being pains in the calves on movement. This man's wife and young family were admitted with fever from the same house; none showed the same critical depression: the nurse and clerk of the ward they were in caught the disease, and both of them died in a few days.

CASE XIX.—M., 40, admitted towards the close of supposed first relapse: m. t. 102° , p. 104; e. t. 99° , p. 92, feeble but regular, is perspiring, sweat acid in reaction: next day m. t. 94° (estimated), p. 60, feeble, regular; tongue brownish, moist, no headache or pains, but simple weakness; the fever had subsided in the night with much sweating, no delirium—heart's action extremely feeble, impulse not perceptible, first sound hardly heard at apex—a remarkable state, the body being like that of a corpse, the skin shrunken, wrinkled and clammy; the man is placid, can move the body about and speak audibly: pupils rather contracted; the spleen seems smaller than normal: breath cool, temperature of mouth $94^{\circ}2$; it is difficult to conceive where the blood has receded to (private notes). The stomach was irritable, not retaining liquids; a few pink spots on the chest: e. t. $95^{\circ}4$, p. 60, small but regular; vomiting continues, no urine passed. Next day m. t. $97^{\circ}4$, p. 76, vomiting ceased, no stool for four days, urine 14 ozs. in the last 12 hours, deep yellow colour, clear, acid, 1022, no albumen, chlorides $\frac{1}{4}$ vol.: e. t. $97^{\circ}4$, p. 70—thenceforward slow but continuous rallying, until another pronounced relapse set in, at the close of which the crisis was as marked and attended with delirium: convalescence fair.

In contrast with these cases were a few others with equal or greater fall, yet even fewer signs of suffering:—M., 33. Crisis in the night with decline of t. $10^{\circ}2$, p. 60, yet pupils normal, the senses alert, muscular powers retained and mind only a little drowsy: next t. $95^{\circ}6$, p. 64, r. 24, and no change, but the advent of pains in all the joints. M., 28, a muscular subject, showed a fall of t. 11° and of p. 72 in 16 hours (probably the t. was lower than 94° F.), and yet very slight inconvenience beyond depression. M., 22, was much distressed and very low at e. with t. 103° , p. 120, r. 58 (? acme), the change next m. being to t. 95° , p. 80, r. 50, shallow; and the state resembling that termed 'algide,' yet no particular general symptoms.

Pseudo-crisis in the Relapse.

CASE XX.—M., 24, condition, an asthmatic subject: admitted apparently at critical fall of invasion-attack, some feverishness and bronchitis ensuing: on 7th day the relapse set in abruptly, and lasted other 7 days, ending abruptly with a fall of 9° : during this attack the fever was well sustained until the 6th day, when a pseudo-crisis occurred, followed immediately by a return of fever, and next morning the true or final crisis. Although the t. was high (mean max. 105°) throughout and the p. quick, yet the man suffered but little distress; there was some pulmonic and splenic congestion and pains in the limbs; spirilla in the blood only indicated in fresh specimens, though distinctly seen in the prepared ones. 5th d. e. t. 105° , p. 150, no sweat or headache, slight thirst, tongue coated and moist, slight cough, spleen doubtfully implicated, pains in the arms, calves, and knees, no stool, no eruption, he is weak: spirilla not quite so common as in the morning (at t. $103^{\circ}6$) and vary in size. 6th day, m. t. 7 A.M. $97^{\circ}4$ (i.e. a decline of $7^{\circ}6$, and two hours later it was 96° , being a decline of 9°), p. 110, full, soft; the fever left at 4 A.M., with much sweating; now the face is perspiring, no great depression, he can just stand upright, no distress, headache or thirst, tongue white and moist, slight cough, no sputum, spleen doubtfully charged, has pains in the calves, one stool, no eruption: heart's action weak and second sound pronounced: it is stated that this cold, depressed state lasted until the sudden return of fever at 3 P.M.; a few spirilla were found of the usual aspect. E. t. $104^{\circ}6$, p. 160, slight chills preceded this exacerbation, no headache, much thirst, giddiness, some uneasiness in the chest and epigastrium, no splenic uneasiness, bronchitic cough returned, skin moist, intelligence good; the spirillum is still present, though less easily found than in the morning; the fever left in the night with much sweating; m. t. $95^{\circ}6$, p. 108, fair volume, regular; much depression yet no distress, the body seems shrunken, the mind clear, spleen felt; not a trace of the spirillum could now be found in the blood, there were many pale cells, some being large and with a large nucleus: prompt and unchecked convalescence next ensued.

Here by employment of the microscope I became aware that the fever had not ended with the first fall of temperature, although appearances were not unlike those of true crisis: in several other cases by the same means I was enabled to predict the end, whilst appearances were still of uncertain import.

Premature and repeated Depression.

CASE XXI.—M., 30, was admitted at close of invasion-attack and quickly rallied. The relapse was distinct, defined and sustained for 4 days, the crisis being prolonged for 3 more: temperature till the acme almost steady at 102° , the pulse at only 100: blood-spirilla many: there was much irritability of the stomach and also diarrhoea: last day of fever m. t. $104^{\circ}2$, p. 100 small, soft, regular, skin dry, no headache, 4 stools passed with straining, frequent vomiting, tongue brown and dry, thirst, no sleep; the blood contained numerous active spirilla and many white cells; at this time, which seemed to be near the acme, the aspect of the patient was compared by me to that of incipient cholera, the depression being extreme, eyes sunken, and though the trunk was warm, yet the extremities felt cold: e. t. (axillary) $102^{\circ}4$, p. 108, skin dry, 5 stools, no more vomiting: he has rallied a little with the decline of temperature, yet the limbs are cold, pulse hardly perceptible and heart's sounds hardly audible, respirations 40 per min. shallow. What was this collapse in the midst of high fever owing to? Cardiac debility was the most prominent feature (private notes). Next day the fall continuing m. t. $98^{\circ}4$, p. 96, no sweats, but sudamina in axillae, 4 semi-fluid stools, urine free, hiccup has come on, eyes yellow, no eruption, some headache, thirst; no spirillum in the blood; the depression was not so marked, though the temperature had declined. E. t. (axillary) 98° , p. 98, very feeble, limbs cold, no stool since morning, vomited once, no sweats; the man is in a state of collapse and remains so; urine said to be scanty; he denies having passed any for two days: the aspect is that of partial asphyxia. Next day, t. $97^{\circ}4$, p. 96, skin dry, no stool; hiccup continues, disturbing him at night, when he was somewhat delirious; now he is at times sensible; still no urine, bladder not distended, no urinous smell of breath; some pain around the umbilicus: e. t. $95^{\circ}6$, p. 82, no

sweats, or stool; dry cups applied to the loins at 10 A.M. and at 1 P.M. urine passed, 25 ozs., pale; tongue still dry and brown and irritability of stomach persists, with hiccup, the collapse is more marked, he yawns and stretches himself. Next day: m. t. $95^{\circ}2$, p. 82, small, soft, regular: no stool or vomiting, delirium continues, and the prostration: pupils of normal size, no distress or dyspnoea and he takes a little food: urine plentiful, several ozs. being passed at a time, sp. gr. 1010, pale, acid, no sediment, turbidity on heating not removed by nitric acid, chlorides diminished: e. t. $95^{\circ}6$, p. 84, skin dry, tongue brown in middle, white at sides and tip, moist, hiccup less, no delirium: next d. t. $96^{\circ}2$, p. 80, the rallying was slow, no sweats; three days later desquamation of the forehead was noted: the convalescence was eventually good.

Lysis.—Subsidence of an uncomplicated spirillar attack at a slow rate, either continuous or interrupted, though rarely seen in Bombay, yet claims attention from the unusual severity of the cases exhibiting this mode of defervescence. The phenomenon is probably not so simple as might appear, and in this place I shall consider only the plainer instances, reserving the obscurer for the Chapter on Pathology. Judging from the clinical Charts, there are many forms intermediate between 'crisis' and 'lysis'; and, for convenience, I assume that when specific fever occupies not less than 2 days in its main decline, it may be said to subside by 'lysis'; usually the time so occupied is longer than this. The blood-spirillum is sometimes seen throughout; at other times the organism disappears at the beginning of fall, not again to be visible in its subsequent course. Temperature and pulse at lysis are separately discussed.

Frequency.—So many fresh attacks not being brought to hospital until near their end, I am unable to state definitely the proportion of lytic terminations; amongst survivors about 10 per cent., and among casualties at least twice as many, of the cases displayed some degree of lysis. It was rarely seen in both invasion and relapse, and very rarely in recurrences alone. A tendency to gradual defervescence either at beginning or at end alone of the main fall is not uncommonly seen, and it is usually accompanied with symptoms in some degree or kind severe.

The lytic decline may be tolerably uniform, though interrupted by brief exacerbations; sometimes it is remittent throughout: its whole depth is shorter than occurs in the critical fall, and I have only once seen a subsidence of 8° ; commonly the range is much less than this. Its duration varies from 3 or 4 days (the more frequent estimated length) to 5, 6 or 7 days: in the longer instances the date of reckoning may be arbitrary. How such attacks begin and their total duration have not been sufficiently ascertained, few patients being seen early enough: I know that an invasion may begin abruptly as usual, and its lytic decline be effected through depression of the latter half of the attack; yet generally it was most probable there occurred an actual prolongation of the febrile state during this gradual defervescence. There may be a history of final *perturbatio* corresponding to the febrile acme, though usually not so. I have remarked that in these attacks febrile exacerbations preceded by chills, but not followed by much sweating, were not uncommonly mentioned as occurring throughout at night. In the instance of survivors, rallying at the end may be prompt as usual or only somewhat delayed.

The subjects were generally young adults; occasionally the old: lysis seems proportionately rather most frequent in women, young and old: debility may be a predisposing influence; Mussulmans were more numerous than Hindus; and cases were seen chiefly after the height of the epidemic.

Whilst there are no special symptoms attending fall by 'lysis,' yet debility and a tendency to prostration were invariable; the typhoid state supervening much more frequently than usual: typhus characters were sometimes seen, yet not exclusively here. Headache was not excessive, nor the splenic complication; epigastric (especially) and hepatic implication were common and marked; jaundice, hiccup, vomiting, dysenteric diarrhoea, were not unusual; febrile delirium was frequent; sleeplessness, injection of the eyes, retention of urine, involuntary evacuations, hyperæsthesia of the body or a sense of heat or formication have been noted: also giddiness, pains in the limbs, œdema of the feet, sore throat, cough, partial sweats: urine not particularly changed: the heart's action was invariably feeble, even when the pulse seemed full.

As most patients recovered completely, though slowly, these symptoms are referable to organic lesion which is transient.

After invasion, the relapse may follow at intervals not exceeding the mean limits, and be either mild or severe, or it may terminate with death from incidental hæmorrhage. Rebounds of temperature after defervescence by lysis, seldom occurred in uncomplicated cases. In survivors, definite complications were rare, though doubtless some of the functional or intercurrent lesions peculiar to spirillar infection, were more pronounced than usual—those of the liver and epigastrium (including stomach with left hepatic lobe and the semilunar ganglia?) being particularly indicated.

Symptomatic fever may obviously interfere with critical fall, so as to induce a more gradual decline; and that of pneumonia was best apparent in this connection.

Diagnosis.—When the previous history of an attack is known, its mode of decline becomes matter of observation; the presence of local complication might be suspected.

Prognosis.—It seems to me remarkable that even a tendency to lysis should be usually accompanied by severe symptoms; and I should anticipate that an attack characterised at its close with signs of prostration and of abdominal lesion, would not terminate in prompt convalescence: in 5 of 6 typhus-like cases lysis was indicated; in 8 deaths from accidents subsequent to invasion-attack, 6 showed lysis at invasion; and it seemed to me that, evident complications apart, whenever the typical crisis was departed from, the patient's sufferings were liable to be increased.

Relapsing Fever: Decline by Lysis at Invasion-attack.

CASE XXII.—M., 49, condition fair, seized in hospital, admitted on 4th day of invasion: the fever is said to remit: there is much headache, eyes injected, general pains and tenderness of the whole body, with irritability of the stomach, some bronchitis behind, considerable enlargement and tenderness of the liver, also tenderness of the spleen and epigastrium: urine scanty, high-coloured, acid, 1022, albumen a trace: m. t. 99°·8, p. 88: e. t. 102°·6. Fifth day—m. t. 101°·6, p. 96, tongue coated, moist; acute hemicrania of r. side, muscular and arthritic pains: liver the

same; bowels regular, vomiting in the night. Many active spirilla seen in the blood this day, as well as yesterday. E. t. 103° . Sixth day—m. t. $101^{\circ}4$, p. 112, weak, small, slight yellowness of eyes, no more vomiting, great tenderness with enlargement of liver and spleen, skin dry, pains the same, one consistent bilious stool, slept a little (sedatives, local fomentation, quinine): e. t. $102^{\circ}4$, p. 116, liver still very tender, urine retained, $4\frac{1}{2}$ ozs. drawn, high-coloured, 1028, acid, no albumen. Seventh day—m. t. $98^{\circ}8$, p. 96, weak, regular, urine 8 ozs., passed at night, high-coloured, 1020, acid, no sugar, no albumen, chlorides $\frac{1}{2}$ vol., on standing a rather copious and very dark precipitate resembling blood but cleared by heating and the addition of liquor potassæ (ammonia then evolved), and under microscope shown to be urates with much biliary matter, some masses being of the form of urinary casts: no uneasiness in the loins. Abdomen, including epigastrium, full and tender, skin dry, tongue tending to dryness, two stools (after aperient) bilious; there was some sweating last night. E. t. $102^{\circ}2$, p. 120, full; no sweats in the day, epigastric uneasiness continues, urine 14 ozs., high-coloured, 1015, no sediment, no albumen, bile-acids and pigment present, no stool, tongue dry in centre, pains in r. shoulder considerable, injection of conjunctivæ and oppression, but no headache. Eighth day—m. t. 97° , p. 80, full, weak. Urine 7 ozs. in the night, 1015, acid, amber tint, no albumen: epigastric uneasiness continues, no pain in r. shoulder, tongue moist, some sweating last evening, no headache, one stool, bilious, eyes yellow, slept a little, pains in hips, abdominal fulness less but tenderness not less, he is giddy and very weak. E. t. $98^{\circ}6$, p. 88, feeble, one stool, urine free, skin moist, slight thirst, tongue white, furred, moist, no headache, giddiness continues. Ninth day—m. t. 95° , p. 68, feeble, regular, tongue brown in middle, cold, great thirst, one scanty yellow stool, slept a little, general tenderness and puffiness of abdomen, the left lobe of the liver is enlarged, projecting in the epigastrium; the patient is semi-collapsed and complains of dryness of the mouth, no delirium or vomiting. E. t. 98° , p. 80, tongue brown, moist, one liquid highly bilious stool (after aperient), tenderness and puffiness of abdomen continues, urine free, 1012, no albumen, chlorides $\frac{1}{4}$ vol., bile-acids and pigments present: he has a slight headache and is still depressed: pains in r. shoulder and hips the same (warm applications to abdomen, stimulants). Tenth day—m. t. $95^{\circ}6$, p. 52, feeble, regular, tongue brown, moist, two highly bilious stools, much thirst, some sleep but now he rambles in his talk, much depressed, slight headache, pains all over, conjunctivæ yellow and much injected, nervous prostration; no eruption now or previously. Urine free, 30 ozs. in the night, 1012, acid, no albumen, chlorides $\frac{1}{4}$ vol., bile-acids and pigments present. E. t. 98° , p. 80, better volume, tongue the same, three liquid yellow stools, thirst, abdominal fulness and tenderness the same, general pains less, no headache. Eleventh day—t. $97^{\circ}2$, p. 80, full, regular, tongue dryish and brown at the back, two stools, slept a little, pains less, jaundice more evident, urine 34 ozs., 1010, no albumen, thirst continues. E. t. $99^{\circ}2$, p. 96, three stools, thirst, no headache, abdominal uneasiness less, urine passed frequently in small quantities, tongue moist and clearing. Twelfth day—m. t. 98° , p. 84, feeble, tongue brownish, dry, one stool, much thirst, no headache, abdominal signs diminishing, pains in joints continue, slept, urine free. E. t. $99^{\circ}8$, p. 100, feeble, tongue brown, moist, much thirst, no headache, drowsiness, abdominal uneasiness less, and so the pains in limbs. Thirteenth day—m. t. 99° , p. 100, soft, tongue clearing, much thirst, slight headache, drowsiness, urine free, abdomen easier, the liver still extends two inches lower than normal. E. t. 101° , p. 92, no headache, tongue clearing, epigastric fulness and tenderness said to have increased, the liver reaches $1\frac{3}{4}$ in. below the costal margin, spleen not enlarged, urine passed with stools, slight scalding, pains in limbs and joints, the left eye is inflamed (ophthalmitis), no sweats, thirst slight, one scanty yellow stool. Fourteenth day—m. t. $98^{\circ}8$, p. 88, no headache, tongue dry in dorsum, slept but little, slight thirst, one stool, urine free: there is still some uneasiness over the r. costal cartilages below, but less enlargement of the liver. E. t. 98° , p. 88, no headache, free sweating during the day, slight thirst, bowels free, tongue moist, clean, abdomen easier. Fifteenth day—t. $98^{\circ}2$, p. 80, no headache, eyes less inflamed, no uneasiness in the body generally: e. t. $98^{\circ}6$, p. 72. Sixteenth day—m. t. $98^{\circ}6$, p. 52, no headache, pain in sterno-clavicular joint, slight hepatic uneasiness, no more enlargement, spleen normal, bowels regular, urine free, eye improving: e. t. $98^{\circ}6$, p. 80, feeble, regular. Seventeenth day—m. t. $98^{\circ}4$, p. 84:

e. t. $98^{\circ}6$, p. 88. Eighteenth day—m. t. $98^{\circ}4$, p. 80, slight cough with mucoid sputum : e. t. $99^{\circ}8$, p. 88, full, regular, slight headache, skin warm, moist, slight tenderness over liver, abdomen rather full and tender, conjunctivæ slightly injected and yellowish, no pains in joints, slight cough and thirst, tongue coated, moist. Blood examined, spirillum not found, though relapse had set in.

Nineteenth day—m. t. $99^{\circ}6$, p. 80, full, regular, no headache, skin dry, no pain in abdomen, or body generally, eyes yellowish, much thirst during the night, cough less, tongue clean, moist, bowels regular, urine free, appetite good, some giddiness : e. t. 103° , p. 100, full, regular, no headache or pains in the body, skin moist. Twentieth day—m. t. $102^{\circ}8$, p. 100, full, regular, urine free, bowels regular, appetite indifferent, there is tenderness and fulness of the abdomen and also of the liver and spleen, some pains in the joints of lower limbs, some thirst, skin dry, some headache, slight sweats during the night. Blood-spirilla present and active. E. t. $104^{\circ}6$, p. 120, weak, slight headache, no pains in the joints. urine free, slight thirst, both liver and spleen are enlarged downwards, the abdomen is full and tender, conjunctivæ yellow, bowels regular, sweats in the afternoon, tongue coated, moist. Twenty-first day—m. t. $102^{\circ}2$, p. 108, weak, slight headache, pain in loins, urine free, no stool, much thirst, nausea and hiccup during the night, no sleep ; eyes not redder, abdominal uneasiness the same, free perspiration at night, tongue coated, moist, skin soft, and moist over the head and upper half of trunk, no eruption, no shoulder pain or cough. E. t. $106^{\circ}4$, p. 128, weak, regular ; headache severe and pain in back, no thirst, skin dry, tongue coated, moist, abdominal uneasiness remains, slight cough, sweats on the forehead. Blood-spirillum not seen at this acme, 4 P.M. : 5 t. $102^{\circ}6$, 5.15 t. $100^{\circ}6$, 6 t. $100^{\circ}4$, 8 t. 99° . Twenty-second day—m. t. $97^{\circ}6$, p. 92, feeble, free sweats since last evening at 5, no headache or pains, is weak and giddy, no stool. E. t. $97^{\circ}4$, p. 84, slight thirst, still some enlargement and tenderness of liver, spleen unaffected and abdomen free from uneasiness : sweats since 1, vomiting, some jaundice, conjunctivæ less injected, no pains in shoulder, has a tingling sensation in both arms and legs. Next day—m. t. $97^{\circ}4$, p. 80, weak, no sleep, no sweats, is exhausted, liver and abdomen easier, slight pain in left shoulder : e. t. $98^{\circ}4$, p. 88, giddiness and slight hepatic tenderness. A slight pain in epigastrium and some thirst on the following day ; and two days later he begged for leave. There was no recurrence of fever, and some weeks later he returned to duty in the medical ward, where he originally contracted infection.

The chart of this case is copied in Plate V., No. 11 : the above narrative will serve also to illustrate some minor symptoms of fever attended with mild complication.

Lysis at Invasion with Cerebral Hæmorrhage : Death.

CASE XXIII.—M., 55, famine-subject with bronzed, clammy skin, emitting a musty smell, depression extreme ; brought to hospital by wife and daughter, with fever of eight days' duration, e. t. $102^{\circ}4$, p. 126, no headache, hepatic tenderness, no eruption ; for 4 days the fever then declined in a remitting mode to $96^{\circ}6$, p. 88, no regular sweats throughout, no eruption, cough, thirst, vomiting, headache (diminishing), no delirium, slight jaundice, pains, tongue becoming dry and brown, at the end he was very low but intelligent, no pains, skin soft, aspect peculiar, eyes yellowish, occasional hiccup, pulse weak but not small. Blood examined daily and spirilla seen until the last. A febrile reaction now set in with early typhoid symptoms, œdema of the feet ; the t. rose gradually for 3 days to $104^{\circ}4$, p. 120, when he became insensible, not comatose quite, and 2 days later he died : at autopsy cerebral hæmorrhage with surrounding inflammation. No spirillar contamination during this secondary fever ; previously no symptoms of cerebral lesion, and other cases show such lesion not invariable in lysis. The man's wife had had fever ; the daughter (aged 8) underwent a pronounced relapse with copious epistaxis at the end, prompt rebound and death by thrombosis of femoral veins.

SPECIAL SYMPTOMS.

1. **Headache.**—According to my personal experience the headache in spirillum fever seems not to differ in general character from that accompanying malarious fever, or even non-febrile derangements : it is commonly and chiefly frontal, sometimes extending to the temples ; seldom general and very rarely limited to the occiput : when temporo-occipital it may become blended with the pains in the nucha, so frequently present.

It is perhaps the commonest of all symptoms, being practically invariable in some degree, at some period of the febrile attack ; whilst it is nearly equally absent during pre-febrile and post-febrile stages. It is, however, included amongst the occasional premonitory symptoms ; and if not it usually supervenes with the earliest rise of temperature, increasing as fever progresses and promptly subsiding with the crisis : in 70 p. c. of invasion-attacks it was a prominent symptom, though less so amongst the weaver class, whose fever was of low type ; during the relapse, it recurs (as a prodrome occasionally) with other symptoms of fever, varying with them and not being so often prominent as before (50 p. c. of first relapses) : it again reappears with subsequent attacks. Patients described the feeling oftenest as 'great heaviness,' sometimes as 'splitting' or 'throbbing.'

Varying in intensity but usually very severe, and complained of especially as preventing sleep ; its duration may be remarkably limited to that of the pyrexial state, and its severity also corresponding thereto, at remission (specially with sweats) and exacerbation, until the acme, when in some cases the headache was described as ceasing (no delirium) ; often, however, then becoming acute : with commencing decline of fever, the headache may at once subside : in some few cases, this symptom (like thirst and pains elsewhere) only slowly declines after the crisis ; and as a rarity due to either idiosyncrasy or untruthfulness, it may be disallowed by a patient during the earlier days of his attack, coming on only towards its close, although the pyrexia and blood-state were previously characteristic. Judging from their aspect, infants may have had this symptom (so also the ailing quadrumana) ; children complained of it, as well as adults : perhaps men suffered most ; yet not the weak more than the strong, or *v. v.*

In detail, correlated states were as follows :—the scalp offered no peculiarity, conjunctival injection and the state of the pupils no fixed relationship ; the foul, moist tongue oftener with pronounced headache than the dry-brown ; no definite concurrence with state of liver (or jaundice) or spleen, or epigastrium (or nausea, vomiting), or with constipation ; nor any with visible state of the blood. Its general connection with pyrexia was shown by the consentaneous remissions and exacerbations throughout : yet not its entire dependence, for it might (with other usual symptoms) be absent at first, or increase when pyrexia does not, and when this attains its acme, might temporarily or permanently diminish and cease : lastly, in 8-10 p. c. of ordinary cases, headache only

gradually subsided in 2 or 3 days after crisis. Thirst, muscular and arthritic pains were the other closest, yet not invariable, attendants.

In the severer attacks marked at the close by delirium, headache was usually severe, and it might cease just before the delirium came on (*e. g.* at acme), yet not seldom these two symptoms alternated, the delirium being present only at night : obviously it is impossible to ascertain with certainty their actual co-existence at any moment, and my impression was that the relationship here pointed to a common origin rather than to sequence or substitution. Several patients were admitted at acme with severe symptoms, who had had the actual cautery applied to temples or vertex of head, for relief of this symptom.

Headache during secondary fever varied, being sometimes slight when pyrexia was high : and it was not a prominent character in fever symptomatic of local complications.

As to its diagnostic import, its marked presence during the late epidemic certainly invited special attention in particular cases. A particular point was the occasional occurrence of headache with hardly any other ordinary symptom about the date of an expected relapse, when it might be interpreted as a sign of suppressed or latent recurrence.

Its prognostic value seems inconsiderable, my notes showing it to be less common or pronounced in cases dying both during and after invasion-attack, than amongst survivors at same periods : when death occurred from specific fever alone, headache was not, apparently, a pre-eminent symptom ; and it was subordinate amongst the signs of fatal complication. Its persistence after the fall usually accompanied the decline of fever by lysis, in cases often serious.

There is no precise evidence of the anatomical conditions pertaining to this symptom. Probably cerebral congestion is present in many cases, such, *e. g.*, as those relieved by free epistaxis, or by artificial depletives and derivatives : however occasioned, this head-symptom must be commonly functional in character.

The cases quoted in detail above illustrate the often variable connection of headache with pyrexia and other signs : that of the woman, No. IX., shows the cessation of this symptom just before the critical perturbation. An adult male relative of hers declared its absence at the acme, with t. $105^{\circ}\cdot6$, p. 132, sweating having begun. M., 22, on sixth day of invasion had A.M. t. $102^{\circ}\cdot4$, p. 112, no sweats and much headache : P.M. t. $103^{\circ}\cdot8$, p. 120, skin dry, no headache : next day (the last) A.M. t. $102^{\circ}\cdot8$, p. 116, much headache, many spirilla ; P.M. t. $103^{\circ}\cdot6$, p. 120, much headache, no spirillum present, and crisis following : at the relapse this symptom was late in appearing, and did not subside until the fall. M., 25, on last day but one of invasion, A.M. t. 102° , p. 106, much headache and sleeplessness from it ; P.M. t. 104° , p. 120, slight headache, with delirium following at night : last day A.M. t. $102^{\circ}\cdot4$, p. 110, much headache, and immediately after the critical perturbation and fall.

From such instances I am unable to deduce any fixed relationships of headache with other symptoms ; the general connection has been stated, and in future enquiries the influence of any drugs administered would require to be eliminated. I have considered this point in the above remarks.

2. Muscular, Arthritic, and Osseous Pains.—Though these make up one of the symptoms of a subjective character, not open to direct observation, yet their reality was attested by many visible signs of distress.

They form part of a widely distributed series of perverted common sensations, and are here separately specified from their frequency and prominence in the spirillum fever.

These pains are probably always present, and they were more or less complained of as severe, in upwards of 70 per cent. of ordinary cases at febrile stages : amongst fatal cases they were not more frequent.

Not uncommonly aching pains in the limbs or trunk precede the onset of fever at a time when spirillar contamination may become visible : they promptly augment (or begin) with the pyrexia and increase during progress of the attack until the end, few cases being then free from pains ; in great part they cease or diminish with the critical fall, but may persist, and troublesome pains may even come on only after the crisis. It would seem they were finally rather commoner and more persistent during average first relapses and succeeding apyretic periods, than during the first attacks and apyretic intervals ; being marked in 45 per cent. of cases at fall and 50 p. c. for the next few days amongst an ordinary series of relapses, as compared with 20 p. c. and 30 p. c. respectively amongst ordinary invasions.

Native patients referred these pains either generally or locally to muscles, fasciæ, ligaments, joints and bones of trunk or limbs, alone or together : they were described as an aching, soreness, boring, gnawing, a sensation as if beaten, or not uncommonly as if the bones were being 'broken' : the joints were highly sensitive, or stiff, or felt as if distended when there was no visible swelling. The loins and nape of neck (fleshy parts of the trunk), lower limbs, calves, thighs, knees, shin, femur ; seldomer the shoulders and elbows, deltoid, muscles of arm and fore-arm : less often wrists and ankles, and still more rarely the terminal structures of the extremities. In the less mobile bones and joints the pains might seem to be spontaneous, being however always increased by direct pressure ; generally in the muscles and smaller joints they were elicited only upon exertion and in proportion to the effort made. Sometimes their character was so acute as to keep the sick quiescent as if paralysed, and render them sleepless. The red muscles acting involuntarily for respiration and circulation may not have been the seat of such pains ; yet occasionally I thought the suffering evidently entailed by the act of coughing might be seated in the diaphragm and other abdominal muscles, and that the cardiac distress at high fever might be seated in the heart. Apparently febrile and non-febrile pains were alike in character.

There were many degrees of severity, from the mild to an almost exquisite intensity.

Commonly the pains appeared with the fever, augmenting and declining with the changes of pyrexia, subsiding at the crisis for good, or persisting 3 or 4 days longer, pains in joints being specially apt to linger ; sometimes they were more intermitting and attacked isolated parts in succession ; and occasionally they made their first appearance or were most intense, at deep critical stages. Whilst the trunk was seldom spared, the lower and upper limbs alone, or mostly, might be successively involved at invasion and relapse ; and the trunk during pyrexia, afterwards the limbs.

Neither sex nor age seemed to be specially concerned : and the

weak, cachectic or emaciated seemed to suffer as much as the more robust generally, though I think the severest pains of all may have happened to muscular subjects, some of whom on admission had cautery-marks along the spine (both sides) previously made to relieve these pains. Amongst Mussulman weavers in 1878, this symptom if not more pronounced was perhaps commonest and most lasting: their type of fever was 'low' (see also the case detailed above, No. III.)

So far as appeared, these pains were not essentially related to other particular symptoms, or state of blood or urine: nor to intensity of pyrexia, depth of fall or sequel: it was observable that whilst headache, thirst and special abdominal signs augmented at the acme of fever (during invasion chiefly) these pains did not appear so to increase: and at periods of exceeding critical prostration they might come on, temporarily, as delirium sometimes does.

The diagnostic import of this symptom was sometimes obvious when patients presented themselves after the crisis of fever: here the previous history would guide with the general circumstances of the case, and also when scorbutic, syphilitic, malarious and rheumatic pains might be suspected; visible local change, nocturnal exacerbations and diathesis-marks being absent in the uncomplicated spirillum-fever cases.

The prognostic import was not strictly clear: their occurrence as prodromes is worth recollecting.

It did not seem that coarse anatomical changes arose in parts the seat of pains, and as well the generally transitory duration of the symptom pointed to its functional character, or if organic to a highly localised lesion speedily compensated after fever.

In my own case, first attack, there were some brief cutaneous red patches over the ankles and shoulders, where tenderness existed: no rheumatic diathesis.

Occasionally an uncovered joint, as the wrist, the seat of pain, was found on measurement to be slightly swollen, but this without evident relationship to the symptom.

Whilst the pains were often generally symmetrical, they were very seldom accurately so, and not rarely they were one-sided when most marked. Their seat at insertion of tendons of the patella was noted in case No. IX.

Some varieties are here noted:—

Pains only at Critical Prostration.

CASE XXIV.—M., 33, health fair, admitted on 5th day of invasion, had no pains in limbs at the acme (t. $104^{\circ} \cdot 2$, p. 130), or at once with the extreme critical fall, t. at nos: 94° , p. 70, when a few pink spots appeared: the body-heat did not rise more than 1° for 24 or 30 hours, and for nearly another day only to $96^{\circ} \cdot 6$, p. 72, when general aching pains came on, and fresh eruption: on the third day after crisis m. t. 98° , p. 70, resp. 30, no headache or abdominal tenderness, extreme pain in shoulders, elbows, (most) wrists, knees and ankles, no pain in the loins, hips, or smaller joints of hands and feet: suffering limited to the joints, is increased on pressure and movements and so prevented sleep and incapacitates him for getting out of bed: otherwise he does not seem particularly weak: the bones, muscles and skin of the limbs are not tender: the left wrist seems a little swollen: syphilis denied. Sedatives and liniment. Next day the pains were less in morning and had ceased at evening.

M., 24, at end of Invasion jaundice and lytic decline, general aches and much hepatic tenderness; 2 days later sudden nocturnal onset of pains and stiffness on right side of body, so much limited as to suggest a hemiplegic character: no head-symptoms and in 3 days prompt convalescence.

In a third muscular subject (M., 28) relapsing attack, the pains were not limited to the joints of the limbs, but implicated muscles and bones, and they greatly increased as the man began to rally after a sinking of the body-heat to 94° (possibly lower still) : in three days longer they had quite ceased.

3. **Thirst.**—The craving for drinks (water being the beverage commonly demanded by native patients) was practically invariable during fever; and although as a subjective symptom it was not open to precise recognition, yet the evidence of its urgency was sometimes so striking as to claim particular notice.

Beginning with or very soon after the advent of pyrexia, marked thirst increased with its progress, often being less with the brief mitigation which precedes the acme of attack, then increasing again at acme, and subsiding promptly with the crisis in at least half the total instances. It was at least equally common in pronounced relapse as during first attacks, though not so urgent generally. After crisis thirst in some degree persisted not rarely, for two or three days. To the best of my knowledge, it was not a symptom premonitory of specific fever.

Amongst modifications of character was the occasional dislike of native patients to cold water (iced water was seldom relished), which they fancied to excite chills, set the teeth on edge, or otherwise cause discomfort greater than relief, or apprehension of hurt : these fancies struck me as being possibly peculiar to the spirillum fever. At close of attack, thirst was often excessive ; having previously followed, tolerably regularly, the exacerbations and remissions of pyrexia. It was manifested at all ages and by both sexes : was not limited to the weak or strong, the starved or well-nourished, or to one period of the epidemic : it was most persistent, if not most intense, in the subjects of low type of fever.

In the absence of controlling data, not much reliance could be placed upon the records of thirst as concurrent with many other symptoms : its relationship to pyrexia seemed clear, yet occasionally this was contra-indicated, even decidedly, as for example at beginning of relapse, or near end of invasion, when the blood-contamination was invisible or slight and subsiding : I have, however, noted the absence of thirst with even copious presence of the spirillum—absence here doubtless meaning no urgent wish for water ; its concurrence with epigastric uneasiness or hiccup was not invariable ; it was usual after skin or nasal hæmorrhage : and attended the defervescence by lysis, commonly also rebounds or secondary fever, and symptomatic pyrexias.

The persistence of thirst after crisis may correspond to that of muscular or arthritic pains, and like them, it may even increase for a brief period at this stage : I could not trace a fixed ratio here of demand for drinks and copiousness of sweating or other drain of liquids from the blood, yet certainly the most copious imbibitions of water I saw were those of a man, after one or probably two relapses, and these apparently to satisfy a natural craving—as a consequence the urine was much diluted and weight of body nearly stationary, plenty of solid food being taken.

Thirst, however, may persist at crisis without there being much sweating ; a dry feeling in the mouth being noted also, with no diarrhoea and the abdomen retracted.

This symptom had no prognostic value alone; in fatal cases it was usually pronounced, yet not more so than some other phenomena, and complications might be present.

4. **Appetite.**—Though usually absent during fever, either a desire for solid food or sense of hunger might then be expressed by patients, who always had the means of gratifying their appetite, yet might be deterred from doing so by inconvenient or unpleasant consequences. A curious distinction was occasionally made by the sick between the feeling of hunger and the immediate desire for food, there being a disinclination to eat from want of 'taste,' or want of 'relish,' such as imparted by salt (though this condiment was not wanting in the food); and it is likely some patients tried to eat in order to overcome the great debility and exhaustion experienced during fever.

As a rule, however, appetite was wanting, and herein a great contrast to the frequency of thirst. In an early series of 30 selected cases, chiefly of famine-immigrants, this symptom was present and attracted attention in 3 during invasion-attack from 4–7 day, whilst fever was high, and vomiting, headache, pains and dry tongue prevailed: it was seldomer recorded amongst weavers with a low type of fever, or in the last series of attacks amongst town residents.

I have noted the occurrence of good appetite near the end of a relapse, just before probable acme and sudden death by cerebral hæmorrhage: yet it could not be said the symptom was common at any time in recurrent attacks.

At the critical fall, the desire and capacity for food often returned as promptly as other signs of relief, and for some days the appetite might be excessive: striking examples of this kind were seen, and not rarely hearty feeding continued until the day (or possibly the hour) of relapse.

Amongst the contingencies modifying recovery at crisis, want of appetite for a brief time was occasionally noted.

Significant was the even ravenous appetite of some typhus-like cases, on cessation of fever: restriction of food had sometimes to be insisted on. The half-starved were not necessarily the greatest eaters.

5. **Vomiting.**—During involuntary emesis glairy mucus and diluted bile of varied greenish hue was oftenest brought up; when irritability of the stomach was excessive even water could not be retained, and frequently patients complaining of hunger would not take even semi-solid food lest epigastric uneasiness should follow: more rarely was there a little blood in the ejecta: occasionally *lumbrici* were passed: eructations of gas were not common: milk previously swallowed came up curdled.

Vomiting was more frequent in some classes of cases than others: thus, amongst hospital patients admitted late at invasion-attack it was not noted in more than 10 or 12 p. c., and at relapse not more than 20 p. c. of those surviving; whilst in a series of 40 examples of contagion at the J. J. Hospital, seen mostly at early stages, it was noted in 75 p. c., and also in 6 of 8 other examples of contagion at the smaller hospital.

It occurs in both abortive and relapsing forms of fever, and at re-

lapses (even second) when marked, as often as at first attack : it may be seen at invasion only and exceptionally at a recurrence alone. Seldom mentioned as a premonitory symptom, it was noted from 3rd day onwards, or as first appearing with the acme (not usual) or the fall (not unusual) : during relapses it seemed to come on later and persist longer, or till the acme : after both febrile events, it might still last 2 or 3 days ; its occurrence was not limited to one stage of the attack, coming on with fever it might continue 3 or 4 days, with acme it usually ceased and with the fall it might last for a time : it was noted oftenest at night and attended either exacerbation or remission of pyrexia.

Vomiting in spirillum fever has the usual active character, and sometimes was so frequent and urgent as to cause much distress and weakness : usually it recurred 2 or 3 times a day, being either spontaneous or produced by the ingestion of food.

The most striking examples were seen amongst young men : infants might be free, and it was not common in females.

It occurred to the well-nourished and well-to-do equally, at least, with paupers : at all periods of the epidemic, and in both sthenic and asthenic types of fever.

It was not connected solely with degrees of pyrexia, or intensity of attack : nor was any fixed relationship evident with morbid conditions of liver, stomach or spleen ; and so far from corresponding with these localised changes, it did not invariably augment at acme or still less subside at fall : epigastric distension and tenderness might be present without vomiting or even nausea, whilst the act of vomiting might give rise to aggravated soreness.

It was only occasionally seen in the rebound or secondary fever.

Whilst noting this symptom in native patients, their habit of artificially irritating the fauces should be recollected ; and also the effects of incessant coughing.

Vomiting cannot be said to bear distinct prognostic import : in any degree amongst fatal cases generally, it was rather less frequent than among survivors : that there might be copious stomachal hæmorrhage without it, was seen in a case quoted below. Its occurrence in certain cases and not in others almost precisely similar, could not be understood : in the severer instances of defervescence by *lysis* and in those of *typhus biliosus* the symptoms when present offered no peculiar indication. It was not observed in the inoculated quadrumana.

Both symptoms and morbid anatomy indicate the possibility of congestion, small extravasations and probably of inflammation, as affecting the gastric and duodenal mucous membrane, which might be direct causes of vomiting : the excessive secretion of mucus and inverted flow of bile co-operating. An indirect cause would be cerebral irritation, though I have not here ascertained this clinically. Mechanical conditions noted were over-distension of the stomach with water taken to quench excessive thirst, frequent action of the diaphragm, as in urgent cough ; and possibly the direct pressure or irritation of an enlarged spleen, or enlarged liver—the left lobe especially, might be concerned, yet I am bound to add no demonstration of this was gained. With unusual irritability of the stomach, mere change of posture might induce the recurrence of vomiting, as was well illustrated in the case of a native student.

It was not very unusual to note specks and small streaks of blood in the ejected mucus, and it appeared to me that such instances were transitional to the rare 'black vomit' proper. Some gastric hæmorrhage was always a likely contingency, and may have been more frequent than reported; vomiting being only an occasional sign of it.

Nausea.—Not an infrequent symptom, either alone or preceding and alternating with vomiting: its local connection with state of stomach was not clear.

6. **Sweats.**—A tendency to sweating contemporarily with remission of pyrexia, is as common in this specific fever as in that due to malaria; and the peculiarly marked critical termination is attended with perspiration, more copious than in ague-fit. Both first and recurrent attacks end thus, and as a rule to which there are few exceptions, the skin becomes moist shortly before the temperature sensibly declines. In most cases the perspiration is so excessive that the clothes, and even bedding, become more or less saturated. A peculiar musty odour was occasionally perceived at this time, not apparently due to dirt: the sweat has a distinctly acid reaction in common cases: when collected (*e.g.* from the axilla) it was a clear liquid without sediment, always containing free granules or masses like micrococci and rods of bacillus or leptothrix, sometimes very long and slender; once a monad with cilia was found: there was never seen a sign of the spirillum, even when the blood and saliva showed it. The cuticle may become macerated.

Prior to the crisis, sweating occurs in the earlier remissions, especially of the relapse; also in the mid-fall sometimes seen, and in pseudo-crises of varying degree, which precede the *perturbatio critica*: sweats with fall of temperature may indicate the separation of specific and consecutive non-specific fever: they attend the post-critical paroxysms, also. Nine-tenths of crises are attended with copious sweats; yet there is much variety here as to exact time, amount and duration of the cutaneous flux, and that irrespectively of temperature-changes. For one, two or three days after crisis, partial or nocturnal sweating is not uncommon; but there is yet little accurate information on either subsidiary or main items of the phenomena in question: illustrations of the ordinary clinical records are given in the cases detailed above. The relationship of critical sweating to loss of body-weight has also been pointed out. When scanty, perspiration may be limited to root of neck, forehead, chest, axillæ and groin, palms or soles: when copious it is general: both sexes and all ages are affected. It is probable that with visible perspiration the temperature of the surface is more or less reduced; yet it does not seem to be due to such reduction leading to a condensation of humidity; and on the other hand, copious sweats are not the cause of loss of heat. It is even doubtful if there is any direct connection of mere sweating with fall of temperature; for in several of my cases the disproportion of the two phenomena, in excess or deficiency, was very apparent, and the induction of copious artificial perspiration did not lead to cutting short of fever. Copious sweating may be deferred until after crisis: but the possibility of partial transpirations at night being overlooked is obvious, and the skin may quickly dry.

The prognostic import of this symptom is highly contingent, excepting at the date when the invasion-attack usually ends ; and even then, the *perturbatio critica* may follow free sweating : here the state of the blood is the best guide. During relapses free sweating is common and more irregular. In fatal cases, sweating was hardly commoner than in survivors, excepting about the acme of invasion when casualties were commonest ; nor was it rarer than usual.

7. **Sudamina : Miliaria.**—This eruption was commonest late in the epidemic, and amongst the weaker weavers and town residents : it may have been sometimes overlooked, owing to darkness of the skin, minuteness of the vesicles and their limitation to covered parts of the body ; yet it was not nearly so frequent as to be characteristic. Nor when even abundant, was it always associated with particular symptoms, severity or stage of the fever ; and though commonest at the crisis (without any definite relation to copiousness of sweating), yet it has been noticed so early as the first day of a relapse. Perhaps the most abundant crop of miliaria I saw, which covered all the front of the trunk and extended on the upper limbs, occurred at the close of a brief specific relapse of two days, intercalated between invasion-attack and relapse proper : there had been no such crop at the first great crisis, and in the succeeding event there was but a scanty one. In some of the most pronounced critical phenomena, sudamina were absent ; and on the other hand scattered vesicles might be seen in the slight or partial sweats occurring while fever persisted. The site also varied ; usually when few, the vesicles were seen at the root of the neck, or on front of trunk ; when numerous, they congregated chiefly on front of abdomen, or chest, in the axillæ and groins ; less on front and inner aspect of arms and thighs, the flexures, and on outer side of fore-arm, back of hand ; about the knees and upper part of the legs : the crop was unusually symmetrical and sometimes seemed to follow lines (? nerves) on the limbs. The size of the vesicles varied from a pin's point to a split pea ; their contents generally clear, rarely opalescent (without severity of symptoms) or sanguineous : their duration varied from a day or two, to a fortnight ; successive crops might appear : when dessicated no sign was left, or a branny exuvium, seldom a more defined desquamation of the cuticle in flakes ; but once the skin was stained over the site of the vesicles, of a petechial tint.

I think this eruption was more frequent in spirillum fever than in others at Bombay, yet it was not limited to this ; and it had not, therefore, a true diagnostic import : its prognostic value, also, was not apparent.

From the fact of sudamina usually appearing with sweats and in similar localities, it may be presumed that a physical connection existed between these symptoms.

This sign was placed in my list of 'complications,' but its natural connection with cutaneous perspiration induces me to consider it in this place.

8. **The Tongue.**—The aspect of this accessible internal organ has the same significance here as in other fevers, with reference to state of the circulation, respiration, digestion and innervation, and the general

nutrition. As pyrexia progresses, the effects of increased vascularity, and of gradual inspissation upon its surface (where broadest and least mobile) of the buccal fluids, leading to change of colour from white to brown, become evident to the physician ; and the special function and movements of the organ may also become impaired. Usually the dry, brown fur is seen only at the back or down the middle of the tongue, then a white creamy layer, and at the edges a florid, moist aspect : occasionally the organ is flabby and indented by the teeth at its sides ; sometimes (in young persons) it acquired a 'strawberry' aspect ; and (in old or thin subjects) with low fever it became shrunk, fissured, blood-stained and motionless. At the height of the epidemic, I frequently noted a triangular clear space in front of the coated surface, which was found to be due to voluntary or involuntary scraping of this part by the upper incisor and canine teeth. Other incidental circumstances influencing the aspect of the tongue were an under-hung jaw, prominent upper incisors, a habit of sleeping with the mouth open, all conditions precluding free breathing through the nares only ; the ingestion of pul-taceous food : during fever (when thirst becomes considerable) the frequent drinking of water. Some native patients would attempt as usual to clean their tongues mechanically, at early morning before the surgeon's visit ; and others indulged as long as possible in the use of customary sialogogues (whence a deep red hue), both women and men doing this : not seldom in dark subjects, the tongue is naturally pigmented at its sides.

In the general clinical description, with illustrative cases, the tongue-changes at different stages of the fever have been described, and variations here are no more than usual. Owing to great elevation of pyrexia and quickened breathing these changes supervene promptly, subsiding at crisis with other symptoms : in about 50 per cent. of ordinary first attacks the tongue becomes dry towards the last day, and in about 30 p. c. of relapses : at the acme of fever, it was dry in at least 70 p. c. and 50 p. c. respectively ; and at the crisis, in about 40 and 30 p. c. respectively : an organ drying for more than a day acquires a brownish hue, which may persist a short time after dryness ceases : thirst and the dry tongue here go together, but thirst tends to last longest. The state of the tongue was seen to correspond even closely with that of the skin during both remission and exacerbation of fever ; their dry and moist conditions appearing simultaneously and even quickly interchanging : hence the condition of the tongue may vary from morning to evening of the same day, until near or quite the end of fever.

During defervescence by lysis, although the temperature is declining the state of the tongue does not necessarily improve, but the reverse, until fever ceases : and after even a critical fall, with its prompt general amendments, I have not rarely seen the tongue still dry and brown for 2 or 3 days longer, there being then a want of systemic reaction or some local complication ; considerable variation may be noted at this time. Pallor of the tongue, as of other parts of the face, attends the crisis ; when it occurs during pyrexia and especially at acme, it partakes of a general significance.

After defervescence, the tongue-changes subside gradually as they supervened, the middle and back part of the dorsum both earliest and

latest showing the dry and discoloured aspect : by the 3rd or 4th day of first apyretic interval the organ has nearly or quite resumed its normal appearance. When febrile complications now occur, the state of the tongue corresponds.

During recurrent attacks, the tongue may become dryish on the first evening and thinly coated, red and thickly coated the next day, and possibly dry and brownish in the centre on the third : the dorsum may then become glazed towards the front : a mitigation may be noted here, as during invasion, just before the acme ; at the crisis, reversion to the normal state is prompt. For some time, however, in this second apyretic interval a smooth and glazed aspect of the tongue may persist, in patches or continuously over the dorsum, which seemed to arise from atrophy more or less marked of the papillary surface, with subsequent imperfect production of epithelium ; such wasting corresponding to the general emaciation so usual after two (or even one) pronounced attacks of spirillum fever ; the tongue also is anæmic, and for a time dryish. In such instances the appetite may be vigorous, and no complaint was made of the loss of the special sense of taste.

In *typhus biliosus* and low types of fever generally, the tongue was apt to acquire the aspects well known in the more prolonged continuous fevers of Europe ; and the florid, parched and contracted state often persisted after crisis.

Febrile delirium or dysentery are not associated with a particular state of the tongue alone ; nor is vomiting or other sign of the epigastric disturbance so common in this fever.

The movements of the tongue did not commonly attract attention in spirillum fever ; their significance being borne in mind.

Glossitis was not seen as a sequel : mercury was rarely given in these cases.

9. State of the Bowels.—It was seldom that the alvine functions remained undisturbed throughout, constipation at some time being the rule ; sometimes irregularity of the bowels, with diarrhœa, and at the crisis there may be seen a mild kind of dysentery. Blood in the stools was rare : the presence of *lumbrici* was not commoner than in other diseases of natives. Severe and fatal cases were often attended with involuntary alvine evacuations, at the advanced stages of fever ; and at the same time *prolapsus ani* might be seen.

Special attention could not be given to this subject in a native hospital, but some illustrations of ordinary experience will be found in the detailed cases ; and upon analysis of others, the following remarks are founded.

Constipation.—The alvine evacuations may be scanty or suppressed for a few days before the onset of primary fever ; there was seldom constipation before the recurrences. During invasion-attack the bowels were decidedly costive in about $\frac{1}{3}$ of the cases, and sometimes remarkably so, the patient declaring there had been no stool for 6, 8, or 10 days : at the acme constipation was noted in 70 p. c. and at the fall in 50 p. c. of common cases ; with these final stages in debilitated subjects, old or young, the stools were sometimes passed in bed : torpidity of the intestines may continue for a short time after the crisis. During

first relapse constipation was not so frequent, and seemed to come on later ; it was decided in 25 p. c. of cases at acme and 30 p. c. at fall : involuntary evacuations were perhaps commoner now, at the end, than in the first attack, whenever the relapse was pronounced. Subsequent recurrences were not peculiarly distinguished. This symptom was not apparently associated with unusual implication of the liver, and it might be present in the absence of jaundice ; yet it was usually pronounced in cases of *typhus biliosus* (uncomplicated) : it is to be noted also that pauper patients had often not tasted food for some time before their admission, that irritability of stomach might interfere with feeding, and that sometimes the whole muscular system was evidently debilitated, the secretions generally being scanty as well. In severe and fatal cases there was a great tendency to irregularity of the bowels, and finally to diarrhœa rather than to constipation.

With costiveness the stools were not excessively hard, white or foul ; though scanty, the tendency was to diminished consistence, and I was often struck with the evidence of abundant bile when it might be supposed the liver was unusually implicated : the stools may be darker than usual.

10. **Diarrhœa.**—Was rare prior to the close of febrile attacks, in a series of 40 cases being noted only once so early as 6th day of invasion, in a lad of fifteen, when there was marked general abdominal tenderness without fulness, and cessation of the diarrhœa spontaneously at the crisis : similarly prior to costiveness and pale stools at acme and fall, there was diarrhœa, probably bilious, in the woman whose case is detailed as No. IX.

As the result of constipation, over-indulgence of appetite, lumbrici, drugs, bilious derangement, chill, or irritability of the enteric mucous membrane, this symptom may at any time supervene, the stomach also sometimes becoming irritable : its connection with congestion or inflammation of the same lining membrane (found not rarely at autopsy) is also intelligible : it occurred in the lad referred to above after the crisis, when such congestion was strongly suspected to exist, the temperature rising a little (99°) and pulse : see also the case H. A., No. X., where the post-critical flux tended to become dysenteric, as was by no means an unusual occurrence.

In cases fatal near end of invasion real or apparent, diarrhœa is apt to supervene after the 5th day, the stools being passed in bed : in cases dying later, the bowels were often irregular, and similar sign of debility not uncommon from the first.

The aspect of diarrhœic stools varied ; bile was usually indicated in plenty, and the transitions towards the mucoid and variegated evacuations of dysentery were not uncommon amongst native patients : stools passed in bed could seldom be inspected, and I would add that the diagnosis of unusual flux in these instances of paralysed sphincter might be fallacious, owing to constant dribbling of the fœces.

Abdominal distension, flatulence, meteorismus, were not usual in connection with diarrhœa ; but rather a collapsed state of the parietes : tenderness was usual, though not limited to the region where the ileum ends. Right iliac gurgling was decidedly rare throughout the epidemic,

even when the stools were liquid and yellow, and pink spots were present similar to those of typhoid fever: yet the results of autopsy clearly indicate the possibility of enteritis localised as in typhoid, though without ulceration of the Peyerian glands. Hypogastric uneasiness with diarrhœa was sometimes noted.

The varieties of this symptom were several, yet rarely of marked character; the wholly critical diarrhœa being very unusual, and though it were indicated a few times, yet not fully, or to the exclusion of cutaneous flux: an instance is quoted below. Under the head of 'Complications' some other remarks are added.

Blood in the stools was rare; it may have been overlooked, but on the other hand might be confounded with black bile in the stools (the point was borne in mind): it was a critical or post-critical phenomenon.

Diarrhœa at Crisis of Fever.

CASE XXV.—M., 15, admitted with his father (case of typhus icterodes) at close of invasion-attack, which ended by critical decline of 70.6 , sweating probably scanty, constipation of the bowels: an eruption of pink spots, depression, abdominal tenderness not mentioned. The relapse was very pronounced and produced much suffering: on the 4th and last day, m. t. 104.4 , p. 112, severe pains in the limbs preventing sleep, tongue coated, much thirst, disinclination for food and has eaten hardly anything of late: aspect dusky and depressed, no delirium, there is much general abdominal tenderness on pressure, bowels free, spleen now to be felt. E. t. 104.0 , p. 110, resp. 32, pains in loins and knees severe, much thirst, spleen larger and tender: the blood still contains spirilla, and also some free protoplasm and large granule-cells. Next day—m. t. 101.4 , p. 100, had no sleep in consequence of being frequently purged, motions watery; the splenic and abdominal tenderness continue, no sweating, no eruption; the diarrhœa ceased of itself with this slight decline of t.; much debility. At noon t. 99.4 . E. t. 96.2 , p. 66, no sweats, splenic uneasiness and fulness less, one stool. The body-heat still remained low—next d. m. t. 96.0 , p. 64, no sweats, again diarrhœa, motions scanty and watery; there is a sense of abdominal distension but no such distension is visible, nor is there any evident tenderness or enlargement of the viscera, much thirst, a few pink spots have appeared; the lad is so exhausted by the purging that he cannot walk. Depression continued this day: on the following day m. t. 97.0 , no more purging, now pains in the joints, some sore throat, some uneasiness on pressure near the umbilicus, no splenic uneasiness: after a rise to 98.0 , the t. again declined next day to 96.4 , p. 66, and there was still general uneasiness of the abdomen, without fulness, no more eruption, no jaundice: after this date reaction began, the appetite became excessive, the local signs subsided, the spleen being nearly imperceptible, bowels regular. It was my impression that the diarrhœa was connected with the acme and crisis of this attack, being probably attended with congestion (? petechiæ) of the enteric mucous membrane, which is known to occur in severe attacks of fever at their close: it was not apparently due to cold or indigestion.

Melena.—M., 25, famine-immigrant, weak, admitted near close of invasion, two remitting paroxysms being seen, last temp. 102.8 , p. 126, tongue coated and florid, a burning sensation in the soles and palms, hepatic and splenic enlargement, and decided enlargement of the spleen: at beginning of fall t. 98.2 , p. 80 (next morning 96.0 , p. 80), blood appeared in the stools which soon were passed in bed; sweating doubtful; the bowels became relaxed and stools dysenteric, and then bilious: perspiration followed the defervescence, the tongue being white and dry: recovery was prompt: no relapse.

Another instance was that of a vernacular student (severe attack, relapsing), who had also epistaxis and may have swallowed blood, there was vomiting and decided hepatic implication, but less splenic.

11. Circulating System.—The Heart.—Almost the only changes noted at autopsy of fatal cases, were distension of the right cavities and

left auricle, with a pallid aspect of the heart-muscle at late periods of fever especially. The absence of more manifest lesion in even severest cases, renders it probable that in the milder surviving the heart does not greatly suffer; and such inference is supported by the prompt rallying and complete convalescence, usually witnessed. Marked functional derangement, however, attends the specific fever; presenting certain invariable and some occasional features, which in their combinations may be peculiar to the spirillar infection.

Derangements noted.—Weakened impulse; first sound weakened, prolonged, attended with a basic murmur; dilatation of right side of heart, and changes in position.

Relationship to stage of disease.—The condition prior to pyrexia of invasion was unknown; with fever derangement begins, and *pari passu* augments to the end; after crisis, debility persists for a time: with relapse the same series of symptoms is repeated, and at pronounced recurrences may be more apparent than at invasion.

Frequency.—Feebleness of the heart was, according to my observation, invariable, statistical data regarding the other clinical signs are wanting, the following remarks being derived from a few, unselected cases specially watched: age of the subject, as well as comparative severity of attacks, might account for variations noted.

Examples.—Symptoms in a lad of 14: Chart 2, Plate IV. At the beginning of invasion-crisis the heart's action seemed forcible, yet the second sound decidedly predominated; p. 142; the blood contained no spirilla, but large clumps of granular protoplasm. During first apyretic interval, slow restoration of the heart (*vide* pulse-rate), action regular; second sound predominant. With onset of relapse, 2nd sound still accentuated, the first had a 'booming' character at apex of heart, most distinct in the recumbent posture (direct comparison was here made with another lad free from fever): in the evening both temp. and pulse had risen, heart's impulse felt inside l. nipple, first sound booming and prolonged, with a decided bruit over a space limited to base of heart and best heard at junction of 4th left cost. cart. with sternum; where systolic sound predominates, at apex downwards, and upwards along aorta it is less pronounced than the diastolic, and its booming character becomes lost; also beyond the cardiac area. On placing the patient in sitting posture, a diminution of the booming and bruit; the prolonged character of first sound more diffused, its bruit slightly transmitted along the aorta: impulse not more apparent, felt lower down than when in lying posture. The pulse was unusually rapid in this case; tolerably full and firm. Second day—temp. and p. reduced: the first sound less booming; bruit fainter, heard in recumbent posture at base, not in sitting posture; impulse distinct; second sound more accentuated. In the evening, temp. and pulse risen: some general distress: the systolic bruit is best heard above left nipple and towards left clavicle, as if produced chiefly at orifice of pulmonary artery: heard also in sitting posture, but it varies in character and intensity; it is rough in tone: over the carotids a bruit is heard on application of stethoscope. Third day—still high fever, pulse now soft: aspect distressed: systolic bruit as yesterday, very local and best heard in lying posture: in the evening, acme of fever, pulse 130, again firm; the bruit is very distinct at base of heart, where there is visible a broad pulsating wave, not felt by the touch, which I attributed to the action of distended auricles; the bruit does not travel downwards and to the right and therefore is not produced by the tricuspid valve, it is heard in the sitting posture nearly as well as in the recumbent: heart's impulse as before, practically absent; second sound predominant: spirilla in blood declining, white cells and large protoplasmic masses abundant. Last day—fall in progress, p. 120, less full and softer, thrilling, not dicrotic: bruit not now heard in any posture, first sound rather booming, no perceptible impulse, second sound predominates. In the evening, p. 80, irritable, small, weak, intermitting at 30-60 beats: heart's action weaker, first sound booming in sitting posture only; the second more accentuated; impulse barely felt.

First day of second apyretic interval: t. $96^{\circ}6$, p. 76, smaller, weaker, but regular: first sound has the same character; impulse none. Second day—still depression, p. 70 lying, 100 standing, weak, small, faltering: heart's sounds—lying posture, the booming character less pronounced; action of heart faltering, excitable, slightly irregular, the diastolic pause decidedly prolonged, and hence an impression of slowness although the beats are 70 per minute: sitting posture—action quickened, first sound becomes so brief as to be almost inaudible and the deliberate character is lost; second sound greatly predominant: standing posture—temporary reversion to the comparative loudness of first sound, not now booming, but simply stimulated by change of position: the second sound at first subsidiary, becomes predominant as the effects of muscular exertion decline: impulse and auricular wave absent. For ten days longer, the heart's action had the same character of abbreviated and approximated sounds, and prolongation of the pause; first sound faint and impulse slow to return (slightly fell on 7th day): on the 16th day, the normal state seemed to be restored.

To my apprehension the above phenomena were referable to the varying state of the blood, and to the impaired nutrition of the heart-muscle; the cardiac valvular apparatus not being structurally concerned.

In the instance of two adults, one of whose cases is detailed above as No. IX., I observed at acme of invasion signs of dilatation of the right side of the heart, in augmented cardiac dullness and extension of impulse to the ensiform cartilage and beyond towards the right. One case also revealed a first sound booming but free from murmur, over the cardiac region: at the base, second sound alone heard and along the aorta, chiefly, it was attended with a roughness, as if from friction. Crisis next morning—the labouring impulse had disappeared, first sound fainter and less rumbling, second sound more predominant; p. 100, very weak: during the ensuing apyretic interval, the impulse did not return till the third day, and then there was noted a slight reverberation of the first sound along the aorta (?). With a brief specific relapse, I noted that the first sound was attended with a soft murmur, heard best to the right of the ensiform cartilage, and over the mid-cardiac region it had a slightly booming character; these features subsided at the crisis.

As an example of changes in fatal cases, I quote the case of a lad, *æt.* 10; just prior to the acme, when the blood was charged with spirilla and large granule-masses, p. 100, soft, the systole of the heart appeared forcible and tumultuous, and the first sound so altered as to raise the suspicion of pericarditis; yet these characters passed away with the crisis: lobular pneumonic collapse ensued, with fatal exhaustion, and after death the heart was healthy-looking. In another case at acme of invasion, p. 96, soft, it was noted that the heart's action was tumultuous, the face congested and there was throbbing of the carotid and temporal arteries: at autopsy muscular substance pale and soft; pneumonia on the left side.

In general, during fever a weakened impulse was always present, progressing with the pyrexia (at exacerbations increasing and *v. v.*) until the crisis, when the heart's action promptly declined on cessation of the febrile stimulus, and but slowly afterwards regained, like other muscles, its normal tone. The conjunction of rapid action with debility, a prolonged booming sound, a full, soft pulse and blood charged with quasi-foreign particles, is probably characteristic.

The weakened first sound was a necessary corollary: at crisis it may be inaudible, and almost so for a few days longer, when depression is prolonged; most patients on their discharge, had still the feebly acting heart.

Alterations of first sound.—The booming or prolonged character was very frequent: it was limited to the cardiac area, almost ceasing at the base of the heart opposite the 3rd or 4th costal cartilage; its seat is the thicker cardiac muscle, and it may exist alone. The systolic murmur is less frequent and seems to differ in its seat at arterial or venous orifices of the heart (see above): though often attending the booming sound, it is not

to be confounded with it. Undoubtedly simple anæmic murmurs were present in fever patients, yet they are not identical with the above.

Diastolic changes.—Accentuation of the second sound necessarily following partial suppression of the first, its early occurrence in spirillar pyrexia is noteworthy : whether or not the valvular sound is ever modified, except in partaking of a general debility, remains uncertain ; it may be widely diffused over the cardiac area, as well as predominant.

Altered relationship of sounds and pause.—For a few days after crisis with a pulse quicker than normal, cardiac action may be of brief duration as well as of enfeebled force, the succeeding diastolic interval then being prolonged : rhythm is apt to be irregular, and irritability of the cardiac muscle marked. Such a hesitating and quasi-spasmodic action was noted with a declining pulse, fourteen days after relapse.

Change of volume.—As estimated by area of cardiac dullness on percussion, this may be obscured by varying volume of the lungs (especially on left side) or of liver, stomach and spleen. In the three cases detailed, I considered that at the acme of fever there occurred distension and consequent enlargement of the right side of the heart, and also of the left auricle.

Change of position.—I found distinctly that the heart was, at acme, displaced downwards and to the right, by conjoined pulmonic (left lung) and splenic encroachment, returning promptly to its normal position when pyrexia ceased. I have also noted the organ to be displaced backwards and upwards, so long as high fever existed, by distended stomach and intestines, and enlarged liver. Much variety obtains here, yet in extreme and even fatal cases, it did not seem that displacement alone led to interrupted or irregular rhythm of the heart's action.

12. The Pulse. Frequency.—Whilst following the temperature, the pulse tends to lag behind ; and this tendency becomes more apparent with successive relapses : it is visible even at the close of the invasion-attack. Such data indicate that under the continued and repeated stimulus of high spirillar pyrexia, the irritability of the pulse becomes more and more slowly manifested. At the same time, when the stimulus is withdrawn, the pulse but gradually reverts to its normal state ; after each febrile attack, it for a time descends below the normal level, the disposition thus to decline varying in different cases. Reference should here be made to the chapter on Pyrexia.

Excitability.—This feature is seen at end of crisis and during apyretic intervals : in some instances the pulse increased 20 to 30 beats, or more per minute, on the patient changing posture from the lying to the sitting and standing : this effect of muscular exertion does not last long. When during febrile or incidental stimulus the pulse rises in frequency, it may become bounding or thrilling, functional irregularities of rhythm often disappearing for a time.

Volume.—During high fever, especially for the first few days, it may be tolerably full or large ; this state tends to diminish, and at crisis the volume is greatly reduced : subsequently it is moderate.

Strength.—A hard or incompressible pulse must have been very rare in uncomplicated cases of spirillum fever, for I do not recollect meeting with an instance : to the touch the feeling was always of easy

compressibility, and I am aware this test is not always a sure one. At the crisis feebleness, and subsequently softness of the pulse. In some fatal cases a firm pulse has been noted : *e.g.* female adult, at probable acme t. 105°·6, p. 140, full, strong and incompressible, was recorded : there was cerebral hæmorrhage, congestion of scalp, firm contraction of left ventricle, kidneys pale, not enlarged ; the blood contained large granule-cells. Granular kidneys were very rare in native fever-patients at Bombay.

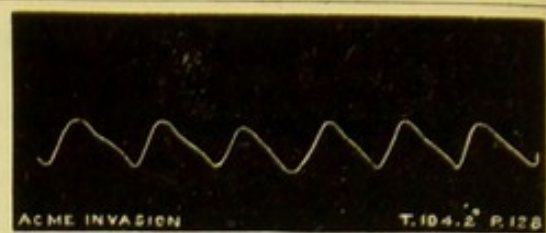
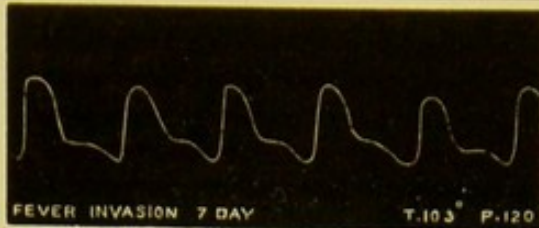
In the course of the chief stages of fever, crisis and apyrexia, sudden changes of the pulse (other than in frequency) were rare. According to my experience, dirotism during fever was hardly ever seen ; at the most a tendency to redoubling of the beat being noted in a few instances at acme, or during a pseudo-crisis : such cases amounting to four or five only. Intermittency was occasionally noted at the crisis.

So far as the pulse depends on the action of the heart, a close relation was observable throughout : so far as it pertains to the arterial system, the pulse, as felt by touch at the wrist, indicated diminished tension : possibly some fallacy lurked here, yet this was my own impression, and allowance is made for occasional rigidity from arterial disease.

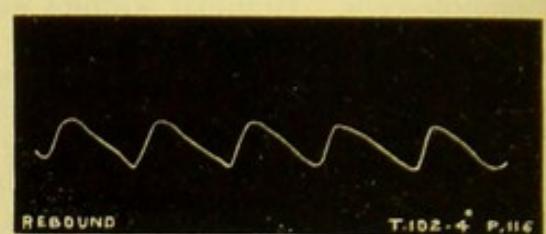
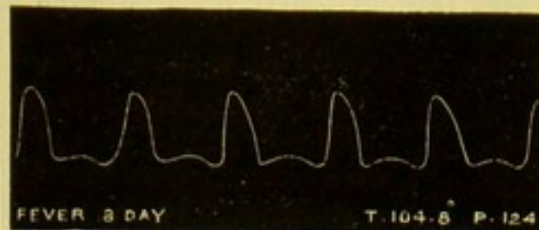
Sphygmograph Tracings.—Those appended are enlarged copies of originals taken at the J. J. Hospital during 1878, with an ordinary Marey's instrument (always radial) : other series are concordant. Nos. 1 to 8 pertain to M., 35, admitted at close of invasion : 1 to 3 are successive tracings on last three days of spirillum fever, they indicate pyrexia with excitement as at early stage of typhus, but differ in the blunter or more rounded end of the pulse wave. No. 4 is at crisis : the wave though much depressed, still shows signs of intra-vascular pressure. Nos. 5, 6 and 7 are days succeeding crisis, the indications of obstruction to blood-flow, becoming more decided ; they are afterwards less apparent : No. 9 is from another case, at 18th day after last relapse, it probably corresponds to the infrequent and excitable kind of pulse alluded to above. Nos. 10 and 11 were taken just after acme of invasion, and two days later during a febrile rebound (*v.* Chart 3, Pl. IV. and Case X.) : they are similar, and they differ from specific fever tracings. The series of tracings from this case shows that with onset of first relapse, the pulse resumes its character at invasion, and maintains it more manifestly on successive days : *vide* No. 12 : the tracing at crisis is also alike, although the temp. had not declined below 99°·8, and for 6 days longer there were indications of augmented blood-pressure. At the brief second relapse, on the first day the tracing is that of specific fever ; on the second (at probable acme) it is different, indicating a much less obstructed blood-flow : normal characters were reappearing when the man left hospital, three weeks afterwards.

Fatal cases.—No. 13 is that of a patient admitted late in invasion, t. 120°, p. 140, with all the characteristic marks but jaundice of acute spirillar infection : the blood contained large clusters of spirilla and many large nucleated cells : death took place five hours afterwards. Nos. 14, 15 and 16 belong to M., 50, who was admitted with his family all ill (*vide* Chapter on Contagion, the Ahmed family), and who sank

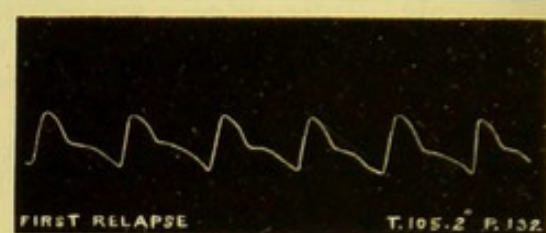
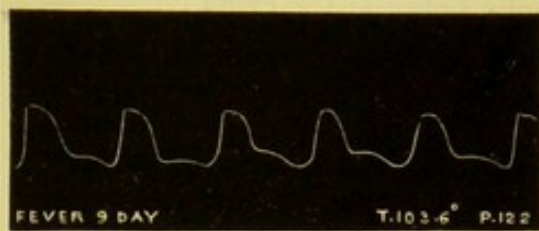
1



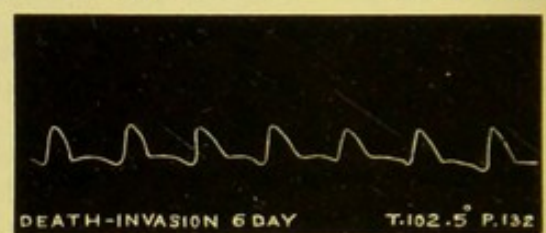
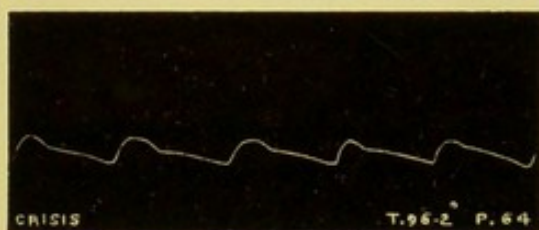
2



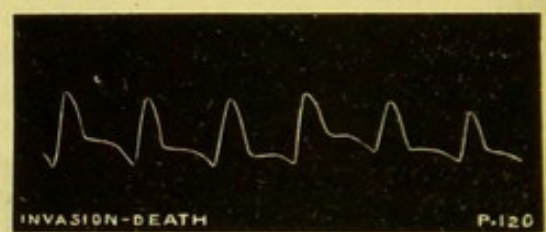
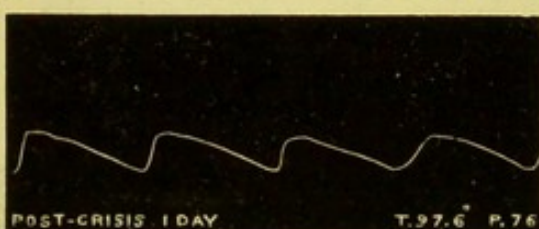
3



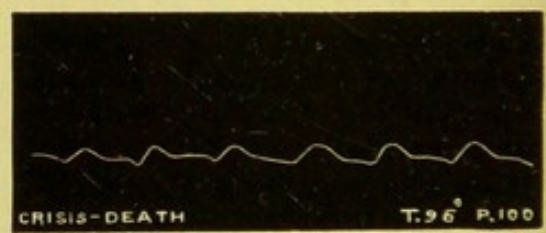
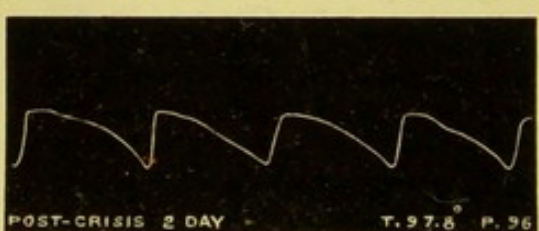
4



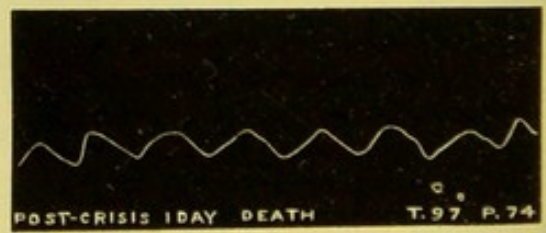
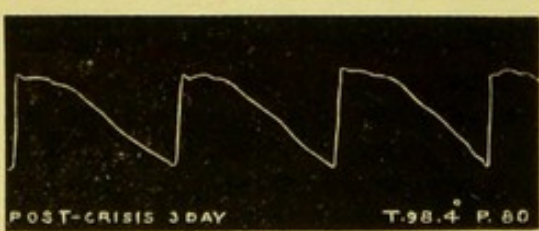
5



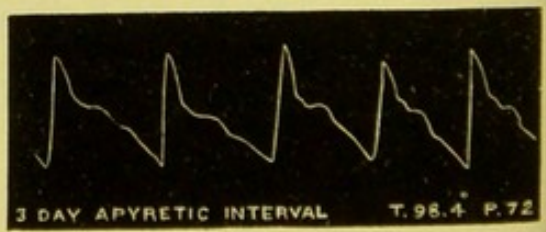
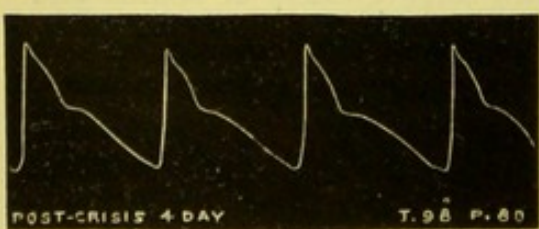
6



7



8



exhausted in three days; the first and second tracings resemble those of survivors at corresponding stages of specific fever, and the last is that of the moribund state.

These sphygmograph records are chiefly remarkable from the absence in them of the dicrotism so frequent in typhus and enteric, and from their presenting blood-pressure signs: doubtless, the two conditions are correlated.

I have above stated that whilst the febrile pulse was rarely dicrotic, it was almost invariably easy of compression, and at present I see no method of accounting for this discordance of clinical and physical signs, other than the supposition that the causes of obstructed circulation exist in the blood itself, being at first the multitude of spirillar filaments, and after crisis the ingress of large granule-cells.

It will be perceived that tracings like the above, are not calculated to afford much help in prognosis.

13. Respiratory System. *Cough: Voice.*—That the lungs suffer severely during the spirillar disease was shown in the autopsic records, pneumonia and pulmonic congestion being common occurrences in the first interval, and a pale and inflated or collapsed state in deaths during primary and recurrent fever, and the second apyretic interval. The life-symptoms correspond, being similar (except in intensity) amongst both surviving and fatal cases. Those indicative of pneumonic congestion and inflammation were, as in other fevers, liable to be simulated or overlooked; whilst those attending the pallid lung-state constitute a kind of dyspnoea, which, taken with other signs, may be peculiar to certain forms of blood-infection including the spirillar. Nothing identical was observed in the course of ordinary remittents, at Bombay.

Rapidity of Respiratory Movements: Morning and Evening Data in average surviving cases.

M., 35, two days before critical fall of invasion-attack, resp. 34 per minute (t. $104^{\circ}6$, p. 124) not rising at the acme (as would appear), at fall the rate declined to 14 p. m. (temp. $95^{\circ}4$, p. 70) and was steady there, not following either temp. or pulse until after a brief relapse on 11th day, when it rose to 22 p. m. (t. 104° , p. 112) and promptly declining continued at 14 p. m. for a fortnight longer. An adult man.

F., 35 (*vide* Case IX.), just after the acme of invasion-attack, resp. 44 per minute (t. $104^{\circ}4$, p. 140), and at fall declining to 26 p. m. (t. 96° , p. 78), rising alone to 32 soon after and slowly declining during the first apyretic interval, as does the pulse but not the body-heat: during the well-developed relapse, respirations rose quickly to 40 p. m. on first day (t. $105^{\circ}6$, p. 108) then following temp. rather than pulse, the rate declined moderately at fall (28 p. m. with t. $96^{\circ}6$, p. 84) and corresponding with pulse declined to 22 p. m., being steady there for fourteen days, excepting a rise with brief relapse happening about then: just before this the pulse had gradually sunk to 54 p. m., there being no similar decline of breathing rate. During the relapse there was a little bronchitis. The influence of sex was manifest.

M., 14, the rate was nearly the same as in the woman: very quick two days after the first crisis (40 p. m. with t. $98^{\circ}6$, p. 100) it descended, like the pulse-rate; until the relapse, when it rose rather before t. and p.; it followed the temp. during fever (maxima 40 p. m., t. $104^{\circ}2$, p. 150), declined promptly, yet moderately, at fall (22 p. m., t. $96^{\circ}6$, p. 76) and for twenty-one days longer the breath-rate averaged 20 p. m., rising at irregular times, both with and without p. and t. Patient a youth.

Extreme rates during fever may or may not correspond with those of

temp. and pulse : the tendency is to such correlation, excepting when in the relapse the pulse, as usual, rises with progress of fever, the breathing-rate does not augment, rather declining towards the end ; the maxima, minima and mean rates are different in different cases ; and the normal preponderance of rate in females and the young appears also during this illness.

In general, neither respiration nor pulse ascends proportionately to the temperature at initiation of relapse ; nor do they decline at fall (particularly does not the breathing-rate) in equal proportion. These statements are made upon the above data, and reckoning resp. 1 : p. 4, and : t. 5°:5.

Variations from the above average-rates were not uncommon at all periods of illness, and seemed to be referable to several causes : mere height of fever was not in detail an invariable determining influence ; pulmonic congestion probably always led to increased frequency—cough or pain in the chest, with scanty, frothy sputa attending ; nervous excitement was sometimes the only apparent influence in causing undue frequency ; often muscular pains of the body ; and special interference with the action of the diaphragm, through either unusual turgescence and distension of the abdominal viscera, or excessive tenderness of parts in the upper zone and particularly the epigastrium. Several instances were noted of rapid breathing when the abdomen, though very tender, was not distended ; it might be even flat or retracted. In the normal state, the rate per minute of respiratory rhythm varies very largely.

The stage of acme was often attended with extreme perturbation of the breathing, and at this turning-point of the attack, the respiration, not less than other functions, was probably always embarrassed.

The period of the critical fall presented many variations : thus, of two adult men, in one the respirations were 26 with a temperature of 94°, p. 70 (no collapse), with a slight rise to 95°, p. 72, they declined to 20 ; next day, with 95°:6, being 24 : in the other case, the breath-movements were 50 at t. 95°, p. 80 (collapse) and then declined with rise of temp. ; they had been 58 at the moderate acme, and were much fewer in the succeeding relapse with a higher temperature ; cough was present during the invasion-attack only. The mean of seven ordinary cases was with temp. at main fall 96°:3, No. of respirations 23.

Aporetic intervals.—So far as seen in hospital, the ordinary breathing-rate was moderate, not continuing to decline, as, for a time, does the pulse : sub-normal rates were unusual, being seen only in elderly subjects and in conjunction with mental depression.

Fatal cases.—In the briefer uncomplicated casualties during fever, the rates varied within the limits already named : even in an infant and a girl they were comparatively slow (lungs found unchanged) ; also in a man of 60 years (*e.g.* 25, with t. 103°:6, p. 120, shortly before death) and in a young man 40 per min. with t. 102°:4, p. 120 (lungs healthy). Complicated cases showed wider ranges, yet seldom extreme, except at the acme of attack. The comparatively slow rate in the infant (20–30 p. m.) was remarkable.

Character of chest movements.—When frequent, the breathing was usually more or less shallow : frequent and deep respiration was very rare and of brief duration, except in complicated cases.

Form of movements.—Respiration mostly thoracic was the commonest form ; its natural predominance in women was noted ; abdominal breathing being occasionally seen in young males and infants. General upheaval of the chest walls, and even an asthmatic type, were occasionally seen in fatal or complicated cases : and towards acme of fever, the type of breathing varied considerably, according to state of thoracic and abdominal viscera.

Connection of respiratory movements with particular states of the lungs. —With the pale and inflated condition (4 cases), temperature was high, pulse very quick, breathing 50–70 per minute, not exclusively of one form, and dyspnœa not a prominent symptom ; moist sounds were audible in the chest : twice there were large, pale clots in the right heart, and the blood was charged with post-spirillar granule-cells. With the pale and collapsed state (5 cases), the organs were dry and dyspnœa urgent, according to degree of collapse, respirations 20 to 48 per minute, temperature varying, pulse frequent and small, the blood was full of spirilla or granule-cells, and both sides of the heart contained clots. In 4 deaths at critical fall, the lungs were collapsed twice, or congested : the breathing was described as laboured, sighing, gasping or compared with bellows-action (as in cardiac embolism) : in the last-named instance, the base of one lung (collapsed) was saturated with fine, frothy serum and a widely distributed clot was found in the right heart cavities and pulmonary arteries. In two instances of well-marked abdominal breathing, the lungs were generally or partially collapsed, deep basal congestion being present once. The explanation of these phenomena is, at present, only presumptive.

Respiratory phenomena at acme and beginning of fall.—Many instances were seen casually in the wards amongst survivors of extreme breath-troubles at these moments, when the blood undergoes a sudden physical change ; though only part of the general distress, yet the dyspnœa with enfeebled heart-action sometimes seemed to predominate, and restlessness, drowsiness, moaning or noisy delirium might be as marked here as in fatal cases. Necessarily, the stress was of brief duration, and chance seemed to determine whether or not life should continue. A contemporary rise of temperature (seen or not) was doubtless the rule, also abdominal fulness and tenderness in varying degree ; the respirations were shallow, thoracic and frequent, rising to 50, 60 or 70 per minute ; and in extreme cases, the chest seemed fixed, the head was thrown back and nostrils actively working, the sufferer meanwhile tossing about in agony, and the expired air being perceptibly cold. Relief was almost always prompt ; and, like pulsation, the breathing-rate subsided slower than the body-heat. With urgent phenomena of this kind, it is not difficult to comprehend the comparatively frequent onset of pneumonia with and after the specific fever ; and many patients were reported to have died with troubled breathing suddenly supervening.

Cough.—A mild degree of bronchitic congestion attended with cough and scanty, frothy expectoration, was noted in upwards of one-half the cases of fever ; such congestion being indicated on auscultation by coarse, moist sounds, and seldom by impaired resonance or signs of

pulmonary oedema. These symptoms might pass through intermediate stages to confirmed bronchitis and pneumonia (*vide* Complications of the Respiratory Organs): they usually supervened on or after 3rd day of invasion-attack, ceasing with or soon after the crisis; yet not very rarely first appearing later in the post-febrile period in connection with mild secondary fever: they were much less frequent during the first relapse, and subsequently.

Some degree of pulmonic congestion was probably an invariable attendant on spirillum fever, whether not the act of coughing were noticeably excited; and it is important to recollect that this symptom is not a precise indication of a really pathological state, readily becoming serious. Autopsies showed that in even fatal cases, cough alone had not much significance, for with it the lungs might after death be found in a quasi-normal state, or, on the other hand, inflamed to a degree much exceeding the urgency of this symptom during life.

Voice.—Moaning as a sound of distress at acme of fever and during lysis-like defervescence, was a sign not heard in common fevers: even the infected monkey had its special cry.

At the semi-collapse of crisis, the voice might be reduced to whisper, reminding the observer of its state in cholera.

14. **Liver.**—The symptoms of pain, tenderness and enlargement are, in general, explained by the organic changes found after death, in contemporary hospital cases differing from the surviving mainly by their greater severity.

Constant pain in the liver was as rare as that in the spleen, and a fatal case showed it might co-exist with a seemingly unchanged condition of the gland. Hepatic tenderness may be so exquisite as practically to induce constant uneasiness to the patient,

Tenderness, soreness, or contact-pain was common, and might be present alone, or, much oftener, with enlargement; splenic and gastric tenderness usually co-existed with it. Its degrees were very various, and its presence was often elicited by action of the diaphragm (*e.g.* during cough), as well as through external pressure; commonly limited to the hepatic region, it sometimes merged into a general sensitiveness of the upper abdominal zone. It has been described as sharp or cutting, when almost certainly acute hepatitis was not present; the patient promptly recovering at the near critical fall. In 10 selected instances amongst the fatal cases, this symptom seemed to be associated with a pale or fatty state of the viscus 4 times, and with a mottled aspect once; whilst 4 times the liver appeared nearly normal, and only once was there found such an inflamed condition of its serous investment as the urgency of elicited pain seemed often to point to. Acute fatty degeneration, as elsewhere described, cannot always account for this local sign of disease, for in 4 cases on my list it was present without any entry of associated hepatic tenderness; a preceding acute congestion, or parenchymatous inflammation might, however, furnish a sufficient explanation in some instances. As with the spleen, so here, the tenderness sometimes seemed to be neuralgic. Sympathetic tenderness, spasm and rigidity of the adjoining abdominal walls were often noted: such might

be present in the upper part of the abdomen, without marked hepatic and splenic implication during life, when after death the subjacent viscera were found much affected. Sympathetic pain in the right shoulder was also occasionally remarked; and a dry, spasmodic cough.

Enlargement of the liver, as ascertained by palpation through the abdominal walls and percussion of the chest, was noted in at least 33 per cent. of all surviving cases; it was found in 50 p. c. of those at febrile periods, and in 10 p. c. of those at non-febrile periods. The corresponding proportions for fatal cases were 48.6 p. c. (total), 60 p. c. (febrile) and 33 p. c. (non-febrile); the chief divergence here (an important one) being the last regarding the state of the liver in apyretic stages of severest attacks.

During specific pyrexia the liver-dullness has measured 7 inches in vertical nipple-line: occasionally it extends upwards to the 4th intercostal space (the abdomen being distended in conjunction with hepatic enlargement); and much more rarely the liver seemed to be pushed downwards by increased volume of the right lung: variations here were numerous, and obviously explicable.

The left lobe of the liver is sometimes disproportionately enlarged, as proved by signs during life and the autopsic revelations: no fixed relations, however, obtained between this and gastric symptoms, for I have noted exquisite gastric tenderness (with hæmorrhage) with a left lobe found after death to be actually small.

Dimensions of the liver (never over-estimated) may, as was shown above, be greatly under-estimated from a variety of causes: hence the presumption arises that minor degrees of hepatic enlargement were often overlooked, for in this disease, especially, it would require unusual skill to avoid error with the lungs on the one hand and the stomach, intestines and abdominal wall on the other, all so liable to concurrent morbid change.

Symptoms according to stage of Fever.—Invasion-attack.—Nothing definite was elicited respecting hepatic changes in the incubation-stage of first attack: in my own case there was not any uneasiness referable to the liver.

First day: my scanty notes indicate that tenderness or fulness of the liver are more to be expected than splenic changes; on the succeeding day, they were distinctly intimated, with epigastric uneasiness; and in infants enlargement may be manifest. Third day: in 18 cases, hepatic tenderness attracted attention 11 times, and was present alone 5 times; its degree varied from the barely perceptible to the considerable, adding to the patient's sufferings: it was usual in children, and found to be commonly most marked at evening with rise of temperature: enlargement of the organ both upwards and downwards was usually present. Fourth day: in 24 cases, the liver was implicated 16 times, tenderness being oftenest noted; enlargement might still be slight. Fifth day: in 23 cases the organ was enlarged or tender, or both, 14 times: when the acme occurred now, its condition might resemble that of acute hepatitis; dry cough may attend this state: on the other hand with even high fever, the liver may seem to escape lesion. Sixth day: in 25 cases, the organ was implicated 17 times: local

symptoms were most urgent when the acme of attack happened now (9 times), yet this event once, at least, passed without attracting attention and local signs were not necessarily proportionate to other symptoms. Seventh and eighth days: 16 cases, and the liver affected 11 times, sometimes more strikingly than before; acme-period common, and it would seem not associated with liver changes so often as with splenic. Critical fall—whole period. In 32 cases the liver was perceptibly tender, turgid or enlarged 22 times: commonly the changes were of brief duration at this stage, and had nearly disappeared at its close.

First Apyretic Interval.—First day: of 33 instances the organ was tender or enlarged, or both, 10 times at least: on the following day hepatic signs were reduced one-half, and still more on third and fourth days; so that on the fifth there was noted only 1 case in 26, and on the seventh day not a single instance. Nothing was noticed of that re-appearance of symptoms on the last day or two of the interval, prior to relapse, shown by the splenic organ.

First Relapse.—As the result of 155 observations made on the same series of cases as previously, it appears that whilst in the whole relapse the liver is affected in at least 32 p. c. of instances, it is not implicated uniformly throughout: thus, on the first day in only 8 p. c., on the second in 16 p. c., on the third in 25 p. c., on the fourth in 54 p. c., on the fifth in 70 p. c., and on the sixth and seventh days in 43 p. c.; at the critical fall, the gland was still affected in 32 p. c. of cases. These ratios are probably below the actual, but it may be allowed that the liver is not quite invariably implicated in the recurrent febrile attack, or always in proportion to intensity of fever.

During short relapses, hepatic derangement was less in proportion to abbreviated duration: it was not so frequent as the splenic.

Second Interval.—So far as appears from my data, the liver may be found affected in about one-third of cases on the first day of this period; on the second hardly at all, and subsequently to be quite free from obvious change, the resumption of a quasi-normal state being prompter than obtains with the spleen. Instances of exceptional persistence of symptoms were not here seen.

The relationship of hepatic derangement to secondary fever was sometimes evident, and the subject is illustrated under that complication: during ordinary symptomatic fever, the liver is not especially implicated: its connection with jaundice is detailed below.

A few instances were seen of specific fever in patients previously affected with hepatic abscess: the local symptoms were then not excessively exaggerated: thus, M., 30, had tolerably clear signs of abscess, during invasion there was very little exacerbation of hepatic pain, tenderness and tumefaction, a slight diminution even of these symptoms took place after the first few days, but dysentery came on: death on the seventh day. The liver weighed 48 ozs. (after evacuation of abscess). Such instances are comparable to those of spleen-disease prior to specific fever; and in both series, it appears that the local affection is not so much intensified as might be anticipated, by the superadded new infection.

15. **Epigastrium.**—The middle segment of the upper abdominal zone

was affected with pain, tenderness and distension in at least 20 per cent. of invasion-attacks and 30 p. c. of relapses : these symptoms were not always restricted solely to anatomical limit, and they varied much in intensity ; sometimes adding not a little to the patient's suffering. Upon some occasions, the sick brought from their homes, bore recent marks of cupping and caustic applications to the epigastric region.

Pain was rare and not peculiar : the uneasiness of over-distension is not here alluded to.

Tenderness was either acute and superficial (even strikingly so), or milder and elicited only on firm pressure ; it was most marked during pyrexia, following its exacerbations and being then attended with distension of the region ; but without this fulness, it was not uncommon at and immediately after the critical fall. Gastric irritability, spontaneous vomiting and diarrhoea at the crisis, may be attended with collapse of the abdominal parietes. Fulness or distension of the abdomen here varied much in degree, and was usually attended with a more or less tense state of the muscular walls : patients have expressed a sense of distension, when none was present : and a similar subjective sensation was referred to joints, unchanged in aspect.

The anatomical complexity of structure in and around the epigastrium, would of itself suggest a manifold explanation of the above symptoms ; and autopsic data point to the same influence : thus, in 10 selected instances, there was found congestion of the gastric mucous membrane alone 2, or with petechiæ and diphtheritic inflammation of the ileum 1 ; disseminated congestion of the small intestines 2, or more localised congestion of the ileum 2 ; sub-peritoneal petechiæ 2, and vascularity of Peyer's patches and large intestine 1. Intestinal congestion seemed to have been present, without marked epigastric or umbilical tenderness ; and great or intense vascularity of the stomach (mid-region or diffused) together with petechiæ beneath the mucous membrane, was twice seen after death with no account of epigastric tenderness during life (stimulants had been administered shortly before decease) ; in other instances where tenderness had been a marked symptom, the stomach seemed to be healthy. I have shown that acute gastritis is, at least, very rare ; and besides patients often become too rapidly relieved of epigastric uneasiness to permit the idea of organic lesion in these cases. Acute congestion of the stomachal mucous membrane probably existed in a case of fatal hæmatemesis marked by acute local symptoms prior to vomiting, and by local pallor of the tissues after death ; and in a modified degree, this state may be even common during fever. In the apyretic condition, the tenderness sometimes seemed to be neuralgic : it also appeared to arise from personal idiosyncrasy, and was common to groups of cases, probably under one infection. It was rare in children, and at shorter febrile attacks ; it might exist alone, and had no certain relation or degree to vomiting, even when the latter occurred at crisis. It was most pronounced in the severer cases, forming one of the characteristic phenomena of 'acme.'

Epigastric tenderness and fulness, on some occasions, appeared to be attributable to enlargement or acute congestion of the uncovered left hepatic lobe, which overlies the stomach ; and this view was supported

by post-mortem examination in three striking instances, but exceptions are known.

In another instance, surviving, the site of extended tenderness and its production on deep pressure, conveyed the impression that the duodenum might be congested as seen in autopsies: there was a yellow conjunctiva and vomiting of food; *vide* Case IX., which also on last day of relapse (with a spleen less tense than before) showed pain on pressure over hepatic, epigastric and inner half of splenic regions—this last limitation being singular.

In a third case at crisis of relapse, it was noted that the epigastric neuralgia (then the main symptom) was excited by firm pressure below and to right of ensiform cartilage, over probable site of the solar plexus. Counter-irritation would relieve such symptoms.

More diffused or general abdominal uneasiness was seen in various forms and many degrees. The most characteristic state during fever was acute tenderness with fulness of the entire upper abdominal zone including the epigastrium and both hypochondria; seldomer was the umbilical region affected (independently of dyspepsia, diarrhoea and worms) when it seemed possible that scattered congestions or petechial extravasations of mucous, muscular and serous coats of small intestine were present, or similar changes in omentum and mesentery. Hypogastric uneasiness elicited on direct pressure, or by coughing, or the erect posture, and not attended necessarily by fulness, was occasionally witnessed; it did not appear connected with the urinary bladder, being rather referable to known vascular changes in the lower part of the ileum, and end of large intestine; iliac gurgling and tenderness, on either side, were rare, yet they were noted (concurrent diarrhoea absent) on the right side, in connection with demonstrated congestion of the lower end of the ileum and of cæcum (ulcers never present): lumbar pains were not traced to the colon.

General distension and great tenderness were seen at the close of some fatal cases, which after death showed parietal and visceral pallor of peritoneum, paleness and great distension (? paralysis) of muscular coat of stomach and intestines, with pallor of the mucous membrane except where congestive patches or small extravasations had been formed. The liver, spleen and kidneys were characteristically enlarged, and full of blood: further, some cases showed subserous petechiæ, and the most characteristic of all diphtheritic enteritis at the termination of the ileum.

Besides the anatomical lesions already mentioned as possibly associated with these vaguer symptoms, there is to add over-distension of the muscular coat of stomach or intestines, and petechial extravasation in the diaphragm and beneath the right *rectus abdominis* muscle, which I have also noted in similar association.

In my enquiries the state of the pancreas did not attract special attention, nor were the solar plexus and sympathetic ganglia so strikingly changed in aspect: a minute examination of these parts was not made.

Epigastric Symptoms according to stage of Fever.—Invasion-attack: absent, so far as known, on the first day, in 4 cases they were noted

once on the second morning when neither splenic nor hepatic signs were present : on the third day they were entered in 28 p. c. of cases, on the fourth much oftener, and on the fifth, sixth and seventh days in about the same proportion ; at the close of fever, including the acme, they were noted in 63 p. c. of instances ; and in 50 p. c. at the fall. It would appear that these symptoms are commonest at the onset and close of attack, especially at the acme, and in general do not present that gradual increase in frequency with progress of fever which is perceptible in hepatic and splenic phenomena : they bear no very evident relation in intensity to other symptoms.

First Interval : Whilst present in 40 p. c. of the above pyrexial cases their proportion during this non-febrile period declined to 9 p. c., thus indicating clearly their connection with fever. On the first day these symptoms persisted in the ratio of 20 p. c., on the second to the extent of 16 p. c., and of only 4 p. c. on the third day ; after which time their occurrence seemed incidental ; on the seventh day they had ceased.

First Relapse : present on the first day in 16 p. c., then in a ratio rising to 33 p. c. on fourth day, to 60 p. c. on the fifth when the acme was frequent ; these local signs were noted in about 30 p. c. of all febrile cases. At the critical fall they persisted in 40 p. c. During this recurrent attack they seemed to augment with progress of the fever.

Second Apyretic Interval : Epigastric symptoms were still present in some cases on the first day of reaction, but very rarely on the second ; and subsequently they were noted in only one instance of a lad, who showed some epigastric tenderness several days longer.

16. Spleen.—Changes found after death, may be taken as a guide to those prevailing amongst survivors ; they are, briefly, enlargement, infarctions (so-called), softening, and, very rarely, inflammation.

The local signs noted at the bedside were pain, tenderness and tumefaction or swelling of the spleen ; and I now proceed to discuss their comparative frequency and significance with regard to the ascertained lesions named ; for, degrees apart, there is no reason to suppose any essentially different alterations prevail in ordinary attacks of fever.

Acute pain in the region of the spleen was very seldom noted, and this fact accords with the rarity of recent inflammation of the organ. At the acme of attack brief, paroxysmal pain may be felt, which subsides with the fall. The symptoms of pleurisy have been noted to commence at this time, limited to the left side, and subsequently neuralgic pains in the left shoulder.

Dull, aching or dragging pain, or a sense of weight and tightness constantly present, and augmented by coughing standing or walking, was not uncommon both during and after fever, together with enlargement or possibly limited adhesion of the splenic serous investment.

Neuralgia.—Considerable tenderness, seemingly neuralgic, may remain for some days after the fall and diminution of volume ; and there may then arise sympathetic or neuralgic pain in the left shoulder.

Tenderness or pain elicited by pressure from below, outside or above (through the diaphragm), was a very frequent symptom. Usually sharp it varied much in degree, being sometimes revealed on mere touch or only on firm pressure over the splenic region. During fever it

commonly attended enlargement of the organ, without being proportionate to this ; and at the apyretic periods, it might be indicative of sub-acute organic changes. In its more exquisite form, it co-exists with tenderness of the upper abdominal zone, and possibly of the abdominal parietes : there is then usually febrile exacerbation, and commonly the acmal.

Morbid anatomy has not yet furnished a complete explanation of this symptom ; thus, of 10 instances analysed 2 showed a spleen seemingly unchanged, 3 large and firm, 4 large and infarcted and 1 somewhat softened. As in 7 other instances the spleen was not tender, with a seemingly normal aspect thrice, or with softening as often, and also not tender with infarcts once, I am unable to specify any invariable condition as alone associated with splenic tenderness. Enlargement and tension during fever do not necessarily entail contact pain, but when coming on late with typhoid symptoms, then local tenderness will usually be present and may be marked.

In one such case, the spleen became abruptly enlarged and tender just before the *perturbatio critica* (which was very pronounced), and I thought it possible that infarcts were forming ; yet such could hardly have happened, since with the fall the spleen promptly subsided and in three days was hardly detectable, though still the seat of uneasy sensations.

That *ante-mortem* softening of the spleen is not usually attended with local tenderness seems to be clear ; and in 2 cases when the spleen capsule was thickened and opaque, there is no entry of tenderness on pressure during life, these changes being evidently not recent. Unusual firmness of consistence, with or without enlargement, was not generally accompanied by tenderness ; and during fever increase of volume with a consistence almost amounting to hardness, may be present alone until the acme of attack, when acute sensitiveness supervenes. Changes of consistence felt during life necessarily imply augmentation of size, yet no rule obtains regarding concurrent degrees of either change, except that a much-enlarged organ was always firm to touch : in all cases, the state of the abdominal parietes has to be taken into account.

Splenic atrophy was not detected in the living subject.

Splenic tumour.—In nearly one-half of the data under analysis (which includes cases seen both early and late in the epidemic, and at all stages of fever) there was some palpable enlargement of the spleen ; and this proportion is doubtless under the correct ratio. Having, however, shown elsewhere that, in even fatal cases, the splenic tumour is not invariable, it may be admitted that sometimes the organ is not considerably augmented in survivors : with regard to minor tumefactions, considerable difficulty exists in their detection ; and even the larger degrees may be under-estimated. As often as not, I find in test examples a fairly close correspondence between post-mortem dimensions of the spleen and those noted just prior to death ; yet as the divergencies were sometimes marked, it becomes desirable to quote the exceptions. The following remarks include references to the liver also, the state of which was commonly noted at the same time :—abdomen full, tense and tender, splenic and hepatic enlargement not detected ; yet after death early next day the liver was found to weigh 60 ozs., its left lobe projecting somewhat into the epigastrium, the spleen weighed 19½ ozs., and also was of firm consistence. In this representative case,

the state of the front abdominal wall tended to conceal the actual condition of the viscera within, and I do not see how such impediment could be overcome. Displacement by the neighbouring organs must also account for anomalies like the following:—Spleen reported of nearly normal dimensions during life, after death found to weigh 14 ozs. (infarcts); as normal, yet found to be 12 ozs.; as only splenic fulness, and yet an organ of 17 ozs.; as no perceptible enlargement, and yet weighing 24 ozs.; and spleen not felt, yet *post-mortem* weight 11 ozs. and infarcts present. More definite estimates may be nearly as illusory; thus—abdomen distended and tender just before death, liver pushed upwards and not projecting more than 1 inch below the r. costal margin, yet it weighed 4 lbs. 9 ozs. (73 ozs.); the spleen reached 3 inches below l. costal margin and weighed 36 ozs.: again, abdominal walls tense, liver estimated to present only normal area of dullness and then found to weigh 68 ozs.; spleen more correctly termed large, its weight 17 ozs. Other data were these—liver reaching to upper margin of 6th rib in nipple line, and downwards 2 inches below the c. cartilage, with weight $73\frac{1}{2}$ ozs.; spleen reaching to upper margin of 8th rib and to $1\frac{1}{2}$ inch. below c. margin, inwards $3\frac{3}{4}$ in. from median line; its weight $38\frac{1}{2}$ ozs. (infarcts): liver reaching upwards to 5th rib and downwards one inch free, its weight 69 ozs.; spleen thought not to be enlarged, its weight 16 ozs., or more than double the normal. All subjects young adults and generally males. Few words are needed to explain the above discrepancies, and I will only add that besides the varying condition of the abdominal walls, and of adjoining viscera, the consistence and fixedness of liver or spleen have some influence; since a firm, resisting mass will be readily estimated, when a more yielding one of the same volume might slip aside. If doubt existed, I usually turned the patient on his side and had the thighs fixed, employing then palpation and percussion.

An excessive estimate of liver or spleen volume was never made, and it will be seen the tendency is to err in under-reckoning; for this reason and upon pathological grounds, I infer that visceral enlargement may more habitually approach the ratio in fatal cases (76 p. c.), than appears from my analysis of survivors alone (40 p. c.). Besides, when the data are arranged according to stage of fever, a close resemblance as well as significant difference, becomes apparent; thus, of total deaths during invasion-period, splenic enlargement was found in 70 p. c.; of total survivors at the same period an equal ratio obtains, and may have been larger: during the first apyretic interval, splenic enlargement was noted in only 15 p. c. of survivors, but in 58 p. c. of cases dying at this stage; and lastly, during the first relapse, amongst survivors splenic tumour existed in somewhat over 40 p. c.; in the fatal cases, 100 p. c.

During febrile turgescence, the spleen becomes more or less fixed, though still moving with diaphragm; and during apyrexia, it is easily displaced. After death, its projection beyond the costal margin is less obvious than during life.

Splenic Intumescence at different stages of Fever.—That the dimensions of the spleen tend to augment progressively during the pyrexial attacks, and to subside in the apyretic intervals, is shown by the following data.

Invasion-period.—Of the state of the organ during incubation-stage of man, nothing is known; only in my own late attack I failed to detect a change: the spleen seemed to be congested in a monkey killed at this stage.

First day of fever: in 4 cases known to me it is probable that subjective or objective change would have been noticed, if present: 2 children had probably a tumid and tender spleen on this day, and a man with prior malarious hypertrophy displayed in the evening-time a slight increase not perceived earlier in the day.

Second day: that the organ may be hardly or not at all affected in the morning appears from the records of 3 cases, although there might be hepatic and epigastric tenderness, and considerable fever. In one of these instances with the evening exacerbation the spleen became tender and probably enlarged, and in an infant it projected beyond the costal cartilages.

Third day: in 18 cases no change noted in 8, in 10 tenderness alone or with enlargement, both in varying amount. The act of coughing or standing and walking, may elicit a painful sensation not before experienced.

Fourth day: in 24 cases the organ enlarged or tender, or both, 13 times; it was so little changed as not to attract notice 11 times. Individual cases varied, but it was sometimes distinctly stated that the spleen was not enlarged, even with high fever: increase of sensibility commonly attends.

Fifth day: in 25 cases spleen decidedly implicated 19 times, as above, and sometimes exclusively of the liver. When the acme of attack happens now, the local suffering may become considerable; on the other hand, the organ may be hardly affected, to all appearance, even at this date.

Sixth day: in 24 cases the organ enlarged or tender, or both 17 times: little or not affected 7 times. The acme of attack being now frequent, splenic excitement coincides (*e.g.* 9 in 11 of the cases), but is not quite invariable or proportioned to intensity of other symptoms. Contact-pain may be now manifested for the first time, and the patient complain of uneasiness leading to change of posture.

Seventh, eighth and ninth day: in 14 of the 16 cases scrutinised there was decided splenic implication, and the 2 exceptions were not well expressed; the acme of attack occurred 9 times and almost invariably with splenic exacerbation. It seems that enlargement and firmness of the organ augment with duration of pyrexia, but not the local tenderness.

Critical fall.—In 32 cases the spleen was perceptibly enlarged or tender, or both, 24 times at the beginning or in course of the fall; it seemed to be unchanged 5 times and may have been so in 3 other instances. When the fall was prolonged, the organ sometimes became reduced to near normal dimensions at its end; and always volume and tenderness declined, even promptly, with progress of the crisis. In a very few cases the spleen was said not to be perceptibly changed from the normal at this time, as previously it had not been. Gastric and hepatic changes usually coincided. It has been noted that the organ became very firm at this time.

Summary.—Splenic implication consists of enlargement and tenderness, which though related, are not always proportionate to each other, or to the attendant pyrexia: contact-pain seems to be an early and a paroxysmal phenomenon, whilst enlargement is rather progressive: in general, the spleen has shown a tolerably regular augmentation of these signs in frequency, from 60 p. c. of cases on third day of attack to 90 p. c. at acme: at the critical fall (*i.e.* before its completion) splenic disturbance persisted in 75 p. c. of cases, but it speedily subsides. Several of the above statements apply to the recurrent febrile periods.

First Apyretic Interval.—The notes of 171 cases taken at this negative stage show that splenic changes were noted (being sometimes prominent) in at least 34 p. c. of cases on first day after end of critical fall, and in 19 p. c. of those on second day; but quickly became less apparent, till on the fifth day they were entered in only 4 p. c. of cases.

Even if approximately correct, the above data show a close association of the local signs with pyrexia and spirillar blood-infection; enlargement persists longer than tenderness: smart febrile rebound may take place at this time without the spleen being affected, and also local inflammations, neither complication being specific. These data reveal another fact of interest; thus, after the 5th day, *i.e.* on 6th and 7th or day of relapse in this series, the organ tends to become affected again, the proportion of instances displaying some tenderness, fulness or tumefaction rising to 10 and 15 p.c. respectively, or nearly that exhibited upon the first advent of recurrent fever: the explanation here seems to be that with the specific incubation-period (generally of about two days' duration) the spleen tends to show signs of disturbance: this concurrence may at all times be difficult to demonstrate, and, in fact, was apparent in only 3 of 12 instances noted of expected relapse ending with fever: further and closer enquiry is desirable, yet it remains a clinical fact that spirilla may be present in the blood without pyrexia or palpable splenic derangement.

First Relapse.—Of 129 observations of fully-developed recurrent attacks, decided splenic implication was present in 42 p. c.; as detailed, it was noted in 12 p. c. of cases on the first day, 24 p. c. of those on the second, in 36 p. c. on the third, in 73 p. c. on the fourth, 70 p. c. on the fifth and in 43 p. c. (cases few) on sixth day: in 88 p. c. of all cases at acme and in 60 p. c. of cases at the critical fall. This series indicates a tolerably uniform increase in splenic tumefaction, with continuance of pyrexia; yet neither at beginning nor in its course does the intumescence necessarily accord with intensity of fever, following rather the ascertained degree of spirillar blood-infection. The inclusive local signs are the same as in the primary febrile attack, being perhaps less pronounced and persistent, and varying more.

Second Interval.—The abated frequency of splenic implication contemporary with shortened duration of fever-periods, has been already indicated; and though at the critical fall of relapse the organ was still largely involved, yet towards the end of this stage (not seldom prolonged) its enlargement had often subsided or was on the point of disappearing, and consequently with reaction and rise of temperature to the normal, the presence of splenic tenderness or swelling was noted in

only one-fourth of the cases under analysis. This is a considerably less proportion than was found at the corresponding date of first apyretic interval, and it was not long maintained; being on the third day reduced one-half, and on the fourth remains of splenic implication were detected in only 2 subjects of 28 belonging to my later series: one of these was a woman of 56 years who showed on the sixth and seventh days a slight increase of the splenic enlargement, without tenderness, and no other sign of a second relapse; and the other was a man of 25, in whom an isolated paroxysm of fever afterwards occurred on the twelfth day.

In some cases, the spleen does not revert to its normal condition directly or uniformly; and traces of the changes it has undergone during the active periods of blood-infection, are not for a long time effaced.

It also appears that the spleen may continue to furnish evidence of periodic disturbances of the system, not amounting to distinct third or fourth relapses; yet to be considered as recurrent events, due to repeated auto inoculation.

Second Relapse.—In a later series of 28 cases there were 6 instances of second recurrent paroxysms, displaying the usual variety of form: 2 were fully developed relapses extending over four or five days, and of these in 1 the spleen was enlarged and tender; but not before fever set in and not so long as it lasted, diminution of the organ commencing one day before fall of pyrexia, though simultaneously with a slight decline of temperature. In the other case, the spleen had been affected with malarious enlargement; on the morning of relapse it was noted as being much reduced in dimensions, and with the advent of fever it became tender and larger: some tenderness persisted for ten days after the fall, when again feverishness came on in the form of a prolonged series of minor daily exacerbations (probably representing an abortive third relapse), and the spleen once more displayed unusual tenderness for a time briefer than the pyrexia, or not proportioned to it; its volume did not increase equally, and with the resumption of normal temperature level, was found to be less than ever before: this case is detailed as an example of conjoined malarial and spirillar infection.

There were 2 examples of second relapse in the form of brief yet smart isolated paroxysms, occurring on the 10–12 day of apyretic interval: in one of these splenic implication was not noted at the beginning of fever (spirillum present), and in the other there was increase of pain or tenderness in an organ previously not sound, with no enlargement (t. 105°·4, spirilla probably present). In a third case belonging to another series, the local symptoms, though brief, were acute enough to suggest 'splenitis.'

In other 2 instances this relapse was represented by a long, low rise of temperature coming on a fortnight after the last fall; in both the spleen was decidedly enlarged and tender, yet not immediately before the commencement of pyrexia; in one the local signs slowly declined after its cessation, and in the other a little before the final rise, not increasing with rebound and still persisting in mild degree after the fall.

The same series of cases furnished 3 instances, at least, of late periodic symptoms (about 6th day of interval), once febrile without de-

tected spleen-enlargement ; and twice non-febrile, with either enlargement or tumefaction alone of this organ. These phenomena might well be regarded as indicative of suppressed relapse.

Two examples of intercalated paroxysm happened in the same series, during the first apyretic interval ; the specific character of one paroxysm was demonstrated, yet in neither instance was splenic derangement noted before, with or after the attack ; possibly from want of sufficiently numerous observations.

I should add that the number of later febrile and splenic derangements might have been larger in this representative series, had not several patients left hospital too soon after the second relapse to permit of adequate study of their cases.

During secondary fever and that symptomatic of local inflammation, the spleen was rarely implicated to a considerable extent, and never at early date ; it is, however, possible that minute and parenchymal lesions would not be manifested by local signs, and that these 'infarctions' of the spleen could not be diagnosed during life.

I would here add that whilst splenic implication, as clinically revealed, is a most frequent feature of the spirillar disease, and even intimately associated with fatal terminations ; there is evidence of its not being a strictly invariable attendant. In the monkey, I never found the spleen much changed in aspect ; and I should regard all extremer alterations of this organ in man, as truly incidental results of infection : this view does not affect the clinical interest of the present subject, for it may be said that marked or serious splenic implication is always contingent.

The anatomical relations of the spleen and adjoining viscera of abdomen and chest, become of practical interest where one or more of these organs are so much altered in volume, weight or sensitiveness as to displace or disturb others near them. There are well-known illustrations of this subject in several local and general diseases ; and, without being peculiar, they were frequent during the course of spirillum fever.

Clinical Review of the chief Abdominal Symptoms. General frequency :—Limiting attention to the contents of the three regions composing the upper abdominal zone, I find at invasion-attacks the following percentage of entries amongst patients surviving :—Spleen 70, Liver 66, Epigastrium 40 ; and in first relapses the following :—Spleen 45, Liver, 32, Epigastrium 30 p. c. Therefore, all of the organic disturbances implied are fewer during the relapse, the diminution being most apparent as regards liver and spleen. Complete information respecting the commencement of invasion-attacks is still wanting, and might lead to a modification of the preceding statements ; yet not, I think, to their subversion.

General course of local changes. Invasion-attack.—There is but a moderate increase of symptoms with progress of fever, from third to seventh day or later. At the acme, a common exacerbation is clearly indicated and probably is never wholly wanting. Even during the crisis some symptoms persist until its end.

First Interval.—The chief feature is a gradual yet prompt subsidence

of local symptoms from the first day to the seventh ; an exception concerns the spleen, which shows a distinct renewal of disturbance in the two days preceding relapse, or at corresponding dates when no febrile recurrence takes place.

First Relapse.—Without absolute similarity there is conformity in the three groups, the common feature being a gradual accession of symptoms from first to sixth or seventh days ; at the acme and fall exacerbation was noted, but the total derangement is decidedly less than at invasion. During the succeeding apyretic period, the spleen, chiefly, displays some of the previous changes.

Comparative frequency.—Amongst themselves these several groups of symptoms at febrile periods maintain a ratio, which is almost the same for first and second attacks ; viz.—Splenic 40 to 42 per cent. of total symptoms, hepatic 35 to 30 p. c. and epigastric 28 to 24 p. c. (first nos. are those of invasion).

Combinations.—In general, none of the three groups existed alone is more than 5–7 p. c. of all positive instances ; the exception being that during relapse, the spleen was not seldom the sole organ affected. Using the expressions L. S. E. for hepatic, splenic and epigastric respectively, the most frequent combination was S. L. E. (30 p. c. invasion and 22 p. c. relapse) and next S. L. (30 p. c. and 15 p. c.) ; L. E. was seldom seen alone (6–9 p. c.), and during invasion S. E. never ; but during relapse in about 8 p. c.

Comparison of the preceding data with those taken from fatal cases and autopsies.—A thorough comparison of the clinical phenomena presented by survivors and by those dying, is not here attainable. Fatal cases were often first seen in a moribund state, or they were complicated with symptomatic fever ; hence, but a small residuum remained available for analysis. The general result, however, appears that splenic symptoms were noted in 60 p. c., at least, of all casualties ; and the hepatic in 65 p. c. ; the epigastric were seldomer noted. As most casualties happened during, or immediately after, the invasion-attack, these numbers agree fairly with those then given for survivors ; and they indicate that the difference here, resides not in frequency so much as in intensity, of the local symptoms.

The other supplemental series of autopsic data has been already referred to ; after the necessary sifting and arrangement they, too, prove to be scanty, only showing that whilst a certain range obtains in the structural changes and dimensions of liver and spleen, there is yet a main accordance with the life data, as regards the increase in volume of these organs towards close of the febrile attacks.

The inference follows that, as regards the abdominal organs chiefly implicated in spirillum fever, a close similarity obtains between frequency of symptoms and (in all probability) of structural changes, in both survivors and those dying from fever alone.

16. The Urine.—Though there were some unusual obstacles to full enquiry here, owing to native prejudice or inattention, and the want of adequate assistance ; yet a few interesting data were acquired, which may be arranged as follows. Quantity, aspect, reaction and specific gravity of the urine at different stages of illness.

Febrile attacks.—Respecting the course of invasion only scattered information was obtained, prior to the acme and fall. In 9 typical cases, the quantity was usually diminished, but twice reported as copious, aspect little changed or high-coloured, sp. gr. 1010–18 (mean 1015), reaction commonly acid, bile ingredients present, chlorides diminished, traces of albumen rare (1 in 6). I do not recollect seeing in a native patient the scanty, red urine, of high specific gravity and with brick-dust sediment, which is found in other febrile diseases; and there did not appear any definite connection of urine-changes, with prominent or unusual symptoms.

At or near acme, the urine was usually scanty, high-coloured, clouded yet without sediment, reaction acid, sp. gr. 1010–18; no lateritious deposits on cooling were seen, and very seldom the frequent micturition due to an irritated bladder. Mucous cloudiness always scanty, and degree of acidity never much pronounced: a sense of scalding was once mentioned with urine clear, though high-coloured, sp. gr. 1019, no albumen present: once the urine was very copious, sp. gr. 1010. Even at this acute stage of fever, albumen is not always to be detected by ordinary means.

With the critical fall, the amount of urine varies; thus, it may be plentiful, but is commonly scanty and occasionally secretion seems to be suspended for a time, no urine being present in the bladder, and the general aspect of the sufferer approaching to that of a cholera patient in the state of collapse. The quantity of urine passed at this stage did not appear to be regulated by the copiousness of cutaneous transpiration; or, so far as ascertained, by the amount of fluid ingesta. Once the sp. gr. was 1020, and chlorides in excess: other instances were the following:—

F., 35, the temp. during night falling from 104°·8 to 97°·2, with profuse sweating, 35 ozs. of urine were passed, high-coloured (bile-pigment), somewhat clouded yet without sediment, acid reaction, sp. gr. 1006: she was particularly disinclined to drink water, but had extra milk allowed.

M., 35 (her relative), the t. falling at night from 105° to 99° without sweats, 15 ozs. of urine were passed in the morning, high-coloured, cloudy (no sediment whatever), sp. gr. 1010, acid in reaction: at 4 P.M. the temp. had sunk to 95°·6 and no urine was seen until midnight. Sheer depression, the spleen large, pupils normal and no head-symptoms. For one instance in which at beginning of the fall, the urine was dark as if from the presence of blood (urates only being found) see CASE XXII. detailed above under 'Lysis.' Real hæmaturia was never observed in Bombay: nor any other example of critical urine-changes.

At reaction after crisis, the urine is apt to be scanty, then attaining its highest known density, or a specific gravity of 1015 to 1022 (seldom the higher number); its quasi-febrile aspect being retained.

When the temperature persists at its lowest for one or two days, the urine continues as above; and with systemic reaction I have noted a brief interval, on the second or third day, where the secretion is scanty, yet pale and of very low density; afterwards its amount increases, and the general aspect of non-febrile condition is assumed. For a striking instance of prolonged depression and anuria, without any signs of uræmia, see CASE XXI. above, under 'Crisis.'

At both acme and fall of the invasion-attack, careful microscopical search was made on several occasions, for evidence of organic renal degeneration amongst native subjects ; the result being always negative.

First recurrence.—From fuller data of a well-developed relapse occurring in an adult woman (CASE IX.), it appears that during fever the quantity of urine passed diminishes from first to last ; in her case being 66 ozs. on the first day, 60 on the second, 31 on the third, 30 on the fourth and 28 on the fifth or last day prior to fall ; its specific gravity at the same time rose from 1006 to 1013, augmentation at the last being most decided. On the second morning the pale tint became deeper, and from the second evening the urine was high-coloured, being also very clear ; it was very high-coloured (or almost red) on the last morning near the time of acme, yet showing no sediment on long standing (no blood present) : the urine was cloudy from the second day onward, but never furnished a deposit on standing : it was distinctly acid throughout.

Microscopic examination of the deeper strata in specimens placed at rest, was carefully made, towards the close of the attack especially, and always with negative results as regards the evidence of renal disease: scanty dumb-bell crystals of urates, or triple phosphate, and a few bacteria, with, on the last day, a little squamous epithelium (vaginal) were alone detected, and not a sign of renal cell or tube-cast. Even when albumen was present distinctly in the urine, organic débris were not seen ; and, I may add, this holds good for the critical fall.

During a relapse lasting part of two days (acme 104°, spirilla in the blood) the urine was diminished somewhat in quantity, though still copious, its specific gravity hardly raised (1004 to 1006), its hue altered to yellowish or brownish and its aspect became clouded for a time : the reaction was always acid : patient a male adult.

In the case of a lad under close observation, the urine was copious on the first day and less pale than before, soon becoming tinged yellow (no play of colours on addition of nitric acid), there being some hepatic tenderness but no perceptible jaundice : specific gravity at first not increased : changes on the second day (high fever still) were not more marked, there being noted a tendency to rapid decomposition ; on third day, urine scanty, high-coloured, clouded, sp. gr. 1015, and scanty at the acme : here, too, microscopic examination gave negative results.

A Mussulman adult furnished urine on the first rise of temperature copious, pale, and of rather less density than before (1007) ; on second day, it was clouded and denser (1012) ; on third day high-coloured, cloudy (no sediment) and sp. gr. 1017, bile-acids and bile-pigment being present at this time and on next day ; on the fourth, still plentiful (57 ozs.) and high-coloured ; on fifth day it again became light-coloured, though clouded, and was copious (sp. gr. 1010-15) ; at this time the liver seemed nearly normal whilst there was deep jaundice, and the man passing into the typhoid state, e. t. 105°·6 : on the following night 30 ozs. of high-coloured urine were passed, sp. gr. 1015, clouded, acid, no albumen ; the brief acme then ensued, and fall.

The general state of the urine at acme was seldom to be learnt : in an adult woman the secretion passed soon after this epoch measured

16 ozs., was very high-coloured (red), clouded (no sediment), acid, and sp. gr. 1013.

When the temperature went down, the urine for a short time changed its characters, becoming paler and of reduced density ; in one instance, it was also copious (39½ ozs.) sweating not having set in ; and in another the amount was only 12 ozs., and sweats were reported to be scanty : in both cases, the urine was pale or very pale, and had a sp. gr. of only 1003.

When the fall was established and whilst it persisted, the urine was more or less high-coloured and comparatively dense, sp. gr. 1013 to 1017, clear or clouded (no sediment) and below the normal amount ; before reaction had fully set in, slight variations of aspect were noted in the three cases under analysis ; and in all, about the 2nd or 3rd day, there was a final brief reversion to the characters of febrile urine, in so far that the secretion became scantier, high-coloured and of increased density, the maximum of 1022 being noted in one instance at this time ; bile-pigment abounded, and once the urine showed a tendency to decompose early.

Non-febrile state.—During the day or two following crisis of both invasion and first relapse, the urine has been found scanty, high-coloured and of increased density (1014) ; it then, whilst still of small amount, became pale and of low specific gravity, for a brief period ; again reverting to a quasi-febrile aspect, without any corresponding change of temperature. At this time there was no microscopical evidence of renal disturbance, only a few crystals of uric acid and urates, or of calcium oxalate or triple phosphate, with a little squamous epithelium, being noted. When rallying became established at near normal state, the urine forthwith was plentiful and of low density (1004–8). These data are taken from CASE IX., and they were generally confirmed by other ordinary examples of relapsing fever.

So far as seen, the first relapse was rarely preceded by any change in aspect of the urine ; the quantities passed on the 1st and part of 2nd days of fever were higher than the preceding mean, showing the effect of pyrexia not to be immediate here. In a lad a few hours before fever returned the urine was scanty, pale, sp. gr. 1018, clouded and slightly ammoniacal (albumen none), though passed only an hour previous to examination ; spirilla were already present in the blood.

The second relapse was deferred and not marked in CASE IX. : the amount of urine passed seemed to augment for 13 or 14 days after the last crisis, and about the 7th and 8th days its density became temporarily increased (1010), then reverting to the average (1006) ; and during the low and irregular febrile movement ensuing, no change was apparent in its aspect.

In the instance of an adult man (Hindoo) the urine at crisis became scanty, only 20 ozs. being found at close of the fall, the amount then suddenly rose to 112 ozs. and it soon became even more excessive, so large was the daily consumption of water (average 83 ozs.) he voluntarily imbibed : after a brief relapse, the daily amount of urine passed became less, yet at the end of a fortnight it was 130 ozs., the tint being very pale, and specific gravity 1002–1006.

In the case of an adult Mussulman weaver this excessive dilution

was not noted, though the quantity of urine passed was probably above the average ; and the same remark applies to the instance of a Mussulman lad, whose urine also showed a tendency to rapid decomposition.

This spontaneously increased flow of urine after fever is noteworthy : low specific gravity, diminished acidity and liability to become ammoniacal often concurred.

Special Analyses.—In two ordinary cases of Hindoos, the urine was examined twice daily for urea, according to Davy's hydrobromic method as modified by Russell and West. The food during fever was the hospital milk diet, estimated in the dry state to furnish daily N. 175·6 grs. and C. 2988·43 grs. ; and during the non-febrile state, it was the hospital ordinary C. diet, supplying N. 195 grs. and carbon 3430 grs. daily : a few ounces of extra bread and rice, or meat and soup, were allowed, with small quantities of stimulants as needed.

Neither patient was seen until after a week's illness at home. In the man's case at critical fall of invasion, the urea promptly increased to about 500 grains ; for the next ten days, the mean daily amount was a little over 200 grs. ; with a brief though sharp relapse lasting part of two days, the quantity arose to 430 grs. and thence it declined slowly in the course of a fortnight, the daily mean being about 170 grs. : body-weight about 133 lbs. : physique and appetite good.

The woman's case is that described as No. IX. ; particulars are shown in the Table below, and will serve to convey a general view of urine-changes. The amount of urea rose at invasion-crisis to 220 grs., thence gradually declining till just before the relapse, when the daily excretion was only 76 grs. With onset of fever the quantity promptly augmented ; it was large during pyrexia and till second day of crisis—maximum 226·5 grs. ; and it then gradually declined. Diet during the first apyretic interval was the C. above-named, and yet the amount of urea was so small as to attract attention : and even if the data are only approximatively accurate, this feature is striking. During the first relapse, a liberal supply of food was ordered (though I cannot say if all were eaten) and the urea became increased ; the patient, however, lost 10 lbs. or more than one-eighth of the total body-weight, at this time. There occurred a slight but prolonged second relapse (spirillum not seen), during which the amount of urea increased at first, though extra articles of diet were reduced : this fact shows the influence of even a moderate degree of fever. It is also obvious, I think, that a plentiful flow of urine corresponds (as might be anticipated) with comparative abundance of urea—compare the figures of first and second post-febrile intervals. As the patient at last was gaining weight, it seems likely that the urea then being eliminated had previously accumulated in the system. There were no head-symptoms during the earlier scanty excretion of the urea ; but at the second crisis, with some distress, the quantity of urea was temporarily diminished. Patient a small, thin woman, with poor appetite.

Chlorine.—The chlorides were precipitated by the addition of a weak solution of nitrate of silver, to urine presenting an acid reaction ; the proportion of precipitate to volume of urine being then measured

off. No attempt was made to ascertain the actual amount of chlorine eliminated. In CASE IX., the silver solution produced change of colour to deep brown, purple or green, and dense clouding; precipitate scanty on standing: this singular reaction began on second day of relapse, and was repeated until critical defervescence when it promptly ceased; it was noted also at the close of invasion-attack on her admission: bile-pigment and ordinary albumen were not apparently concerned. In this

TABLE II.—MEAN DAILY DATA OF URINE, F., 35. BODY-WEIGHT ABOUT 75 LBS. DIET MIXED.

Stage	Temp. F.	Ozs.	Sp. gr.	Urea grs.	Chlor. prop.	Album.
End of Inv. .	104·6	29	1010	131·6	?	$\frac{1}{6}$
Crisis . . .	96·6	57	1008	220·	?	trace
	96·5	24	1009	141·6	1·7	none
	97·3	42	1006	188·4	7·5	„
	98·	32	1005	102·9	5·8	„
	97·8	34	1005	89·7	6·4	„
	97·7	45	1005	85·1	7·9	„
	98·2	54	1007	76·	5·8	„
Wt. 76 lbs. .	102·2	66	1007	174·2	10·	„
Relapse 1 . .	104·3	60	1010	213·8	6·9	„
	106·	31	1011	153·3	3·6	„
	105·2	30	1011	207·4	?	trace
	104·6	28	1013	212·7	?	?
Crisis . . .	96·8	31	1013	180·	+ 8·	none
Wt. 66 lbs. .	96·8	26	1013	226·5	5·3	„
	97·2	33	1009	152·	5·9	„
	97·4	64	1005	197·1	8·	„
	98·3	52	1006	130·	6·5	„
Wt. 73 lbs. .	98·1	64	1006	155·4	8·6	„
	98·4	72	1006	183·7	4·1	„
	97·9	80	1007	158·4	5·	„
	98·6	63	1007	110·8	4·	„
	98·4	96	1007	157·6	5·9	„
	97·7	72	1007	183·7	6·2	„
	98·6	84	1007	166·3	9·	„
Relapse 2 . . (slight).	98·5	91	1006	228·2	5·9	„
	98·9	87	1005	153·1	6·8	„
	99·3	83	1005	142·4	6·	„
	99·6	87	1006	172·2	7·2	„
	102·	87	1006	172·2	5·4	„
	98·6	72	1006	177·4	4·5	„

instance the chlorides diminished from first to last day of the relapse; and in another case, they greatly declined during a long recurrent attack. During a brief recurrence of a day and a half, they seemed to increase promptly, then greatly declining the day after. At the critical fall of both invasion and relapse, the chlorides at first were absent, as a rule; with rallying, they reappeared either slowly or quickly; e.g. at the woman's first crisis gradually, at her second very promptly and in

considerable excess ; also in excess in the slower fall and rebound of relapse, in an adult man ; and greatly in excess on first day of crisis in a lad.

The total proportion of chlorides was roughly estimated for Table II. (p. 122) by dividing the total daily urine by volume of silver precipitate, and so in some other cases, the result being a general concordance in the above data, and it appeared that for some days after crisis, there occurred an intermittent increase. As observed with urea, a plentiful flow of urine generally corresponds with augmented elimination of chlorine.

Abnormal urinary ingredients.—Bile-acids : from a few observations made it seemed that the presence of bile-acids (generally detected) was limited to the later part of febrile attacks, with the ensuing fall. Bile-pigment was also frequently noted in excess, at the time of crisis ; both these indications concurring with known liver-changes. In jaundiced patients the above-named ingredients were abundant in the urine, and apparently not more so in the casualties. Bile and albumen did not necessarily concur.

Albumen.—In small proportions was not uncommon—perhaps even frequent, chiefly at the close of pronounced febrile attacks and a little later, the first attack most generally : its amount seldom exceeded $\frac{1}{6}$ vol. of urine, and might be less in even severe cases. Almost never was there other evidence of acute renal congestion (such as blood-discs and tubular casts) : when present, albumen was not necessarily associated with marked or peculiar symptoms, and these might exist without its being found. The form of albumen precipitable by heat and acids, is here alone referred to. I may add that collections of micrococci and fine granules have been seen in the urine, which resembled in size and form tubular 'casts' ; these seemed to be only incidental.

The presence of sugar, as ordinarily tested, was never made clear, at any stage of fever.

Fatal cases.—The aspect of the urine has not been found to differ remarkably : there are no complete data respecting the amount of urea : albumen, if present, was never strikingly abundant, even when the kidneys after death were distinctly implicated ; and it may be absent. Granular disease of the kidneys was seen only a few times at autopsy of native patients, and on one of these occasions the urine was found to contain a small amount of albumen, with a very few hyaline casts ; the lesion was evidently chronic. I am not aware of fallacy lurking here, if such exist ; but whilst the renal secreting epithelium is clearly liable to serious change, the evidence of increased blood-pressure was not, to my apprehension, nearly so clear, and in all cases, both surviving and fatal, the urine-changes in relapsing fever (like the symptoms comparable to the uræmic) have appeared to me singularly slight.

CHAPTER IV.

DESCRIPTION OF THE PYREXIA.

DEFINITIONS. NORMAL TEMPERATURE AND PULSE. ABORTIVE AND RELAPSING SERIES : PYREXIA AT SUCCESSIVE STAGES OF ORDINARY SPIRILLAR DISEASE : COMPARISONS OF T. AND P. VARIATIONS OF PYREXIA AT THE SUCCESSIVE STAGES, AND IN FATAL CASES.

Temperature-definitions.—By the terms ‘rise, and ‘decline’ or ‘fall,’ is meant elevation above or depression below the normal extremes of 99° and 97° F. : a ‘remission’ is a decline not amounting to that complete suspension of fever, which is expressed by the term ‘intermission’ : fever is ‘continued’ when the remissions do not commonly surpass the normal mean range of about $1^{\circ}\cdot5$ F. : it becomes ‘remittent’ when the majority of febrile declensions exceed 2° F. ; and it is ‘intermittent’ whenever the abatement descends to the normal mean of 98° F., or below this.

Duration of febrile attacks.—This must often be arbitrarily reckoned, from the difficulty of learning when fever precisely began or ended ; and also from the actual duration of pyrexia not being necessarily limited by the current account of days. Usually I have included in my estimates the first day of decided rise, as well as the day of critical fall.

Kind of heat.—Whilst the actual temp. attained was probably as high amongst Natives as in European patients, yet I rarely noticed, by touch, the different modifications of heat usually mentioned in English works on Fever : the heat has, however, seemed ‘burning’ in children ; and, contrarily, the soft integument of some dark-skinned adults has felt to me only warm, whilst the thermometer marked as high as 105° F. To the cool palm of Native clerks the skin at crises has felt warm, when to myself it seemed cold : such differences are readily explained on physical grounds.

Irregular distribution of heat.—In advanced periods of fever, a decided difference might be felt by the same observer between the temperature of trunk and limbs, especially the hands and feet ; and this local variance might be made aware to the patient himself, as I happen to know. It is verifiable by the thermometer : *vide* CASE II., at relapse.

At the critical fall, I have found a temperature in the mouth and rectum, hardly above that of the axilla.

Exaggerated sense of febrile movements.—Several times intelligent patients described their primary fever as having been like ‘ague’ ; yet never that I recollect, was the course seen in hospital of such intermit-

ting character. I have, however, noted in some cases of well-sustained pyrexia that chills, and even rigors, were complained of as attending mild exacerbations ; and even frequently they were noted at the acme of attack, when, to all appearance, the temperature did not rise proportionately high. Free sweating, also, may attend moderate remissions ; and hence, ordinary statements of the sick need cautious interpretation.

Another source of fallacy was the history given of long duration of fever prior to admission ; 15, 20, or even 30 days have been named by patients, when judging by more valid data, such long periods of continuous specific pyrexia were in the highest degree unlikely. I have not made use of these cases for analysis here, judging them to have been instances of relapse with apyretic intervals disregarded, or complicated by other than specific fever. Such fancied extension of illness has been noted in ordinary typhus cases.

Patients being admitted at different periods of illness, it became necessary to consider their state when first seen, before deciding upon the intensity of pyrexia ; for not a few were brought either worn out by suffering, or so exhausted by want and exposure, that the general depression was considerable and but slowly rallied from.

All temperature observations were made with good English thermometers, placed in the axilla under required conditions : possibly the readings were sometimes within actual limits, but this seems unlikely in the warm and uniform climate of Bombay. Cases for illustration were selected, and the figures are quoted precisely as entered.

The pulse reckonings are simultaneous records ; and these I have decided to retain, knowing them to be of some value. The rule was to take the pulse after an interval of rest, in the recumbent posture. I am aware that many circumstances tend to excite and some to depress, the heart's action ; and, also, that higher velocities are difficult to ascertain with accuracy : whenever practicable, I personally tested exceptional instances. The qualities of the pulse are described under a separate heading : see the last Chapter.

It fell within my experience to note that unless the temperature readings be carefully and punctually entered, even observers of large experience might fail to recognise the peculiarities of true relapsing fever. Where malarious fevers are the common type, some additional attention is needed for apprehension of new forms of fever ; and I early resolved not to make use of any hospital records wanting in clinical essentials.

Normal Temperature and Pulse of Natives of W. India.—During September (a sultry month) observations were made on 6 healthy males of ages ranging from 12 to 40 years, in the same manner as those made on the sick, and at similar 3-hour intervals throughout the entire day : these individuals were hospital servants, clad and fed as usual, each being subjected to 2 days' continuous examination.

Temperature.—Considerable uniformity obtains in actuals, means, range and hours of change ; the main result showing a body-heat of the scantily clothed native rather below that of the inhabitant of England ; but following a similar daily course. The air at Bombay was moist, and

varied very little from a daily mean of 80° F. Hindoos take two meals daily, before and after noon. Total observations 102.

Degrees.—The 8 daily 3-hour means were as follows, beginning with the minimum at 4 A.M., and ascending to the maximum at 4 P.M., and thence onward through decline at night:— $97^{\circ}18$ (min.), $97^{\circ}38$, $97^{\circ}91$, $98^{\circ}11$, $98^{\circ}33$ (max.), $98^{\circ}05$, $97^{\circ}7$, $97^{\circ}35$: mean range $1^{\circ}15$: actual maximum $99^{\circ}2$, actual minimum $96^{\circ}6$, absolute range $2^{\circ}6$. The mean of all maxima $98^{\circ}6$ (range 98° to $99^{\circ}2$), of all minima 97° (range $96^{\circ}6$ to $97^{\circ}4$); these data showing but narrow variations. Total daily mean t. $97^{\circ}78$ or about 7° less than the accepted English mean.

Movements.—Whilst the mean maximum was at 4 P.M., the tendency was to somewhat earlier rather than later hours; and so the minimum tended to occur rather before 4 A.M. Twice in 12 observations, a temperature equal to maximum was recorded at 10 A.M., or about the hour of morning meal; once the A.M. decline of body-heat was prolonged to the same hour.

Variations within the limits named, were numerous; but for the purpose in hand, they do not call for record.

Pulse.—It seems desirable to append some normal data for comparison with the sick statistics. Observations 102; made on subjects at rest in recumbent posture.

Pulsations per minute.—Inclusive means, beginning at 4 A.M. and proceeding at 3-hourly intervals:—68 (min.), 72, 77, 78, 79 (max. at 4 P.M.) 78, 72, 68: the daily mean 74, range 11. Maximum 104, minimum 46, extreme actual range 58: mean of maxima 86 (range 104–66), of minima 60 (80–54).

Daily course.—In general, the pulse follows the temperature throughout, and at extremes: the slower pulses tend to quicken early in the day and soon attain their maximum, whilst the quicker rising at about 10 A.M. (possibly with food-ingestion) longer maintain their greatest velocity, sinking more promptly, but not below the common mean.

Variations.—Are numerous, and from them it appears that the natural velocity (or natural irritability) of the pulse is a datum variable in itself, and distinct from contemporary state of body-heat, within certain limits. During febrile illness, personal idiosyncrasies were still manifest.

Preliminary general Remarks on febrile Temperature-charts.—

The intervals of ordinary 7 A.M. and 4 P.M. observations being 9 day-hours and 15 at night, temperature movements then occurring remain practically unknown; and such defect is but partially remedied by the eight 3-hourly observations, which were all I could attempt. The need here of a self-registering apparatus capable of acting for a whole daily cycle, is very apparent.

Upon arranging my data, I find that by the ordinary bi-daily method whilst the general course of pyrexia is fairly indicated, yet maxima and minima, prolonged exacerbations and minor perturbations of specific fever may be overlooked in the lopping off (as it were) contingent on this method; the resulting deficiencies being identical and equally evident, in charts of both man and the smaller animal. Thus, of 11

brief, isolated paroxysms noted in the monkey, only 3 were fully shown and 8 would have been much reduced or effaced, in bi-daily observations : also 7 prolonged attacks would have lost their prompt beginning, smaller day-movements and prolonged night exacerbations, with the final perturbation and full degree of critical fall. In the human series, a brief night paroxysm would have escaped notice ; also the really variable course of an apparently simple one-day paroxysm ; and during longer attacks preliminary or abrupt initiatory rises, high night temperature, day perturbations, sustained e. elevations, slighter remissions, the critical perturbation and full amount of fall, would have been overlooked.

Important as are the above considerations, it is not necessary to discard the usual Chart-forms, for these must yet compose the bulk of clinical data, and do in fact convey valuable information as to the degree and duration of specific fever ; their general concordance with preciser observations, being shown by their furnishing very similar m. and e. dates for most chief febrile phenomena.

Specific Pyrexia.—Experience in Bombay agreeing with that elsewhere, all ordinary surviving cases of fever may be arranged as follows :

1. *Single attack: Abortive form*—probably uncommon.
2. *Recurrent or Relapsing form* :—
 - with 1 Relapse—the most frequently seen.
 - 2 Relapses—less frequent.
 - 3 Relapses—rare.
 - 4 Relapses—very rare.

More multiple recurrences were not met with in hospital, except on hearsay evidence.

The distinction of two main forms has been a compulsory one, since in a practical sense the fever does or does not return ; and recurrences which are not seen by the medical attendant, or not felt or recollected by the patient, may be regarded as non-existent. Although I have freely allowed that, in a theoretical sense, truly abortive infection may be extremely rare (if it ever occur) in man ; yet in the lower animals it seemed to be the rule : and, therefore, the clinical discrimination now made, may be as correct as it is convenient.

It is proposed to describe, in order, the several forms of specific pyrexia, with their commoner varieties.

The following Table III. applies to survivors admitted at all stages of sickness, casualties being omitted. The entries are given just as heard, or as estimated at first view ; separate analyses were afterwards made, and these are not found to differ very considerably.

The Average duration is that of greatest number ; the Mean duration is calculated from total actual days divided by total cases, all unknown instances being first excluded. As the extremer duration of invasion and relapse rests solely on hearsay, most probably the mean is here rather over-stated.

TABLE III.—ESTIMATED NUMBER AND DURATION OF EVENTS IN ORDINARY RELAPSING FEVER.

Days' Duration	Invasion		1 Int.	1 Rel.	2 Int.	2 Rel.	3 Int.	3 Rel.	4 Int.	4 Rel.
	Abort.	Rel.								
1	—	—	—	6	—	16	—	1	—	—
2	—	—	—	15	1	3	—	1	—	1
3	—	—	3	30	—	7	—	1	—	1
4	5	7	7	52	1	6	—	1	1	—
5	7	18	15	64	2	4	—	1	—	—
6	15	16	41	45	4	1	—	—	—	—
7	22	45	59	12	3	1	—	—	—	—
8	16	35	34	11	5	2	1	—	—	—
9	19	33	31	—	5	—	1	—	—	—
10	8	31	11	—	7	1	—	—	—	—
11	10	7	3	—	1	—	—	—	—	—
12	3	7	2	—	3	—	1	—	1	—
13	3	2	1	—	3	—	—	—	—	—
14	1	1	1	—	2	—	—	—	—	—
15	2	1	—	—	3	—	—	—	—	—
16	2	3	—	—	1	—	1	—	—	—
17	3	—	—	—	1	—	—	—	—	—
18	1	1	—	—	—	—	1	—	—	—
?	6	44	—	—	8	9	—	—	—	—
	123	251	208	235	50	50	5	5	2	2
Average	7	7	7	5	10	1	—	—	—	—
Mean .	8.6	8.2	7.3	4.7	9.7	3.1	12.6	3	8	2.5

1. *Abortive form.*—Judging from the mean and average duration of demonstrated intervals between first and second attacks of spirillum fever, after a period of 10 days a relapse is very unlikely; and since patients could not always be induced to stay in hospital longer than this time after the crisis of their first attack, I had to regard all instances of non-recurrence within 10 days as examples of the abortive form: in practice, a period of 8 days was almost equally valid. Examples of invasion-attack stated by the sick to be of over 10 days' duration are also here excluded, because it was possible they might, through oversight of non-specific intervals, include part of the relapse. Thus arranging, I found 98 non-recurrent attacks in a total of 411, or a proportion of 23.8 per cent., which is not inconsiderable: again, a series of 42 well-known examples of contagion and inoculation in hospital furnished 9 abortive attacks (21.4 p. c.) or if exclusion be made of 3 instances seen for a less time than 16 days after crisis, this number is reduced to 6, equal to 14.3 per cent., which gives a ratio still in excess of proportions noted of late in Europe.¹ Of the reality of this form of spirillum fever I had

¹ The older series of cases (1843-6) quoted by Dr. Murchison (*l.c.* p. 379) gave a proportion of non-relapses close on 30 per cent., whilst a later series (1869-70) furnishes only 11.5 per cent.; and a recent author (Dr. Litten, *l.c.*) mentions a ratio of only 1.5 per cent. It is uncertain if these differences point to real variations in the several epidemics concerned, or depend rather on methods of reckoning: the numbers quoted in the text do not include

personal proof in my own last attack ; and have shown it to be the commonest, if not exclusive, form witnessed in inoculated *Quadrumania*.

The insulated or solitary event is a characteristic phenomenon, presenting a defined and compact febrile attack which is attended with the usual symptoms, lasts nearly a week and leaves as abruptly as it came on. Practically, no constitutional disturbance preceded, and little or none may follow ; and, briefly, there has happened a prolonged febrile paroxysm having for its pre-eminent sign an abundant foreign growth in the blood. When, as usual amongst paupers, the attack was more severe, it still retained its defined character ; and the additional symptoms seemed due to personal influences, whereby functional derangements passed into organic lesion, or fresh complications arose. As a rule, the longer the duration of the attack the more serious its effects—a difference of even 1, 2 or 3 days being of import here, especially when the prolongation was attributable to defervescence by lysis, in place of the prompter critical decline. About a quarter of the total cases were 'mild,' five-sevenths 'marked' and one-twentieth 'severe.' The monthly majorities were entered in September 1877 and May 1878, a divergence which taken in combination with the maintained uniformity of character, sufficiently shows the slight import of annual and epidemic seasons : nor was a determining influence apparent of age or sex, personal or social condition, or probable source of infection. I did not perceive any fundamental difference between these single primary attacks and those followed by relapse, and this circumstance might be urged against such distinctions as are now attempted ; yet apart from the manifest desirability of some clinical division of the subject, it seems highly probable that a real difference does obtain in either parasite or host. Lastly, I note that more than one-half the total deaths at Bombay occurred near the close of, or immediately after, this first or invading attack, which thus became the only one visible to the physician ; and were I to add such fatal cases to the surviving total, the proportion of single attacks would become distinctly raised.

2. *Relapsing form.*—That the term 'relapse' should be held to mean a recurrence of specific pyrexia, seems a natural proposition ; yet much discrimination is needed here, for other than specific paroxysms may occur during the course of illness, which in common parlance might also be called relapses ; and, indeed, probably have been so regarded. According to my experience a true recurrent attack is always periodic, is less pronounced than the preceding attack, is essentially free from complication with other disease, and is attended with visible blood-contamination.

The successive repetitions of fever always tend to become briefer, less sustained and longer separated ; their series may be early checked or abruptly cut short ; hence it happens that no relapse, whether first, second, or third, possesses a constant character ; an abbreviated first recurrence, for example, presenting the contour of an ordinary third ; and it is impossible to predict the number or severity of relapses from

instances of mere hearsay evidence. In Finland, Dr. Holsti (see Ziemssen's *Cyclop. Supplement*, 1881) noted only one crisis in 20 per cent. of a large number of cases seen during 1876-7.

the symptoms which have gone before. The many complexities of this subject will, therefore, be obvious; and, at present, some are still without a clue. Table III. displays the comparative number and intensity (as measured by duration) of successive attacks, according to common experience at Bombay. The practical difficulties in accurately estimating the number of recurrent events in a large series of cases, I found to be insuperable.¹

Relapses occur nearly equally in both sexes, at all ages, and in weak and strong subjects; in the adynamic, typhous, and icteric types of fever, they may be quite distinct, or even severe: their frequency appeared similar throughout the epidemic. The irregular occurrence of relapses under identical outward conditions, was illustrated in the lists of individuals affected by contagion in hospital; and in other compact series of cases, where uniformity might have been anticipated. Nor does the number of recurrences in each case seen, follow any rule; except that only a single relapse commonly occurs (near 70 p. c. of all recurring attacks), with subsequent perturbation too slight for notice or practically absent.

Upon revising my detailed lists of 206 selected instances of Relapsing fever seen during 1877-78, I find 51 (or 24·2 per cent.) were possibly second relapses; but if all forms of febrile paroxysm after close of first relapse are to be regarded as representing a second relapse, then the number of second recurrences would become nearly as large as that of the first. During 1879, I had under care 28 other instances, which included 3 examples of proved second relapses, 3 of intercalated paroxysms similar to an additional relapse, and 3 of febrile paroxysms undetermined in character: then subtracting these 9 cases for comparison, there remained 18 instances, but of these 6 had to be rejected because the patients would leave hospital sooner than 10 days after end of first relapse, the mean stay in hospital of the remaining 13 being 18 days: there was now a total of 22 cases, and if all the above febrile events be regarded as relapses, there would be reckoned 41 per cent. of second recurrences; if only the 3 first-named, the proportion would be less than 14 p. c., and if the first 6, it would be about 27 p. c. Lastly, there was available a series of 31 instances belonging to the contagion-in-hospital list of cases, which includes 6 examples of second relapses, viz. 2 of intercalated paroxysms and 4 of proved recurrences: on deducting these 6, of the 25 remaining 16 only were suited for analysis, their mean stay in hospital being 20 days after end of first relapse, and never less than 10 days: if all the above 6 events be regarded as relapses there would have happened 27 per cent. of second

¹ As illustration of varying opinion in Europe, I may refer to Dr. Murchison's work (*l.c.*) where the proportion of relapsing cases recorded 35 years ago is named at about 70 p. c., some authors giving a second relapse in 5, 7 or 9·4 per cent. of all recurrences: about 10 years ago the relapsing form was estimated at upwards of 95 p. c., in one instance second relapses amounting to 8·1 p. c. of this: and still more recently Dr. Litten (*l.c.*) at Breslau found 98·5 p. c. of relapses, of which 43·5 were first, 35·5 second, 1·75 third and 75 fourth relapses. At the close of 1877 my own estimate of Indian experience was about 75 p. c. relapses, of which 62 p. c. were 1st, 6 second, 5 third and 2 p. c. fourth; the subsequent data of 1878-79 are embodied in the text. Here there is noticeable, either change in disease, or difference of reckoning, with advancing years. Bombay results seemed most in accordance with the older Europeans, and experience leads me to add that whilst true relapses may be overlooked, there is also a risk of regarding non-specific events in the light of veritable recurrences.

recurrences in this series, and if only the 4 proved events, the proportion becomes 18 per cent. From this summary it appears that the estimate of second Relapses, becomes greatly a matter of judgment: for if the restrictions adverted to above be adopted, the number of second recurrences could not be said to exceed 20 per cent. of all relapses; but if greater latitude be taken in the use of terms, that number may be reckoned considerably higher.

With regard to third Relapses the proportion of 5 per c. which was first mentioned by me may be excessive, since the experience of two later years in the epidemic shows third recurrences to have become rarer than this in hospital. At present, however, I do not think it possible to state their correct proportion, because so few patients could be persuaded to stay long enough for adequate observation; and it yet remains to apply strict microscopic blood-tests in these late events.

I formerly estimated fourth Relapses to happen as frequently as 2 p. c. of all attacks; the statement may remain, but there is still required a complete scrutiny of the later course of spirillum fever until the establishment of convalescence. At least, I was not able to carry out this at Bombay with the aid of riper experience, prior to being compelled by sickness to leave. Judging from personal experience, I think that in some instances there would be found a gradually diminishing series of more or less periodic febrile paroxysms, devoid of visible blood-contamination, as at present detectible; whilst in other cases, all evidence of residual phenomena might be wanting.

DETAILS OF PYREXIA.—Both 3-hour readings and the ordinary M. and E. entries, prove that within varying degrees the spirillar pyrexia conforms to the normal daily cycle; and the m. temp. exceeds that of same e. only when a nocturnal paroxysm lasts somewhat longer than usual, or when a prolonged depression takes place: the m. t. of one day exceeds that of the previous e. when a prolonged rise occurs. Charts may be further considered as regards the contour lines of daily *maxima* (which indicate the intensity of fever) and of *minima* (showing its sustained character); the interval between these lines being the extreme *range*, which is also of clinical interest. I have been accustomed to employ the terms level, convex, or depressed, ascending or descending with reference to such lines, for expressing the general course of pyrexia.

Mean temperatures have only a limited value, and seldom correspond to the average or more frequent course; but as 'means' are in common use, they are added here.

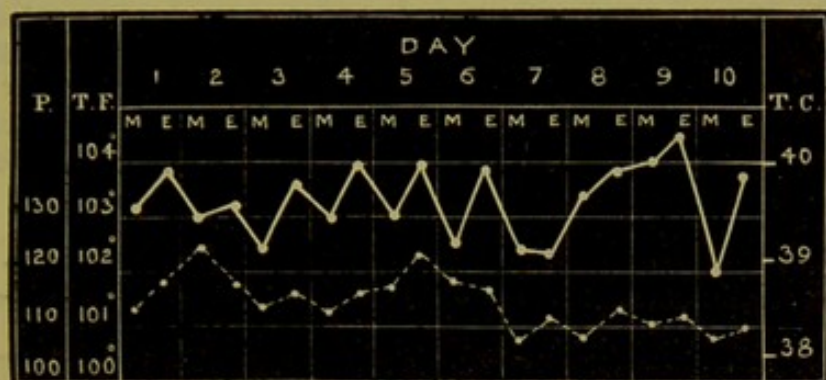
Pulse reckonings are also given.

Pyrexial Characters of Invasion.

Fever begins abruptly: in 33 cases mean t. of m. onset $103^{\circ}\cdot2$, of e. onset $103^{\circ}\cdot8$. A sudden e. rise of 6°F. has been noted, and probably is common: it may occur in two or three hours. The pulse was 113 and 119; it varies considerably and at first may gradually quicken.

TABLE IV.—MEAN T. AND P. ON SUCCESSIVE DAYS THROUGHOUT.

Day	1		2		3		4		5		6		7		8		9		10	
	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.
M.	103.7	113	103	124	102.4	113	103	113	103	118	102.5	118	102.4	108	103.4	109	104	111	102	109
E.	103.8	119	103.2	119	103.6	116	104	117	104	123	103.8	117	102.3	111	103.9	111	104.5	112	103.7	110



Starting at $+103^{\circ}$ pyrexia declines on the second day or till third morning, when a prolonged rise begins extending to seventh day, or usual date of crisis: in prolonged cases, another brief elevation takes place. Individual charts show the contour of maxima to be tolerably level; whilst that of minima varying more descends, the daily range rather increasing to the last. The pulse, in general, follows the temperature; but quickens more gradually at first, and towards the end does not so freely rise.

The general elevation of t. varies in different cases throughout the attack: in children and women it was seldom very high, nor was it considerable in cases of an asthenic or typhus character, which were seen amongst weavers. The pulse, on the contrary, is apt to be quicker in all three instances. In robust male individuals, there might be a high temp. with a pulse hardly so quick as in the mean.

Extreme Temperatures and Range.—In the typical series under analysis, the maximum t. noted was $105^{\circ}8$ on the sixth evening, and $105^{\circ}4$ on the following morning; and a somewhat similar connection was noted on the fourth and fifth days: these tendencies possibly indicating natural periods in the course of the attack, and fresh accretions, more clearly than in some other specific fevers. At such times, the normal daily cycle becomes temporarily obliterated. In exceptional moments such as the critical perturbation, the t. may rise above 107° , and doubtless the actual maxima were often overlooked. In monkeys, however, the t. does not rise higher, though starting from a more elevated normal level.

Minimum t.—The tendency of the spirillum fever in man to decline on the 7th day was shown by the low minima of $99^{\circ}8$ both m. and e. occasionally noted: these figures have been retained as being above normal, but it is proper to remark that the crisis followed on. In another table, the data pertaining to known periods of the invasion will be furnished; here allusion is made to the mean t. found upon any days

(excluding the fall) in attacks of all durations. A similar but less marked tendency to droop was observed on the third day, and again at the end of attacks prolonged to the tenth day.

Pulse.—The quickest mean p. was found on the second morning (124) and fifth evening (123): the slowest on the seventh day m. and e. (108 and 111).

Ranges.—The greatest mean daily range of t. is ordinarily not quite 2° F.; of p. about 14 per minute: the daily range is greatest at beginning and towards end, and least about midway. The febrile state whilst it lasts is well sustained, and in this respect resembles the fever symptomatic of acute inflammatory lesion, or typhus, rather than that of remittents, hectic or enteric fever. The following figures were derived from four mild cases of moderate duration.

TABLE V.—MEAN T. AND RANGE THROUGHOUT INVASION.

Day	1	2	3	4	5	6	7
M.	—	102·5	102·7	102·4	102·	102·5	96·2
E.	103·2	104·	103·4	103·2	103·7	103·5	—
Range	4·7	1·5	·7	·8	1·7	1·	7·3

The range between extremes of temperature was greatest on the seventh day, both m. and e. (+5° F.); or more generally from sixth to eighth days inclusive; and least, at first and last (+2°): on the fourth day this range was less (2°·6) than on either third or fifth: the m. t. had a rather wider range than the e. t., except on first and eighth days.

Extreme variations of pulse.—These could be adequately illustrated through the means only of individual cases; but in the larger series it was noticeable that the maximum frequency of pulse both morning and evening was entered on the second day (144 m. and 134 e.) and again on the sixth (136 m. and 142 e.), whilst the common minimum occurred on the fourth day (99 and 90). The morning p., particularly, tends to decline at the close of prolonged attacks: at initiation, the pulse range was 96–120 for morning initiation, and 104–124 for evening onset.

The above memoranda being based on bi-daily observations, I wish it were possible to control and expand them by means of 3-hourly readings; but there is not with me a complete series of the minuter records for man. Some details are, however, given under the heading of 1st Relapse, and these may not differ fundamentally from those of the invasion.

Having now discussed the data in their collective form, it is desirable to allude to them in connection with main epochs of the invasion; and as regards the single or recurring forms of the fever.

The mean initial t. of 5 ordinary cases of abortive form were 103°·7 (m.) and 104° (e.): of 5 cases of the relapsing form 102°·6 and 103°·5; the pulse in both series being nearly the same, viz. 110–117 (m.) and 120 (e.). Notwithstanding the higher mean t. at initiation of the abortive series, there was not enough uniformity in particular instances, to warrant a prognosis of character and duration of the illness being inferred from this datum.

Later course and termination.—In another set of 24 ordinary cases not attended with complications, of which 14 were relapsing and 10 non-relapsing, the mean t. and p. of the invasion-attack, so far as seen, were as follows :

TABLE VI.—ABORTIVE SERIES OF VARYING DURATION.

Fall			Days prior to Fall									
	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.
M.	96°	93	103°	120	103·2°	124	103°	110	102·8°	126	102·5°	106
E.	—	—	104	130	104	127	104	120	103·2°	109	104·2°	115

RELAPSING SERIES OF VARYING DURATION.

Fall			Days prior to Fall									
	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.
M.	96°	85	102·6°	112	103°	113	102·5°	116	100·6°	119	103°	104
E.	95·6	81	104	115	103·7°	118	104	120	104	121	104	123

When the m. fall was prolonged till e., the t. of the previous e. was 1° less in both series : the p. declined most where fall was prolonged.

It is probable that some of the indicated differences between the above two series, have a foundation in reality : *e.g.* that the non-recurring attack (which tends to be the briefer) has a somewhat higher and more sustained t., and a more prompt critical fall ; with the pulse slower to rise, but maintained at the end.

Contour of Invasion.—In upwards of one-third of 46 cases, the chart contour line is almost level, not seldom it somewhat descends, and less frequently, it is either convex or ascending ; a mid-descent is rare and a decidedly wavy contour is quite uncommon.

The type of fever was in two-thirds of all cases of a continued character, daily range of temperature not exceeding 1°–2°F. : in the remaining one-third it was distinctly remittent, *i.e.* the daily range was over 2°F. : the intermittent type in which the temperature at some time of the day descends to 99° was not seen in this series, and elsewhere it was extremely rare ; so much so, indeed, that I remember seeing only 2 or 3 instances during the entire epidemic, and these were complicated with dysentery.

Of these bi-daily series a perceptible *perturbatio critica* was absent in two-thirds, and in the remaining third, it seemed comparatively mild ; it is well known, however, that the final exacerbation may escape recognition in the absence of more frequent readings.

Contour lines of the pulse are before me, showing its tendency to follow the t. both throughout and in diurnal changes ; but the sequence is an interrupted one : thus, on the first day the pulse does not rise so promptly as the t., and on the second day it still rises for a time (then attaining its maximum), soon however to follow the t., which has fallen somewhat : on the third, p. and t. may be said to correspond, next day

the t. rises and the p. also, with a promptitude which carries it to its second maximum on e. of fifth day ; thence it descends rather slowly to about the level it had on the first morning. Now there occurs either the crisis or a further considerable rise of t. prolonging the attack to the tenth or eleventh day, but the pulse does not ascend proportionately after the close of a week's fever.

As regards the differences between invasions which are followed by a relapse and those which stand alone, what was elicited is as follows :—the abortive attack tends to be highest at first (with no final rise) ; seldomer it is level and rarely convex in contour ; only exceptionally do distinct remissions occur, but a disposition to mid-descent may appear ; the *perturbatio critica* was more or less indicated in nearly half of 42 cases. The primary event which was followed by a recurrence, differs somewhat in oftener presenting a more level summit ; the tendency to decline midway is frequently noted, and the final exacerbation may be more marked ; the type of fever is more apt to be distinctly remitting and, on the whole, there is a wider variety in its contour : the intermittent type was extremely rare (38 cases analysed).

Such is the summary of repeated attempts to elicit a normal or fundamental form of the spirillum fever, which *à priori* might be thought to exist in so specific an affection ; yet I cannot say that the results have been decisive : it is true there are manifest variations in the abundance of the attendant blood-parasite, yet here again I have failed to connect the phenomena which seemed probable. Enough concordance appears, however, to permit of an accurate idea being gained of the clinical course of the first and chief febrile event ; and this I did not myself possess, until the present analysis was undertaken.

Pulse.—In the two chief forms of invading spirillar attack, the following differences are noticeable :—abortive form ; fever descending, remittent, the p. rises promptly to its first maximum, then falling to its minimum on the third morning, it ascends and maintains a moderate level, till finally rising at the close to reach its maximum on the fifth or sixth evening, in the shorter attacks. Recurring form : the pulse does not rise and fall so promptly at first ; its mid-course is tolerably uniform ; at the end it quickly rises and more slowly descends, not again recovering, even in prolonged attacks. The maxima and minima of p. and t. nearly correspond in date to the 1st and 5th evening, and the 3rd, and 6th, and 7th morning, respectively: this statement being of general application.

Ending of the Invasion.—With rare exceptions this occurs by way of crisis, and the fall of temperature is not only abrupt but excessive ; much surpassing the initial rise (itself equally prompt), and not being strictly accordant with the previous elevation and duration of fever. The pulse hardly participates in this disproportionate decline of body-heat. As the crisis offers a uniformity of character equal to that of the febrile state, it may be regarded as an essential feature of the entire specific manifestation.

Perturbatio Critica.—There are many degrees of pyrexial disturbance to be seen on the last day of fever, and the most prominent of these assume the form of a distinct febrile exacerbation, preceded by chills, pronounced in intensity, brief in duration, and directly initiating the

critical fall with copious sweats. Although there is reason to regard this final acme as being common, if not to some extent invariable; yet in the ordinary clinical charts, its occurrence seems unusual. Thus, a distinct critical exacerbation was not seen in more than 10 p.c. of all invasions; and as regards the less pronounced febrile disturbances all that can be said is, that in somewhat less than half the cases the temperature on the last evening rose from $\frac{1}{2}^{\circ}$ to 1° , seldom more, with no particular symptoms and but little exaggeration of the febrile stress. Such exaggerations of the normal e. rise when final are not necessarily followed by a fall descending below the critical mean (96°), nor after a well-marked *perturbatio critica* does the t. inordinately subside, in general; on the contrary in six instances the mean t. was $96^{\circ}3$; maximum $97^{\circ}4$, minimum $95^{\circ}4$. It may, however, happen that after a prominent final exacerbation the fall is very considerable, and these are the instances which furnish the most striking figures in illustration of the range at crisis.

An example of the progress of pyrexia is the following:—

CASE XXVI.—An adult ward-servant, in previous good health, contracted relapsing fever in hospital, and came under special clinical observation. The invasion was pronounced and attended with the usual symptoms; on the sixth and last day, the t. at 7 A.M. was $102^{\circ}5$; p. 120, at noon it had risen to 104° , at two P.M. 105° , at four P.M. $105^{\circ}5$; p. 130; at five P.M. the t. was $107^{\circ}2$, pulse 134, and at six P.M. the acme of $107^{\circ}5$ (p. 132) was attained: at seven P.M. the t. had sunk to $103^{\circ}2$ and there was copious sweating, at half-past seven the t. was $101^{\circ}4$, at eight the same, at nine $101^{\circ}2$, at eleven $100^{\circ}8$ and at midnight $100^{\circ}4$; at six A.M. of the following day the t. was 99° ; a slight rebound followed, prior to min. t. of $97^{\circ}8$, p. 80. J. J. Hospital.

In my own late brief single attack the fever distinctly culminated during the final night of 5–6 day, the temp. after constant cold sponging not going below $105^{\circ}2$, p. 120: it declined at early m., being 103° (p. 114) at 7 A.M. but rising shortly to $104^{\circ}4$ (p. 118), thenceforward declined during the day. The pulse, as usual, followed the temp. but somewhat in arrear; quickening to 120 at 11.30 A.M. when the body-heat had already begun to decline. For comparison with critical phenomena at first relapse, see CASE IX., Chapter III.

Crisis. Date.—In a series of 60 cases happening at all periods of the epidemic, crisis occurred on the 7th day twelve times, 8th day eleven times, 9th day nine times, 6th day eight times, on the 10th and 11th days six times each, 4th day four times, on the 5th day thrice and on the 12th day once. As much of this evidence regarding dates was hearsay, and patients probably rather overstated the duration of their illness, I will here mention separately the day of crisis as witnessed in cases of disease acquired in hospital: amongst sick patients it was in the mean 6 days (extremes 4 and 7), amongst students 7.5 days (extremes 6 and 8) and amongst servants 7.6 days (extremes 6 and 9): in the four last instances of contagion at the G. T. Hospital the day was the 7th thrice, and probably the 8th once. The predominance, therefore, of the seventh day as that upon which the crisis, chiefly or wholly, has been seen, is sufficiently apparent; next in frequency follows the eighth, sixth or ninth: I have never witnessed throughout any attack lasting ten days or more, but have the full records of a few of only four or five days'

duration, so that it is certain the crisis may supervene at periods varying from four to nine days.

As regards period of day at which the complete fall was noted, of 79 charts 53 showed the morning, and 26 the evening; or in the proportion of $\frac{2}{3}$ and $\frac{1}{3}$ respectively: whence appears a tendency of the spirillum fever to subside between the hours of 4 P.M. and 7 A.M. The specific character of spirillum fever is, however, indicated by the fact of about $\frac{1}{2}$ of crises occurring mainly or wholly during the daytime, when the normal temp. habitually rises and reaches its maximum; this proportion, indeed, becoming larger if the 24 hours be physiologically divided into periods of normal elevation and depression.

Rate of critical decline of temperature at Invasion.—Bi-daily readings show that, as a rule, subsidence of fever was completed in 1 day, and commonly within 12 hours; estimates depending partly upon the method of reckoning, when, as often happened, the fall was not strictly continuous. The main decline may not, however, occupy more than 3-4 hours, or occasionally less; e.g. in cases specially observed there has been seen a fall of $6^{\circ}\cdot8$ in 3 hours, $5^{\circ}\cdot4$ in 2 hours, and $4^{\circ}\cdot3$ in 1 hour: the slower descent before or after the main one, is a singular phenomenon which I failed to comprehend; and analogous to this is the persistent depression sometimes seen at end of crisis. The mean rate throughout in eight detailed cases was about 8° in 11 hours, the observed extremes being $4^{\circ}\cdot3$ and 2° per hour: commonly the longest fall occupied most time, e.g. 12 hours for 9° - 10° ; and the shorter a less time, e.g. 7 hours for 7° . In 3 of these cases the decline was most rapid at its beginning, in 5 it was quickest at middle or towards the end; from my own case it seems that with free sweating the cooling may be hastened, but on other occasions this connection was not stated or implied. It is noteworthy that the rate of main fall was decidedly quicker in day crises than in night crises, the respective means being $6^{\circ}\cdot4$ in 4 hours and $7^{\circ}\cdot8$ in 9 hours; which is a datum hardly to be anticipated, on the supposition that the decline of febrile temp. would naturally be hastened when concurrent with normal decline of body-heat. As regards the final lingering of critical subsidence, I noted in 32 early cases that about one-fourth of the preponderance of m. minima was due to prolongation of the fall in 7th m. until m. of 8th day; the occurrence being almost limited to these particular dates, which were also the commonest entered. Much variety obtains at the lowest turning-point of crisis, rallying being prompt, delayed or intermitting (as it were) in very various degrees.

Form.—As represented in ordinary charts the crisis assumes one of three forms, namely either an uninterrupted line of descent, which is the most frequent; or a descent rapid at first and towards the close slower or delineated as a sloping line, which is not unusual; and, lastly, as a line oblique at first and finally vertical. The last form is so rarely expressed that it might be regarded as hypothetical, yet an approach to its character was noticeable in the cases where for the last two or three days of the febrile state the t. had perceptibly declined from its acme, to fall suddenly to an extent diminished by so much as it had already been gradually reduced. Occasionally, too, the t. has been low and stationary on the last day of fever, not rising as usual at evening. I

mention these instances as exceptional, and therefore of interest as indicating what features are essential to the fever, and what may be modified without its characteristics being annulled; already it will be apparent that in such cases as those last mentioned or even the earlier ones, should the decline be further interrupted by subsidiary 'daily febrile paroxysms, it will come to gradually assume the character of 'lysis.'

Degree of critical fall.—The minimal points actually recorded must be regarded as only approximate. As the figures stand, I find that at the height of the epidemic there were amongst 18 cases of Hindus, 4 in which 94° – 95° (actual minimum $94^{\circ}5$) 6 in which 95° – 96° , 6 in which 96° – 98° and 2 cases in which temp. of $98^{\circ}+$ have severally been entered as the lowest points of the fall: in a later series of 18 cases amongst Mussulman weavers, the same point was only once entered so low as $95^{\circ}6$, and only twice was as low as 96° – 97° , the remaining instances showing usually about 97° , thrice 98° and thrice 99° , as the minimum, all in uncomplicated instances. Such less degree of fall accompanied less pronounced high temperatures, and both features seemed to belong to the modified (possibly typhus) type of fever which prevailed most amongst the weavers.

The following table pertains to the same series of cases as the tabular statement of mean t. and p. during pyrexia, which was quoted above.

TABLE VII.—MEAN T. AND P. ON SUCCESSIVE CRITICAL DAYS, WITH NUMBER OF M. AND E. MINIMA.

	4		5		6		7 completed				8		9		10		11	
	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.
M	97 ⁰ .1	97 ⁰ 96 ⁰ .4	92	96 ⁰ .1	80	96 ⁰ .7	87	96 ⁰ .8	79	96 ⁰ .5	80	—	—	—	—	96 ⁰ .3	77	
E	97.9	96.95.6	—	97.2	90	97.6	91	—	—	94.5	—	97.3	—	97.5	80	96.8	82	
M	2	2	3	2	4	6	0	0	2	2	0	2	2	1				
E	2	1	2	5	0	1	2	2	1									

In general with a prolonged decline the t. sank to 96° , or even lower; but the minimum falls were, even with an abrupt and direct decline, when the t. subsided within, at most, as many hours as there were degrees of subsidence; for example, 10, 11 or 12. Reckoning the departures from a normal level of 98° , it will be seen that while the ascent of t. at the initiation of attack may be estimated at 5° or $5^{\circ}5$, the descent at the crisis exceeds this figure by 2° or 3° ; which represent an amount of depression not met with in other continued fevers. The less degree of fall to 98° or 99° in cases otherwise typical, has, however, undoubtedly sometimes occurred; and I did not detect extreme critical depression in the sick monkey.

Respecting observed extreme ranges, the data are only approximate, but in such instances there appeared no reason to reject the estimated record of $94^{\circ}5$ or 94° ; or less: the following case occurred at the J. J. Hospital in Feb. 1878:—

CASE XXVII.—F., 40, Mussulman, immigrant, widow, not emaciated, was admitted at near close of invasion, with usual symptoms and considerable splenic enlargement: on 7th or last day of fever, e. t. 104° , p. 128: day of crisis, m. t. $96^{\circ}\cdot4$, p. 72, e. t. $93^{\circ}\cdot6$, p. 68, and next day of prolonged depression, m. t. 93° (estimate), p. 72, she was then delirious; e. t. $93^{\circ}\cdot4$, p. 72; the extreme depression and active delirium slowly passed away during the next three days, and in six more she was discharged convalescent. The mercury sank below the register.

Once the recorded descent of temperature amounted to 12° F.; a prompt decline of 10° , or even 11° , was not very uncommon; these deeper depressions depended partly upon the height of temp. at the previous acme, and the deepest were those in which there happened a prominent *perturbatio critica* before an excessive critical fall; such conjunction was comparatively rare. Supposing, however, a concurrence in the same individual of the two extremes known to me (viz. of $108^{\circ}\cdot4$ and 93°), a descent of 15° F. would have been indicated.

Pulse at crisis.—Here the chief notable feature is that the rapidity of the pulse does not decline in amount corresponding with the fall of temperature: this fact was invariable, and it is well worthy of attention. Already in the course of invasion seizures it was observed that the pulse did not rise with the pyrexia towards its end; and this want of concordance, indicating a lagging behind of the circulation, became still more apparent at the crisis. The mean p. at fall was shown in Table VII., from which it appears that at crises occurring on the 7th day and prolonged to the 8th m., the pulse sank to 79 beats per minute with a mean t. of $96^{\circ}\cdot8$; in the earlier crises it might be quicker, in the later of equal velocity or possibly lower with temperatures higher and lower respectively. In the main, the pulse follows the temperature and hardly shows a greater tendency to variation, when individual cases are separately considered: idiosyncratic influences, however, remain, and at the crisis, as previously, in children, women and cases showing a low type of fever, the pulse often continues even high. Experience at Bombay seems to have been somewhat unusual, as regards the tendency of the pulse to remain frequent at crisis; nor were the excessive slowings sometimes recorded in Europe so likely to be met with there.¹ The minimum p. in the collected series, was 60 in a case (abortive fever) where the circulation was unusually slow throughout the entire attack; the maximum p. noted in the crisis was 112 (quick throughout) in an abortive attack; and 120 in one of the relapsing form, characterised in other periods by an unusually quick pulse.

In crisis prolonged from morning till evening, the pulse continues to sink with the t. in most instances, and may become so slow as 70 per m.: sometimes it happens (one-fifth of cases) that it does not subside after the main fall, and, as a rare event, it has been found to rise a few beats after the main decline; although in both these instances the t. still further subsided to a slight extent. As a rule, with moderate fall of temp., there concurs a moderate declension of pulse; and with pronounced fall (e.g. 10° or 11°) the pulse becomes much slower (e.g. by 60 or 70 beats per minute).

¹ Dr. Fräntzel at Berlin, 1868-9, stated that the pulse sinks to less than 60 per minute, as a rule; is often less than 50, and sometimes only 40: it becomes 40 or 60 beats slower than before, and once was noted to decline 80 beats at crisis. Virch. Archiv. vol. xlix. 1870.

The pyrexial phenomena of crisis were almost identical in first attacks which were abortive, and in those which relapsed ; being only somewhat less prompt in the recurrent form.

Lysis.—Towards the close of a primary attack of spirillum fever, the pyrexia occasionally assumes a distinctly remittent character, in which the morning t. is as l w as 101° or 100° whilst the e. t. rises to 102° or 103° or more, till the crisis arrives, when the attack ends for good : sometimes the t. on the last evening was no more than 101° . Another set of cases seen in about one-tenth of all, was that in which after the beginning of the crisis, the further descent of temperature was effected gradually, or by a series of short daily paroxysms, exacerbating at evening and diminishing in intensity, until the normal level was reached in the course of two or three days.

In both these modified forms of crisis the final minimum of t. was generally not lower than 97° or $96^{\circ}5$, the pulse being quick as often as slow. In a last series, much more rare than the others, but merging at points identical in all, this critical termination became altogether undistinguishable, and true lysis was established.

This I have witnessed in 9 or 10 instances only, and almost always in invasion-attacks. Then the last 2, 3 or 4 days of continuous high pyrexia were replaced by a corresponding series of daily paroxysms, commonly of increasingly remittent character ; which commencing gradually or abruptly and oftenest at morning, promptly became intermittent, and with declining evening exacerbation then subsided to the normal level, or to 1° or 2° below it. This lytical termination varies in duration and intensity : when watched throughout, the mean duration of the invasion-attack was not apparently prolonged, only its last three days being thus modified, and there was a slight tendency to rebound at the end. A relapse was the rule, and it, too, modified at the close. The mean t. and p. in 6 early instances are shown in the following table : others seen afterwards fairly concord, and it is worth noting that of the 7 patients whose cases are specially considered, 5 were females :

TABLE VIII.—MEAN TEMPERATURE AND PULSE IN LYSIS.

Day	1		2		3		4		5		6		7	
	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.
M	$102^{\circ}3$	105	$100^{\circ}9$	103	$99^{\circ}4$	100	$98^{\circ}9$	96	$97^{\circ}9$	92	$96^{\circ}7$	96	$96^{\circ}6$	108
E	$102^{\circ}7$	125	101°	104	$98^{\circ}6$	97	$97^{\circ}6$	100	97°	72	97°	100	—	—

The minimum of t. and p. was reached on the 4th day of lysis (about the 7th day of the attack) three times, and once on the 5th, 6th and 7th days of descent, when it would seem that the fever was prolonged. The variations of pulse here noted at the close were due to particular cases, and those of t. were partly so ; it being noteworthy that the morning t. and even. p. were highest on the three earlier days of lysis, corresponding to the terminal ones of the ordinary invasion seizure.

When the slow fall lasted for 5, 6 or 7 days and a relapse followed, it appeared that the first apyretic interval was encroached upon : in one instance the entire attack extended over 14 days, and there was no recurrence.

I will add that though it might be supposed subsidence of fever by this mode indicates mildness of the attack, yet such is not commonly the case ; and all the above six cases were severer than usual in exhibiting symptoms approaching those of continued, *i.e.* typhus-like fever.

Lysis at close of a first relapse was very rarely seen : a few well-marked instances, however, came under notice, and these were attended with much suffering and prostration. See Chapter III.

Duration of the Invasion-attack.—This has been reckoned from the first onset of fever until the main fall at crisis inclusive ; and the data are derived from 341 earlier cases seen at the hospitals. In by far the majority of instances the statements of patients was the only authority for dates, and often, at least, might be depended upon, for the onset of fever is generally so definite as to be unmistakeable ; error, however, might occur in two directions, namely by ante-dating the attack when premonitory symptoms were included, or by post-dating the real onset by reckoning from the day when the patient had to lay up. Such opposed calculations would be mutually antagonistic, and that the approximately true duration of the attack has been elicited, seems to be shown by the remarkable concordance of all the figures, when duly checked and compared. For this purpose, the contagion series embracing the cases of students, servants and patients infected whilst under observation, was available at both hospitals ; and the result has been an apparent uniformity of duration in the initial spirillum fever of man not to be met with in other specific pyrexias, with the exception of the vaccinal, which, like the spirillar, is due to a contagium capable of being transferred at will from one individual directly to another. The limits of variation in the human events are, however, much narrower than those observed in the artificially induced pyrexia amongst quadrumana. Mean duration 7.5 days, average 7 days, range 4 to 13 days : with respect to the last item I should observe that the shorter period was seen, but the longer has not been actually witnessed ; the actual number in each extreme was only two or three. It is also worthy of note that in 13 of 26 sick persons attacked in hospital furnishing accurate data, the duration of attack was, in the mean, at least a day shorter than in all other instances, with an average of 5.5 and a range of 4 to 7 days : whence it might be inferred that a weakened or diseased state of the body interferes with the full development of the pyrexial agent, and that a perfectly healthy condition would be favourable thereto. The clinical interest here rests upon the assumption that duration is a measure of intensity ; and this last may be also connected with the activity of contagium, as well as with the state of the body its recipient. The term day here stands for a period of 24 hours.

Marking early the prominent distinction between febrile seizures which occur only once, and those which are repeated ; I have sought to ascertain whether or not their respective duration differs, and find there is practically no distinction to be made here. On comparing closely two selected series of cases, the only difference I note is a tendency of

the relapsing invasion to last somewhat the longer ; and it was amongst the Mussulman weavers that this tendency prevailed most, or wholly ; the Hindoo famine-immigrant not showing it.

First Apyretic period.—Except when a rebound immediately follows the critical fall, the temperature is low for two or three days, according to the promptness of general reaction : and it is rare to find the normal t. at once and permanently established. The pulse at first retains the slight preternormal frequency it had at the fall. Subsequently there is a gradual ascent of the t., most marked at first, until about the 5th or 6th day, when the normal level of $98^{\circ}5$ is attained, and thence a level course is preserved, until the day of relapse. Amongst instances of deferred relapse there may often be detected a perturbation of t. on the 7th day, as indicated by slight rise at evening, or a morning decline, with possibly some headache or other slight symptom ; and not seldom a day or two before the relapse even when not deferred, there occurs a mild depression of t. which I have known to correspond with the initiatory appearance of the blood-parasite : such perturbations do not exceed in range 1° or 2° , or the limits of normal variation, and are liable to be overlooked. This is also the case with a similar phenomenon, usually rather more distinct, which takes place about the same date as the so-called abortive attacks ; and in them may indicate a suppressed tendency to relapse. The ordinary charts may also show other minor degrees of pyrexial agitation, amounting in the end to a recognisable second outbreak, consisting of brief daily paroxysms, single or repeated and blended : so that whilst in general it may be said the relapse is either present or absent, yet in particular, there are milder degrees of recurrence whose import will be differently estimated.

The Pulse.—Whilst the t. gradually ascends during this first interval, the pulse is found to decline in frequency, and their respective contours, as displayed in the chart, commonly cross on the 2nd or 3rd day, that of the pulse continuing to decline in a more or less gradual manner until the 5th, 6th or 7th day ; and then promptly rising to its normal velocity just before setting in of the relapse. It concurs with the t. in sub-latent pyrexial perturbations, and may by its own changes of either increased or diminished frequency, more clearly or alone indicate their occurrence. As these slighter disturbances do not always correspond exactly in date, they do not necessarily appear in outlines of means, and are best studied in individual instances. Non-periodic changes of p. or t. have a different import, and with due care might generally be discriminated. In consequence of its peculiar course, the m. pulse bears a high relation to that of the evening, and for a time is quicker on the same day. The sinking of the pulse after specific fever may continue until the day of relapse, and in abortive attacks it may persist for many days, its actual degree being sometimes striking. Similar decline occurs after malarious fever, but is not so pronounced.

TABLE IX.—MEAN TEMPERATURE AND PULSE DURING FIRST INTERVAL.
17 CASES.

Day	1		2		3		4		5		6		7		8		9		10	
	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.	T.	P.
M.	96°·9	80	97°·5	78	97°·4	76	97°·4	73	98°·1	73	98°·0	72	97°·9	72	98°·2	71	98°·2	70	98°·2	75
E.	97°·4	84	97°·6	80	97°·9	77	98°·1	75	98°·1	76	98°·3	73	98°·3	73	98°·7	76	98°·2	78	98°·6	82



The temperature is lowest on first day after crisis : the normal daily course is maintained in that the e. t. is highest, with rare exceptions : always the temperature rises towards the end by about 1° ; its mean level is below the normal, showing the generally depressed condition which follows the pyrexial attack, and sometimes in the weak and old this was strongly indicated.

The pulse is highest at first, and that of the m. may be quickest, its mean range is about fifteen beats ; its general level probably over rather than under the normal.

This apyretic period is liable, at its commencement, to a smart febrile rebound following the crisis, which seems to me mainly functional : it is also liable to interruption from many complications, usually arising soon after the crisis, and only occasionally does it offer the strictly neutral features of simple convalescence. There are some differences as regards the two forms of invasion, but these were neither considerable nor uniform ; and the great similarity of phenomena in non-relapsing and relapsing attacks, appears to me well worthy of notice.

Amongst Mussulmans was found a subdued type of the spirillum fever (the blood-parasite meanwhile abounding), marked by shortened crisis but persistent depression, and the mean level of t. in place of steadily ascending during the apyretic interval, rose only at first ($98^{\circ}\cdot4$) and from the second day to the last slowly declined till the day of relapse (97°). The pulse also while quicker at first (88) than in other series, did not descend, as in them, so as in the chart to cross the line of t. ; but following the t. very gradually declined till the last day of interval (72). It might be said that in these less robust subjects, there was defective reaction after the first febrile attack, combined with persistent irritability of the circulation.

Duration.—The first interval was computed to last from the day after the crisis to the day before the relapse, both inclusive ; and it was

found in 190 continuous instances to have a mean duration of 7·4 days, an average duration of 8·1, and an extreme range of 3 to 12 days. The instances of extremest range were very few, especially those of the shorter period. It is remarkable how nearly alike is the duration of the invading attack, and that of the first apyretic interval—viz. as 7·5 to 7·4 days: still the two events are probably not connected, for a relapse is by no means invariable, and analogy points to the association of each non-febrile stage with the succeeding pyrexial attack. Apparently there is no fixed relation between the duration of interval, and that of preceding or following febrile event; or between it and the intensity of fever in these attacks. Nor has experiment on the quadrumana elucidated the conditions of varying incubation-period and febrile manifestation: probably they are not simple, but complex. The close of this first interval is attended with a visible blood infection and both t. and p. may indicate some concurrent perturbation; as a rule, however, there is little that is certain recorded, and more elaborate research is still required; my experiments on the temperature observed at even 3-hour intervals not being quite conclusive.

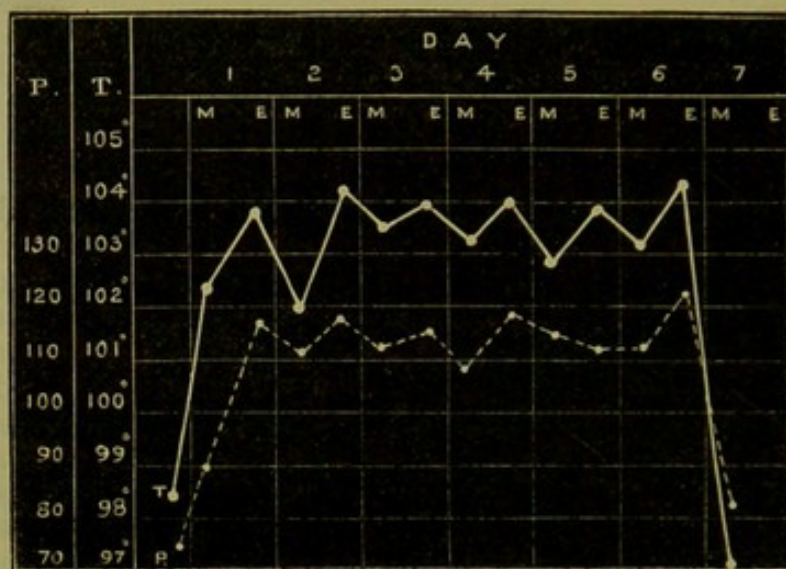
Second Febrile seizure; first Recurrence or Relapse.—Usually this is a prominent feature in the patient's illness, being almost tantamount to a reproduction of the invasion seizure terminated a week before: less often it is comparatively insignificant, and between these two extremes there occurs almost every variety of form, intensity and duration of pyrexia. Even when brief, the fever commonly presents marked diurnal exacerbations; and this paroxysmal character tends to prevail in relapses of 3–4 days duration; in pyrexia of 5–6 days it is still apparent at first, the subsequent tendency being towards more continuous type.

In consequence of so great liability to variation, it becomes difficult to convey briefly an adequate conception of the relapse: 30 cases have been selected for analysis, all in survivors and free from complication, of 4–7 days' duration, and dating throughout the epidemic. The whole group will first be considered; then each series of most to fewest days' length; a few examples of short relapses being added to complete the description: remarks are based upon bi-daily records.

Initiation.—Some hourly observations made at the beginning of the relapse shown in Chart 2, Plate IV., prove that when chills occur at onset of fever, they do not supervene until the temperature has already risen:—7 A.M.—t. $100^{\circ}\cdot4$, p. 86, no chills or headache, bilious vomiting with uneasiness over site of gall bladder: 8—t. $100^{\circ}\cdot4$, p. 90, no chills or other sign of uneasiness: 9—t. 101° , p. 100, no chills, some aching in joints, no splenic uneasiness: 10—t. 102° , p. 108, no chills: 11—t. 102° , p. 110, slight chilliness, no headache, hepatic uneasiness: 12 noon—t. $103^{\circ}\cdot4$, p. 112, still sense of chills, no headache: 1 P.M.—t. $103^{\circ}\cdot6$, p. 126, still sense of chills, no headache: 4—t. $104^{\circ}\cdot2$, p. 150, chills ceased.

TABLE X.—MEAN T. AND P. OF FIRST RELAPSE: T. IN CONTINUOUS LINE, P. LINE DOTTED.

Day	1	2	3	4	5	6
T. { M. E.	102°·3 103°·8	102° 104°·2	103°·5 104°	103°·3 104°	102°·9 103°·8	103°·2 104°·3
T. range	1°·5	2°·2	°·5	°·7	1°	1°·1
P. { M. E.	99 117	111 117	112 115	109 118	114 112	113 122



Temperature.—The general mean temperature was m. 102°·9, e. 104°+, with daily range of 1°·16, or 5° to 1° F. above that of the invasion: range greatest on second day: general course indicative of a slight ascent from first to last, initiation and fall abrupt. The line of mean t. here shown is more or less closely conformed to by some examples of continued recurrent pyrexia; but it is of artificial construction, and the fever of spirillar relapse is not found to be generally continued in type, when charts are viewed in detail; for these individually display a highly diverse contour of mingled form.

The extreme temperatures were contemporary with the mean, being min. 99°·3 on 2nd morn., and max. 106° on last even. at acme: daily range greatest on 2-4 days, and least on first and last day; so that it is not so much at beginning and ending of the first relapse as during its early course, especially on second day, that great variations of t. were noted.

The daily course of the fever after initiation may be summarily described as follows: the second day is marked by a morning fall usually very decided, the evening rise which follows being equally pronounced; and these events may prove the minimum and maximum of the entire attack. The decline may be deferred until the third morning, and may

be so considerable as to amount to an intermission ; the succeeding rise is the commencement of a second paroxysm and is almost universal.

Third day : similar morning remissions and evening exacerbations occur, but both less pronounced. When the initial paroxysm has been prolonged over the second day, the marked fall and rise of that day are transferred to the third ; and in cases where the attack ends on the fourth day, the final, and it may be the highest, rise occurs on this evening.

Fourth day : the majority of instances display a rise and fall more limited and approximated, so that the tendency is for the pyrexia to increase and become more equable. Modifications result thus—in the prolonged and, as it were, slower morning paroxysms of long attacks, the m. t. may come to represent the second rise usually happening on a previous evening, and as it then exceeds the t. of the same e. there ensues the seeming anomaly of a higher morning t. : this event, though rare, shows the independence of the spirillar paroxysms. In case of an attack terminating next day, the evening t. may rise beyond the mean on this day.

Fifth day : instances are now fewer : m. fall decided, ranging about 103° ; evening rise considerable (over 104°) in the cases when the attack is to terminate next day ; more moderate in the slower rises of prolonged attacks, or as the low summit of an interrupted and deferred later fall.

Sixth day : temperature either m. or e. forming the acme of a final rise (usually not considerable) or the main fall may be in progress this day : range 103° – 104° or lower.

Seventh day : may display an interruption in the fall of an attack prolonged until now, or a superadded and semi-detached paroxysm similar to a first in the series composing the attack.

Contours of first relapses.—Of 100 fully developed relapses the upper contour was ascending 22, convex 22, level 16, declining 16, cleft (bifid or trifid, with parting sometimes equal to an intermission) in about 14, a wavy contour or slight decline 6, and the remainder a single upheaval, as it were, of temperature. The lower or minimum contour-line was less regular ; in prolonged seizures the remissions tending to shorten at last, and in the briefer to become more pronounced.

Form of fever.—No absolute uniformity was observed : thus, analysis of 69 representative cases gave the following proportions—continued form 26 p. c. ; the remittent 70 p. c., and intermittent (excluding single paroxysms) 4 p. c. : predominance of the remittent type, as commonly estimated, is here indicated ; and the occasional aspect of high fever perfectly sustained for two or three days, at one level, might be illusive and due to insufficiency of bi-daily observations. The continued form was oftenest declining or level in its contour, seldom ascending or dipping midway ; it was commonest in long relapses, and most frequently seen towards height of the epidemic : the remittent form prevailed throughout and was well marked in brief relapses ; it not uncommonly showed an ascending contour, the descending and level course being next in frequency : the intermittent form offered no predominant contour.

Construction of the longer relapses, first and subsequent.—There was often perceptible a division of the whole event, by deeper remis-

sions, into 2, 3 or 4 main parts (secondary constituents or specific paroxysms) more or less alike, and variously composed of 1 or more daily exacerbations (primary constituents or fundamental paroxysms): and it seemed to me that all the febrile phenomena following spirillar infection, might be accurately as well as conveniently regarded as being constructed of 1, 2 or more of such specific paroxysms. Such hypothesis also assists in comprehension of the disintegrating process noticeable in different degrees at successive relapses, and exceptionally at invasion.

The pulse in first relapses.—The pulse concurs with the temperature in its general course, but is apt to rise more slowly, and finally not to attain a frequency proportionate to the higher temperature of the relapse: hence the mean p. rate is not so rapid in the second attack as in the first, although the t. is 1° greater. I also find that the circulation sometimes continues to be excited subsequent to the acme and beginning of crisis; after the fall is established, the pulse declines rather more than at the invasion crisis, yet not descending to its minimum until some time after the lowest t. has been reached, and febrile reaction has made some progress.

Whilst the perturbations of temperature are greater in the relapse than in the invasion, the pulse in following these changes is found not always to accord with their direction; and not seldom the morning p. was in excess of that of the same evening. This divergence was most frequent on the 3rd day of the relapse, but was also seen on the 2nd, 5th and 4th, the evening t. on all these days being in excess of the m. t.; one-third of all cases showed this peculiarity on the 3rd day, one-sixth on the other days; and the explanation seems to be found in previous febrile excitement at night followed by depression of temperature, but not with so prompt subsiding of the pulse. In the two main varieties of relapse seen amongst Hindoo immigrants and Mussulman weavers, showing respectively a briefer, sharper and ascending course of the pyrexia and a lower, level and more prolonged course, the p. was found to correspond pretty closely; thus, in the first series, it began low (77) and rose in frequency to the end (110), and in the second, beginning high (110) it remained so, being most rapid of all (124) in this low typed and prolonged form of pyrexia. Upon wide review, the variations of pulse were found to be at least equal to those of temperature; and instances of non-concurrence were somewhat more frequent than during invasion.

Initiation of the relapse.—Whilst both briefest and longest relapses have a tendency to begin in the morning, those under notice of mean duration commence, like the invasion-attack, oftenest in the afternoon, in the proportion $\frac{3}{8}$ to $\frac{2}{8}$: about 60 p. c. of all first relapses commenced during daytime. The morning rise in accordance with normal movements of temperature was sometimes clearly made out, but generally it had commenced at some hour not known during the previous night, when the body-heat was declining: the exact time being rarely indicated by chills. At the hour of 7 A.M. the mean t. was 102° or a little over, which is 1° lower than that of m. initiation of the invasion: subsequently the t. continued to rise throughout the day, and in the e. it was 104° . Some 3-hour readings made during one of these short recurrences, showed a distinct check in the day-rise, taking place before noon, and

indicating the occasional influence of normal cycle in even one-day relapses.

The initial evening rise (rather the commoner) began usually after noon when the normal t. was proceeding to its maximum, and it continued to rise till beyond this point in a few instances specially observed or until 8 to 11 P.M. As noted at 4 P.M. the mean t. of evening rise was about 103° , or about 1° above that of m. initiation; but 1° less than the e. t. following, and also nearly 1° less than the evening initiation of the invasion-attack. As compared with onset of the invasion-attack there is here observed both lower range and greater variability. I have often known the relapse set in as suddenly and sharply as the invasion, but generally it is not so, at either morning or evening; and the second seizure tends to begin in an interrupted or gradual manner, which has not been seen at first.

Pulse at initiation.—The pulse is quicker or slower as the rise of t. is more or less marked; but in every series of cases examined there was an evident initial slowness of the circulation, which in some instances extended over 1 or 2 days.

Perturbatio critica.—Probably this phenomenon was somewhat more frequent in this relapse than at invasion; its general character being the same. A clear instance was that already described in the woman's case No. IX., when at 3 P.M. the t. had quickly risen to $108^{\circ}6$, p. 150; an hour afterwards the heat had declined to $104^{\circ}2$, the pulse not having changed, being doubtless too rapid to allow of precise measurement by ordinary means. *Vide also CASE XVII.*

The crisis in First Relapses.—Was generally more pronounced than either at invasion or in late recurrences. In 85 p. c. of cases it was noted at morning visit, and so far corresponds in time with the normal decline of animal heat: reaction was then usually prompt: in 15 p. c. of instances the fall happened mainly or wholly during the day, usually being complete by the following morning.

Rate of decline of temperature.—This does not appear to be more uniform than at invasion; e. g. being for the completed phenomenon in the woman's case above, No. IX., $12^{\circ}6$ (including the critical perturbation) in 19 hours, temperature falling quickest at first and midway (about $1^{\circ}5$ per hour), very slowly at the last (1° per hour); the event occurred at night. Once there was a decline of $9^{\circ}8$ in 22 hours, being quickest at first ($1^{\circ}6$ per hour) and also at the end (8° per hour); this was a day fall: and a third instance showed a decline of $6^{\circ}8$ in 11 hours, rather quickest at first (1° per hour); subsidence during the day. In two very brief relapses, a night decline at rate of about 1° per hour and of moderate degree was noted, being as usual rather slower as the minimum was approached. Other quicker rates for short periods were recorded, one of the more striking being noted at the height of the epidemic; patient a man of 18; t. at 5 P.M. $105^{\circ}8$; half an hour later t. 99° , twelve hours later 96° ; here the main decline of near 6° took place in the first half hour of crisis.

Degree of critical fall.—The mean elevation of fully developed first relapses being about 1° greater than that of ordinary invasion, by nearly as much is the crisis more marked in them; its actual degree depending

partly upon previous severity of fever. Thus, in relapses of 4-7 days' duration the mean min. t. was $96^{\circ}2$, and in those of 2-3 days' duration about $97^{\circ}5$. The more prolonged critical decline was not necessarily the most pronounced, but rather the contrary: for crises quickly completed were those furnishing generally the lowest thermometer readings: exceptions, however, were met with, and the most striking of my early series was one of e. fall prolonged till next m., when was read the low t. of 94° ; there was another instance of decline prolonged from one e. to the next e. after a marked seizure of 6 days' duration, when the thermometer reading was $95^{\circ}2$. These phenomena are striking.

Low critical t. usually attended either 'continued' pyrexia or those relapses of over 4 days' duration in which the number of distinct exacerbations was fewer than the number of days occupied by the fever. Acute isolated paroxysms seldom had a deep fall, but prolonged exacerbations were often followed by a great descent: intermissions during a relapse did not imply a deeper fall at the end.

The Pulse at crisis.—Decline in frequency of the heart's action whilst following the temperature was less pronounced and less regular: it was most considerable in the marked and abrupt crisis, yet in general striking and unusual perturbations of temperature were not immediately attended with such fluctuations of pulse as might be anticipated if the normal ratio of t. and p. were maintained during fever; and in every instance at the end of crisis, did the pulse remain in excess of the temp. as measured by the mean normal proportion of $98^{\circ} : 74$. The amount of this excess varied according to idiosyncrasy of subject and degree, and probably rate of fall. When the body-heat began to be restored, the pulse still continued languid, and for a time even declined. In three ordinary cases the pulsations at acme were respectively 150, 132 and 120, with t. of $108^{\circ}6$, $104^{\circ}8$, 103° ; and at end of crisis 86 (t. 96°), 70 (t. 95°), and 88 (t. $96^{\circ}2$).

Duration of First Relapse.—In a series of 167 cases seen between May 1877 and October 1878 the mean duration was 4.75 days, mean extremes 4 and 6.2 days; actual extremes 1 and 7 days; average duration 4.3: hence at its first recurrence the spirillum fever though usually very distinct, and occasionally nearly as pronounced as the invasion, may yet be exceedingly brief. At invasion-attacks of man there is no such wide variation; yet almost as great were noted in the monkeys' first and sole attack.

Having reviewed the fully-developed relapses as a whole, I now subjoin a brief description of each individual series included in the group.

7-day relapses: rare: Of 4 instances, 1 approaching the continuous type, 2 remitting, 1 intermitting: it is noteworthy these long events were never composed of a single upheaval of temperature, but by depressions on 2nd and 5th mornings (deferred sometimes till 3rd m. or 5th e.) their construction of 2-3 main paroxysms, of varied prominence, was more or less clearly indicated, max. t. high and sustained: onset prompt and fall either direct and moderate (m.) or prolonged and deep (e.)

6-day relapses: 8 cases; a compact group of high pyrexias, exhibiting 2, 3 or 4 main paroxysms, those on 2nd and 5th days being always

indicated by corresponding m. depressions; general course of fever ascending and remittent (6 times) or level or descending and rather continuous (2): max. t. high: onset chiefly m. and prompt: fall marked and at m.: intermittency not seen in this group.

5-day relapses: 8 cases in a compact series of intermittent type, the contour once only being in part continuous: main paroxysms 2 (by dip on 3 m.) or seldom 3 (when another decline on 4th m.); general height of fever moderate; contour level, with depression near mid-course: max. t. 105° , min. t. 101° : m. onset 5, sometimes rather low: fall at m. and of mean degree.

4-day relapses: 21 cases: fever lasting 3 days and crisis on the 4th: never quite uniform throughout, but divided by deeper remissions on 2nd or oftener 3rd m.: a main depression on 2nd e. and similar anomalous movements now seen, probably being peculiar: 1 or 2 main paroxysms, the last culminating in the *perturbatio critica*, as seen in the case of a girl with acme at 108° and fall the same night to at most 97° ; m. onset 8, e. onset 13: general height of pyrexia not quite equal to that of longer attacks: fall prompt and decided. In this large group fever markedly remittent, with tendency to change; this character being most apparent amongst Hindoo agriculturists, whilst amongst Mussulman weavers the tendency was to less remittent or even continuous type.

3-day relapses: 10 cases, with fever estimated to last 32–44 hours: t. range $101^{\circ}5$ or larger than in longer recurrences; fall usually moderate: onset e. 6, m. 4: composition of 2 distinct paroxysms, varying in height, continuous or sustained (2), remittent 5, intermittent 3: the e. t. always predominated. Bi-daily observations are insufficient to determine the actual duration of the febrile paroxysms, and a correct estimate of these brief relapses, which offer a natural analysis (as it were) of the spirillar pyrexia, is still a desideratum.

2-day relapses: 18 cases. As fever lasts but part of one day, subsiding on the next, these events appear in common charts as single, isolated paroxysms resembling other brief febrile attacks of various characters. Onset—noted at e. 11 (previous m. t. 98° – 99°); noted at m. 7, with t. 99° – $103^{\circ}6$ and the lower rising during the day (previous e. t. about the mean); time of advent of fever prior to 7 A.M. not known, but it certainly may occur in the night and thus prolong these brief relapses to the 3-day series: estimated duration of pyrexia 6–48 hours during these remarkable single, isolated paroxysms; but when most prolonged, doubtless remissions occurred midway. Not very seldom the main paroxysm was preceded or followed by minor exacerbations, for a day or more; and two distinct events might occur separated by a day's apyretic interval, such occurrence being repeated also as second relapse. Temp. $100^{\circ}8$ to 106° , mean $103^{\circ}3$; acme at e. almost always: critical fall, seldom marked but the t. has been known to descend to 97° after highest acme, and, as if to show how peculiar the fever, it was once noted at 94° after an acme only about the mean: possibly with more frequent observations such sub-normal fall might be generally detected, for in a later instance than any of the above, and examined at 3-hour intervals, a min. t. of 96° detected at 5 A.M. would have been overlooked

in the ordinary chart which gave 2 hours later $97^{\circ}2$: this point is of interest in a diagnostic sense.

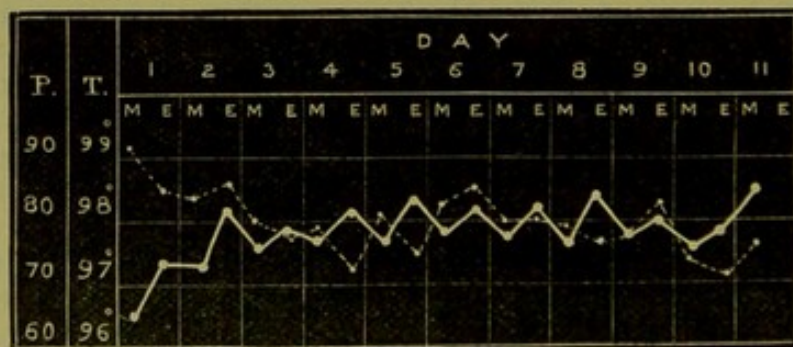
Date.—Both fully-developed first relapses and these highly abbreviated forms may be said to commence after an ordinary apyretic interval of similar mean and extreme duration: for the present series these being 8.3 days and 3.12 days respectively; the average 6–10. No instance occurred of an isolated spirillar paroxysm after invasion, outside these limits; and at the earlier of these dates but few were seen, in small children and women. The patients generally were of ordinary type and condition; nothing being noted to indicate the reason of their partially suppressed relapse.

The pulse rose with the temperature, and in general proportionately: sometimes it was slower than might have been anticipated, or lagged behind. In the briefer attacks the mean p. at m. prior to e. rise was 83; and 75 at e. prior to subsequent rise (the difference being notable): at acme its mean was 105 with mean t. of $103^{\circ}2$; and at fall 83 with t. 98° , commonly sinking a little lower afterwards, but not in a definite manner: sometimes the p. was slow throughout: the max. was 120 with a t. of $106^{\circ}6$. In attacks lasting two full days or a little over, the p. attained 112 with t. $104^{\circ}7$ at acme; and declined to 81 with t. $96^{\circ}6$ at fall; afterwards becoming still slower in some cases; at the min. t. of 94° , it was 80: it may continue rapid, or still rise, after the acme of temperature.

Second Apyretic Interval.—The second interval lasts longer than the first and is, like it, a quasi-normal state; the general level of body-heat is slightly lower, but the course is alike: rebounds and complications are less frequent, the tendency to convalescence being more evident. The pulse is, on the whole, slightly quicker than it was during the first interval; it follows a similar course. Fourteen typical examples have been analysed, uncomplicated and taken at early and late periods of the epidemic: the majority correspond fairly well, but the four cases of weavers show more variations of p. and t. than were found in the robuster agricultural immigrants.

The following tables and charts were those of a series of cases seen at the J. J. Hospital in 1877.

TABLE XI.—MEAN T. AND P. IN SECOND INTERVAL.



M. 96.7 97.3 97.6 97.7 97.7 97.9 97.8 97.7 97.8 97.6 98.4
 E. 97.3 98. 97.9 98.1 98.3 98.2 98.2 98.3 98. 97.8

Individual charts show many variations: the mean morning t. was highest on 6 day; mean e. t. were always higher than m. t., the maximum being on 5 day: the m. pulse was higher than the e. p. at first; the p. declined often considerably at the end. The cases collectively show a distinct perturbation of T. and (more) of P. about the 6th day, which consisted of a rise and fall: in another series the p. alone indicated this disturbance, once falling to 64 on the sixth m. and rising to 92 on the following m.: general symptoms may be quite absent, and the blood-parasite has not yet been found on these occasions.

The above chart refers to cases in which a second relapse occurred: regarding the corresponding period not followed by relapse, I found similar indications of systemic disturbance (best manifested by the pulse rising and falling) on the 6th, 8th and 10th days: these perturbations are, however, most marked in the relapsing set, beginning a day or two before the attack comes on, when the blood-parasite may be present. There is a third series before me in which, whilst the general course of t. was nearly level, more marked variations of both t. and p. were present; the t. being most depressed and p. most variable, when a second relapse took place: this set belonged to the weavers who in general displayed a lower type of fever, and more minor complications.

Reckoning from the first rise after crisis, the initial t. ($96^{\circ}5$) is rather lower than at the beginning of the first interval: the pulse is usually about 80, but varies considerably: at the end whilst the t. becomes almost normal, it is apt to decline considerably.

The duration of the second interval was 9 days in four instances, 10 days in three, and 11 days in two: another series showed a range of 6 to 14 days, the tendency being towards the longer periods.

Second Relapses.—These hold the same relationship to first relapses as those to invasion seizure; and the pyrexia is found to become still briefer, less sustained and less pronounced: it might usually be termed mild, and there were gradations of decline to mere febrile perturbation, hardly to be perceived. Such slight indications of the relapse were probably common, but might occur at night and hence appear in the chart as a depression following fever.

Form.—Amongst 27 examples continued pyrexia none, remitting 7, intermitting 13, isolated paroxysms 7: contour of remittents mostly level or descending, of intermittents more varied; the longer relapses sometimes showed an increscant course of their component paroxysms. The tendency to disintegration and dissociation of pyrexial elements, or even their partial suppression in mid course, being combined with mildness and brevity, it follows that these later relapses are, in all respects, less severe than the earlier.

Temperature.—The max. t. for all forms was 105° , seen only four times: for both remittent and intermitting the mean max. was $103^{\circ}3$; for single paroxysms $102^{\circ}5$, these data pointing to a tolerably well-defined limitation. In the absence of continuous fever, a distinct *perturbatio critica* was not seen; but the final paroxysm of a remitting attack was sometimes so predominant, as to suggest that here might be perceived the real paroxysmal character and relation of such final exacerbation.

Minimal t.—A morning t. below the normal at initiation of relapse was common, as resulting from the generally depressed state of the preceding apyretic interval: remissions of pyrexia were usually well marked, and the not infrequent intermissions at 2nd morning, or even 3rd, may descend to 97° : such low mid-temperatures were extremely rare during the two earlier febrile events, here they were noted in at least one-fourth of comparable instances.

When the low level was prolonged, the number of febrile paroxysms became reduced to fewer than that of days: in similar instances the m. t. was occasionally higher than e. t., contrary to the rule otherwise invariable here.

The *crisis* of these modified relapses was seldom marked; but its occurrence was sometimes indicated, after even isolated paroxysms, by a depression below the normal not seen in other kinds of brief fever. The rate of febrile subsidence was either abrupt or more gradual, when the minimum t. was not attained until the 2nd or 3rd, m. after main decline: this feature also appears peculiar.

Range.—The extreme daily range noted amounted to 7° , but the mean was considerably less than this, owing to the very moderate height of the usual paroxysms; still, however, being in excess of that in earlier relapses attended with so much less tendency to intermit.

Duration.—In 7 instances the pyrexia lasted a part or the whole of 1 day, the fall being noted either next m. or not until e. (whence a varying duration of these briefest forms); in 6 the relapse was of 3 days' standing (2 remittent and 4 intermittent); in 8 of 4 days' (3 remittent and 5 intermittent); in 3 of 5 days' duration (2 intermitting); in 1 of 6 days', and in 2 of 7 days' duration, these last examples being intermittent in character, exceptionally long and possibly complicated. This enumeration is instructive as pointing, by a sort of natural analysis, to the ultimate construction of late specific febrile events; and also indicating how near the fundamental elements approach to those of malarious fever, or possibly of all other acute febrile infections.

The Pulse in second relapses.—The pulse follows the temperature, though not very promptly; hence at the first rise, it often seems comparatively slow (*e.g.* $100:104^{\circ}\cdot4$) its maximum not being noted until several hours after the indicated maximum t. ($110:103^{\circ}\cdot4$ in an example just quoted): for the same reason the p. is apt to be quickest at the close of a prolonged and uninterrupted third attack; there were instances also where the pulse did not rise at an initial paroxysm, with a t. of 101° or 102° ; though it sank with the mid-depression, and rose with later exacerbations. So far as seen, its frequency was never excessive, *e.g.* $104:103^{\circ}$, or $110:104^{\circ}$; yet there are here, as well as at other times, individual exceptions of a rapid pulse throughout, and also for a brief interval after an attack; in children, too, the p. was always quick, even during the apyretic state. An interesting case is included in the present series, which serves to show that the p. follows the t. and not alone spirillar inspection of the blood; and there are others showing a minimum pulse in the mid-depression of an interrupted specific relapse, so that it again seems as if these late events were composed of distinctly separate recurrences. With a gradual rise of t. culminating in, or underlying, or following an acute paroxysm, the p. also rises above the mean;

and in perfectly intermitting paroxysms of specific character, it has been found to sharply vary in rapidity.

Owing to the extremely diverse forms assumed by second relapses, I have found it impracticable to draw up a useful table and chart of mean t. and p.

Third Apyretic Interval.—With the rarity of 3rd relapses, this corresponding incubative period becomes equally uncommon in hospital practice; and in an earlier series of 150 cases of relapsing fever, only 6 instances were met with, the 3 better verified being here quoted. In 2 its duration was 14 and 17 days, or 5 and 4 days longer than the second interval, and 7 and 10 days respectively longer than the first, and in the 3rd case, its duration was also 17 days, the previous apyretic intervals being of average length but interrupted by intercalated paroxysms. From these data (which are confirmed by others) it appears that whilst the successive febrile events diminish in duration, their incubation-stages progressively increase, the means here being 8, 12 and 16 days of first, second and third intervals respectively.

This prolonged stage may differ but little from the normal; once there were complications, in two cases the t. was slightly depressed at first: a brief decline on 9th day (t. $97^{\circ}2$ and p. 76) with some headache and pains in the joints was noted in one of these two; and in the other, similar general symptoms on 10th day without evident alteration of t., yet with a p. then at maximum (84). Trivial as they seem, such indications of periodic perturbation are comparable with the similar and more marked disturbances seen during the preceding intervals, being also probably of specific nature. The crossing of t. and p. shortly after crisis is only occasionally seen in these late charts.

When no recurrence ensues, the third post-febrile period may speedily assume the normal form; sometimes, however, there were minor perturbations of t. or p. with or without slight general symptoms, about the 10th day; e.g. in one instance on 12th and 13th days with no recorded deviation of M. and E. temp., the m. pulse promptly declined to 48 per minute, and then rose to its previous level of about 68; headache, giddiness, debility, pains in the joints, were complained and the spleen was enlarged: a minuter scrutiny was not made.

Third Relapses.—These are milder, and at least equally varied; being either distinct events, not to be overlooked, or comparatively trivial, or so obscure as to become hardly distinguishable: 6 examples are here analysed, the charts being of ordinary kind.

Temperature.—The pyrexia lasted 1 day once, 2 days twice, 3 days twice and 4 days once; its onset was at M. 3 times and as often at E.; its termination usually noted in the morning: the prolongation beyond two days was attended with gradual decline, uniform or remitting.

The form of fever was that of isolated paroxysms (3 times) or remittent; the tendency always being to early acumination, and hence the general form paroxysmal, rising abruptly and remitting by degrees.

The intensity of pyrexia was moderate, mean max. t. 103° , extremes $104^{\circ}8$ and $99^{\circ}8$: in absence of marked crisis the mean min. t. was only $98^{\circ}2$, absolutely lowest $97^{\circ}4$, which contrasts greatly with earlier crisis; here, again, a decided sinking of the t. is consistent with previous very

moderate rise. The single, isolated paroxysm of one day attained $104^{\circ}2$; of two-day seizures the max. was $103^{\circ}2$; of three-day $103^{\circ}6$, and four-day events $104^{\circ}8$; hence no rule was apparent here: the t. at morning, especially at first, tended to exceed that at e.: the whole daily range was from 1° to 5° , and neither first rise nor end usually reached the full range.

The Pulse in third relapses.—Whilst following generally the temperature, its course is often more variable and not concordant at morning or evening; its comparative exaltation is less (the velocity never being excessive) and its range more limited: thus with t. of $103^{\circ}2$ the p. was 96, and with $103^{\circ}8$ it was 102 (isolated paroxysm): when the relapse was rather prolonged, the p. did not rise promptly and might not attain its maximum until after the t. has begun to decline; it has been seen to remain low whilst the t. was rising. At the end, it may not decline below the normal; or not until two or three days after the slight crisis.

Fourth Relapse.—For the comparative discussion of the latest events in more prolonged spirillum fever, no adequate materials were procurable from hospital practice; since patients showing a tendency to frequent repetitions of the fever, could not by persuasion be detained in the wards long enough to allow of demonstrated absolute convalescence. There is but one instance of a Hindoo lad who after a well-pronounced first relapse, had three other minor attacks at increasing intervals of 5, 7 and 11 days, but he left before the end of the series was determined. Here the fourth interval of 11 days displayed a level temperature 1° below the normal; the pulse was also uniform about mean, except at first when it was depressed, and again at the close when it sank on the second morning before onset of fourth relapse. This event was represented by a distinct, isolated paroxysm extending over two days: acme on the second m. with t. 104° and p. 120: both commencement and decline were abrupt: at the m. fall the t. was $97^{\circ}4$, whence it slowly rose to $98^{\circ}2$; the p. being 76 and gradually declining to 60 on the third m., after which it rose to a level still below the previous average: the succeeding five days of his stay displayed a common level nearer the normal than in the last preceding interval.

Comparison of Temperature movements and Pulse, during the Fever of Man.—The generally uniform concordance of temp. and pulse observable in health was invariably indicated during the febrile state, a suspension or reversal of such concordance being unknown; personal variations noticed in health were also repeated in disease: these statements are of clinical importance. Besides the many M. and E. pulse data collected from the beginning of my enquiries (for I endeavoured always to associate clinically these elements of the pyrexial condition) which are analysed below, there are some tri-hourly memoranda (also detailed here) which may be summed up as follows.

Brief isolated paroxysm.—F., 50, whilst the body-heat rose 6° , the pulse quickened only 26, but six hours after the febrile acme of attack it became a little more rapid, although the temperature had declined $5^{\circ}2$, hence in even a short paroxysm of 12 hours' duration, the pulse attained

its maximum less promptly than the temp. it also declines more slowly : see the Chart 9a PLATE IV.

During a longer specific paroxysm (M., 35) the pulse remitted much more strikingly than the temp., and after the common acme it declined more slowly, not having subsided to within 10 beats of the normal at the end of fall, when the temp. had sunk 8° , or to $2^{\circ}5$ below par.

In the case of the lad S. J. (*vide* Chart 2), the pulse quickly rose to its maximum, its subsequent course being however sustained ; and at the crisis it declined more slowly than the temp. In detail, it was seen to remit at the beginning when the temp. did not, it also declined at high levels when the temp. did not, yet it did not subside so quickly at the crisis.

In the case of the woman M. (*vide* Chart 3), whilst there was a distinct concurrence of pulse and temp. throughout the relapse, yet their independence was shown by the varying change of level shown by the pulse, and hours are noted when the pulse lagged or fell not in accord with movements of body-heat, the reverse conditions not being so evident : if confidence be placed in these last observations, the divergencies I now note become of much interest, and certainly claim attention. Other more apparent features of the spirillar pyrexia are shown in the Chart, and are detailed below.

I have not access to corresponding data with regard to other 'fevers,' but from a few observations made in malarious attacks, should not suppose these comparative movements of temp. and pulse to be peculiar to the spirillar pyrexia.

Comparison of Febrile movements with Normal daily course in Man.—Four examples of spirillar pyrexia lasting 12 hours ⁽¹⁾, 30 hours ⁽²⁾, $3\frac{1}{2}$ days ⁽³⁾ and 4 days ⁽⁴⁾, showed that brief fever may culminate at 10 P.M. ⁽¹⁾ and may remit in its progress upwards ⁽²⁾ ; that the beginning of a longer attack may happen during the night in anticipation (as it were) of normal rise, the high level be contemporary with or oftener prolonged after hours of normal elevation, the ensuing decline (so-called 'remission') being also delayed till 7 or 10 A.M. ; at close of attack, as at initiation, variety is more probable, as an added exacerbation or remission not represented in the mean normal chart : the critical fall may occur between 1 and 10 A.M. ⁽³⁾ ; and lastly the longer attack displayed a similar P.M. initial rise, remissions within normal range and exacerbations so prolonged as to occupy the midnight hours usually attended with decline of body-heat ; this last fact clearly indicating the operation of superadded pyrogenetic agency. From these data I infer that there exists a decided tendency of the specific pyrexia to follow the normal daily cycle, subject only to question of degree.

I note, too, first, that a reversal of the entire daily normal movements of body-heat was never witnessed ; next, that some, at least, of these mid-febrile movements might be termed exaggerations of normal variations : and lastly, that the prolonged rise and decline of febrile temperature might be regarded as due to suppression of paroxysms, nearly or quite complete.

Comparison of Febrile movements in the Human and Quadrumanous subject.—In comparisons of the kind here attempted, it is not required to

include minute perturbations, nor is it the degree so much as the course of temperature which is important ; a similarity being established when the general course of body-heat is alike. Although its normal standard is about 3° F. higher than that of Natives, yet such similarity of the monkey's temperature to man's is evident enough ; and hence the inference of febrile movements also being possibly similar.

The common mean daily cycle in health may be estimated as follows—a low level from 11 P.M. to 5 A.M. (minimum soon after midnight); a rising of body-heat from 5 A.M. to 11 A.M. ; a high level 11 A.M. to 5 P.M. (maximum near the close); and a final period of decline from 5 P.M. to 11 P.M. The febrile movements in Man being already indicated, I found those in the smaller animal to be thus:—in 11 instances of specific pyrexia lasting part of a day, the beginning fell within limits of normal rise 10 times, once being rather earlier ; the acme of attack came within high level limits 8 times, being earlier 2 and later 1 ; the critical fall concurred with limits of normal decline as often, being twice somewhat deferred. In 7 more pronounced attacks lasting from $1\frac{1}{2}$ to 4 days, and altogether including 21 distinct day paroxysms, I found 18 of these paroxysms happening within normal elevation limits and 3 coming on later ; whence appears the strong tendency of spirillum fever to concur with the normal rise, exacerbating also at rise-periods or soon afterwards ; the acme of fever was seen 19 times during normal, high level limits, tending to be deferred till night and the latest at 10 P.M. : 'remissions' or partial decline of temperature were noted at both normal decline and low level periods, and the critical fall rather oftenest occurred within low level limits. Apparent exceptions to rule here being due to the febrile state anticipating or extending beyond normal high level periods, it may be said that the course of fever was essentially the same as in man.

Lastly, having reduced the comparative charts to the common clinical form and contrasted them with ordinary charts of contemporary human patients, I find as follows:—in Man of 22 febrile paroxysms seen at close of invasion-attack, 19 were entered as E. the 3 at M. including strictly abnormal critical perturbations : the 'fall' was entered at M. except when taking place by lysis or prolonged more than usual. Of 41 paroxysms seen in the relapse 37 were entered at E. : of 12 initiations 9 were noted at E. and of 12 critical 'falls' 7 were noted at M. the remainder being prolonged later. So in the comparative series, 7 of 9 brief attacks came under E. ; and of the longer attacks all 7 beginning at E., 15 of 16 mid-paroxysms were also entered at E., and 6 of 7 critical falls at M.

The force of these statements rests on the fact that when the detailed normal charts of either Man or Monkey are condensed into the ordinary M. and E. charts of clinical medicine, there is shown simply an E. rise with M. decline : and since in pyrexial charts most rises or exacerbations were marked at E. and most remissions or crises at M., the correspondence of main normal and abnormal temperature movements becomes apparent. Such correspondence may not, however, apply to details ; for, so far as known, subsidiary perturbations are more marked and probably more numerous during spirillum fever than in health.

Non-spirillar fever.—Respecting later relapses and other more incidental sequellar paroxysms, not being in possession of fully detailed data I can state only that in ordinary charts the temperature movements were

similar to the verified specific. Also with regard to rebounds or secondary fever, the E. exacerbations and M. remissions being common where pyrexia is maintained (the beginning and ending almost necessarily offering variations), it may be inferred that these features belong to several forms of 'fever': and such inference would be supported by the charts of my experiments on monkeys with salivary poisons, by those of pyæmia, hectic and certain other forms of symptomatic fever, the instance of enteric being good for essential pyrexias.

No peculiarity therefore can be claimed here for spirillum fever.

Variations of Spirillar Fever.—Commonly the results of infection are so complex and prolonged, that *a priori* it might be supposed they would be liable to much variation; and as matter of fact, they are never identical in my two cases. More remarkable, however, is the natural tendency of successive febrile attacks to diminish in severity, and to become deferred in occurrence. And, briefly, the variations I am about to consider are essentially modifications of this progressive decadence, as it affects either febrile stages or apyretic intervals. Changes in the 'type' of fever are not here included (*vide* Chapter VIII. of this section); and fever with complications is not now referred to.

Variations of Pyrexia.—Regarding the several stages of Invasion and Relapse, since the spirillar pyrexia is never absolutely continuous and never exceeds the limits already named of duration and intensity, variations here stand for unusual phases of checked development, as represented by shortening, sinking or disintegration of pyrexia; or non-specific paroxysms may be mingled, and the briefer relapses apparently lose part of their specific character. Regarding the sequence of phenomena, whilst the natural tendency is to subsidence, as extremely rare exceptions this order may be reversed and become increscant; or it may be interrupted by intercalation of an additional paroxysm, or by apparent reduplication of a relapse; and, on the other hand, a relapse may be deferred. An individual may undergo repeated infection; and, for convenience, instances are appended to this Chapter. Truly errant forms of spirillum fever were not numerous, and such as are known being connected by intermediate gradations with the common type, it would be difficult accurately to estimate their relative frequency.

Some possible modifying influences were the following:—

Age.—Infants at the breast displayed relapsing attacks with high pyrexia, or died during the invasion as do adults; some children under puberty showed quite typical relapses with high fever (*e.g.* 108° at acme of 1st recurrence by a girl of 10), or more irregular intermitting pyrexias: the critical fall of temperature was seldom pronounced: single attacks seem to be rare. All cases belonged to affected families: girls were unusually frequent in the lists, a malarious taint and the presence of lumbrici were common. In all young subjects the pulse was very frequent. Old age does not entail essential modification of specific pyrexia.

Sex.—Amongst women aborting the spirillum fever was not apparently severer than usual, or complicated with local disease or secondary fever, thus 3 of 6 cases actually witnessed were all relapsing, the invasion-attacks being moderately prominent and the recurrences (after

abortion) brief or irregular: in three other instances known chiefly by the patient's testimony, no peculiar features were observed.

Typhus biliosus.—The connection of this fever, of either abortive or relapsing form, with pronounced jaundice and the typhoid state did not necessarily lead to peculiar modifications of the course, duration or intensity of pyrexia.

Malarious influences.—Its conjunction with malarious fever probably did not entail essential change in the earlier or more pronounced specific attacks; and with reference to later, mild relapses, there exists at present considerable difficulty in discriminating the real character of such pyrexia.

Symptomatic fever.—May be so blended and continuous with the specific that an attack really composed of two distinct elements might, without aid of the microscope, be erroneously regarded as of uniform character throughout. This remark applies also to the complication with secondary fever. Variations of pyrexia witnessed in fatal cases may be considerable, without displaying any particular form.

Varieties of Invasion.—Judging from actual observation, a prolongation beyond 8 or 9 days must be excessively rare; and I placed no dependence on the statement of patients naming 12, 14 or more days as the duration of their first illness; nor, I may add, is it likely their assertions were correct that the fever then intermitted. There is, however, analogical testimony showing that the primitive spirillar pyrexia may vary much in duration; for in the *Quadrumana*, I found its range to be from 6 to 86 hours, these extremes far exceeding in relative proportion any yet noted in man. Amongst ordinary hospital patients the first attack was never seen throughout; and the comparatively few individuals seized in the wards furnished the instances quoted below.

1. The sole attack may be mild though continuous, and not lasting more than 3 or 4 days. Notes of three such cases are before me, and I feel confident that but for special blood scrutiny made with the microscope, they would have escaped notice in the crowded wards. Such were possibly only samples of this fever strictly comparable to the mildest forms of typhus and enteric fever; which pass unrecognised except, may be, under the vague term of 'simple continued fever.' Moreover, in the East, malarious pyrexia of many minor degrees being common, there arises even greater likelihood of confusion; and though I have not, as yet, seen in man the highly abbreviated simple paroxysms following infection in the monkey, yet on this negative point much reserve is needful; and personally I should not be surprised to learn that the sole febrile evidence of spirillar infection amongst men, may consist of brief pyrexia but little more pronounced than is shown below to obtain in some relapses.

CASE XXVIII.—S. A., 35, lascar fireman, admitted August 1877 for mild ague, dysentery and secondary syphilis, general condition fair; there were occasional rises of temp. never above 100° F. for the first week, when pains in the joints and ulcers on the chin came on, but he was gradually improving when October 10 he had a sharp ague-like paroxysm, e. t. 105°·8, p. 126, which left with sweating the next day: I examined the fresh blood during fever and could not detect the spirillum. A small sore now appeared on the penis, and in 9 days (no fever meanwhile) the temp. again suddenly rose with chills e. t. 103°·6, p. 120, and it remained high for

part of 3 days, then abruptly declined to $97^{\circ} \cdot 2$, p. 80; promptly regaining and keeping at normal level for 10 days, when he insisted on his discharge. On the second day of fever m. t. 103° , p. 120, I found the blood-plasma clouded, fibrillation close-set and thick, white cells, large granule-cells and free protoplasm present, red discs piled; numerous, active and rather large spirilla. Throughout hepatic or splenic implication not detected, no jaundice, but vomiting at the crisis, no distress: after fall, much weakness and general aching pains. There were other specific fever cases in the same medical ward at the time of his illness, and I regarded the case as one of mild infection in hospital, which also might have been overlooked.

2. The attack is irregular in form, being remittent, intermittent, or even interrupted in its course, and as this happens with severe general symptoms, it is evident that non-continuous pyrexia does not always imply mild disease. Some of these temperature-charts are so little like those of ordinary first attacks, that it once more becomes apparent the affection could not be recognised from them alone. How often cases of this kind occurred amongst ordinary hospital admissions, I am unable to say; those recognised were found only amongst patients seized in hospital with specific fever, there being 4 or 5 in the series of 30 instances: the example quoted below might have been overlooked, had I not been struck with the man's physiognomy. This case I regarded as one of infection in hospital, yet should point out that although 16 days had lapsed before the man showed symptoms, yet it is just possible an apyretic interval might extend so long, and the attack seen have been only a relapse: this is matter of judgment.

CASE XXIX.—M., 30, famine-immigrant only 8 days in Bombay, was admitted with looseness of the bowels and pains in the large joints; the former of 2 months' duration, the latter of fifteen days; no mention made of fever: t. on admission $99^{\circ} \cdot 8$, p. 84: much debility, no swelling of the joints, chest and abdomen seemingly unaffected; the above symptoms abated in the course of a fortnight, and it was noted he is improving. 16 days after admission paroxysmal fever of intermittent and interrupted progress suddenly came on, lasting six days, and without manifest crisis, being succeeded by two or three minor brief elevations of temperature, which were non-specific in character: discharged convalescent. For the Chart see PLATE IV. No. 5.

3. In children an isolated attack of spirillum fever may be so disguised as regards pyrexia, that without extraneous aid the affection would in India be referred to smart febricula, or aguish attacks of undefined character; yet the blood-parasite may actually abound at the time. Amongst infants and children, however, the vagaries of febrile reaction generally are well known; and perhaps the most prominent instances of irregular compound attacks of this fever which I have seen, happened in young persons: much care is therefore required for discrimination of specific pyrexia, and the microscope becomes indispensable here. The fever in the youngest seems either to kill rapidly, or to be but mild; prolonged and marked illness not having appeared in such subjects. Pyrexia may be high.

4. Fatal cases.—Variations of pyrexia are more numerous and considerable here than in the surviving, even when no complication is present; the initiation of illness being irregular and pyrexia ill-sustained, and not accordant with other symptoms: the pulse, too, varies much, although usually frequent. Such instances diverging amongst themselves, useful comparison becomes impracticable: but I would specially mention CASES XIII. and XVI. as examples of specific fever preceded for

three or more days by daily paroxysms of uncertain character, death occurring very soon after pyrexia became continuous: both subjects were infected from a common source. Not all fatal cases are thus irregular, at either beginning or course: when complications supervene, derangements of temperature become frequent. See Charts 15 to 19 Plate V. and 25 to 27, Plate VI.: examples at extreme ages are Nos. 16 and 17, Plate V. Lastly, the proportion of variations at invasion is not so considerable as in relapses—perhaps not more than one-tenth of all cases showing them; but much remains to be learnt respecting the lesser effects of primary spirillar infection, in man.

Varieties of relapse.—The order of natural decadence as indicated in Table III. is seldom widely departed from: variations of this kind known to me, include instances of a progressively increscant tendency, duplication of a relapse, and the intercalation of an additional paroxysm.

Respecting individual relapses, the same table shows how greatly recurrent attacks differ in their duration, and hereby in clinical prominence. No rule obtains, for after a well-defined invasion there may follow either a pronounced or insignificant second attack, and so after a well-marked first relapse the next recurrence seems indifferently either very distinct or, in a sense, imperceptible. The more unusual variations here correspond to degrees of clinical effacement, through abbreviation, depression, or disintegration of specific pyrexia. Every collection of Temperature Charts contained examples of this sort.

1. Variations referable to reversed or interrupted orders of occurrence.

a. Instead of the recurrent events more or less quickly declining, the first, second, or even third may sometimes be seen to augment in severity. Not uncommonly, the first relapse is rather more pronounced than the invasion-attack; and less often, it may be equally prolonged or even longer by a day or so; afterwards a second recurrence being rare. Two out of 11 native students seized in hospital with relapsing fever showed a predominating second attack, and in both the invasion had terminated by lysis rather than critically: the same features were noted in an adult woman attacked in the wards; and also in an adult Negro, whose illness more clearly than usual was traceable to contagion at home: amongst five other examples selected for analysis, I cannot now trace the way invasion subsided. In these cases there was also a tendency to abbreviation of the preceding attack, and the apyretic interval might or might not be shortened: in all the general symptoms were pronounced.

Inevitably little could be learnt of the beginning of these increscant attacks, but from a few data acquired amongst the hospital contagion series, I was led to suppose that spirillum fever here either begins mildly with a few isolated paroxysms, or is preceded by similar paroxysms of character unknown: this is a topic meriting attention as future opportunity may occur. A striking example of the increscant variety was the following:—

CASE XXX.—M., 30, famine-immigrant, thin and feeble; on admission said to have had fever for 10 days, t. $98^{\circ} \cdot 2$, p. 80, resp. 22; depression, headache, pains, some hepatic and splenic implication: the man had a sloughing ulcer on the back of the r. foot. There were febrile paroxysms on the 11th, 13th and probably 14th days,

and on the 15th the t. rose to 103° , p. 110; a few spirilla in the blood; splenic enlargement had preceded: on the two following days, rather milder paroxysms, and Mr. S. A. did not find the spirillum. With the exception of an isolated exacerbation on 24th day, the man now remained tolerably free from fever for 9 days, or till the 27th day of illness, when a pronounced relapse set in, fever high at acme $106^{\circ}\cdot 2$, p. 140, form remittent, duration 3 days; a few spirilla seen on each day; critical fall on 4th and 5th days; min. t. 95° , p. 60, much depression: rallying was slow, but the ulcer improved, and at the end of an interval of 5 days the t. was normal. A final recurrence now began (37th day of illness), onset sudden, range high, max. t. as before $106^{\circ}\cdot 2$, form remittent, duration 5 days, spirillum in the blood; critical fall next day, min. t. $96^{\circ}\cdot 4$: a brief rebound followed (with dysentery) and thenceforward the body-heat was at normal level for 35 days, when the man became an out-patient. The nature of the fever prior to his admission may have been malarious, or due to the ulcer, or spirillar: the first specific attack detected was short and intermittent, the second seen more pronounced, and the third still more prominent: so that the usual order of events was reversed: visible blood-contamination seemed to increase with repetitions of the attacks. It did not seem that the remarkable augmentation of specific symptoms was due to infection replenished through the fresh contagion in hospital, and there are obvious objections to such a view: the co-presence of a sloughing ulcer appeared merely incidental.

b. Duplication of the 1st and 2nd Relapse.—The following instances were recorded during my absence from Bombay: notes preserved and reconsidered:—

CASE XXXA.—M., 20, weaver; after the invasion-attack of 9 days, form intermittent and decline lytic, there was a brief apyretic interval of four days, and then a first relapse extending over 10 days, and cleft by a deep intermission of some hours' duration between 3rd and 4th days; termination by moderate crisis; no apparent complication to explain this renewal of fever. Then ensued a long apyretic interval of 19 days, level and uniform, when fever returned as a series of 7 intermitting paroxysms, varying in intensity, and of nature undetermined microscopically.

CASE XXXI.—M., 17, hospital sweeper (brother also attacked) caught specific fever in the hospital; the invasion-attack lasted 7 days, the first apyretic interval 6 days, and the first relapse 6 days: then followed an apyretic interval of 3 days, a febrile attack (non-spirillar) of 2 days, and another fever-free interval of 5 days; the sum of these minor periods being ten days, which might be regarded as representing one second apyretic interval which had been interrupted by an intercalary paroxysm (see below), or else as narrated, as two successive intervals separated by a relapse: the next recurrence therefore represented either a 2nd or 3rd relapse, it consisted of two moderately elevated febrile attacks respectively of 4 and 6 days' duration, which were separated by an intermission of 36 hours' duration; according to the record this recurrence was specific throughout: then followed a fever-free period of 19 days and discharge of the patient convalescent.

Both the above attacks were unusually severe: commonly, a like prolongation of high fever was found to be due to either complication or secondary pyrexia, as defined in this work; but since there seems no valid reason why spirillar pyrexia might not be re-duplicated, these instances are now quoted as such a very rare variation of relapses.

c. Intercalated Relapses.—Under this term are included febrile paroxysms happening between ordinary attacks of spirillum fever and apparently distinct from them. Such paroxysms were not very rare: notes of the five last examples seen, show their liability to occur during the first apyretic interval, though they were also noted in course of the second: they last 1 or 2 days, are isolated and pronounced (range 102° – 5°), and had no clear distinguishing marks from the many brief final relapses reckoned as 1st, 2nd, or 3rd: they appeared about midway of this interval, with 2–4 non-febrile days preceding and following. Both young and middle-aged showed them, and, it so happened, only men;

all subjects in average condition, and occurrence not exclusively limited to the later epidemic period. The preceding invasion-attacks were not peculiar so far as perceived, nor were the ensuing ordinary relapses ; so that no means appear of predicting such intercalations : local complications were seemingly absent. The detection of these phenomena needs care ; in 2 or 3 of the above 5 cases, the blood-spirillum was present, once the blood was not (I believe) examined, once I could not be sure of the blood appearances seen : thus, results of microscopic scrutiny resembled those obtained in the examination of other isolated, periodic paroxysms occupying the position of a relapse : when not seen the parasite may have been overlooked. Prognosis of the cases not more unfavourable than usual. Such intercalated paroxysms may be regarded either as additional or supplementary phenomena, or, on the other hand, as relics of a partially suppressed relapse : the question here appearing to be one of judgment as much as of facts. It happens that 3 of these cases belonged to families, other members of which were affected with spirillum fever not offering these forms : the following was one of them :—

CASE XXXII.—M., 35, Mussulman weaver, famine-immigrant from N. India, in Bombay 15 days and 8 days ill with fever (a not uncommon statement), was admitted with his son and other caste men in Jan. 1880 : pyrexia moderate, much prostration, deep jaundice, a copious eruption of pink spots and many spirilla in the blood. The *perturbatio critica* quickly followed, and a slight critical fall with rebound attended by pharyngitis ; 4 days after the crisis there occurred a smart febrile attack consisting chiefly of a single paroxysm, t. $104^{\circ}6$, p. 130, spirillum present : there came another fever-free interval of 4 days, and an ordinary 2nd Relapse lasting 3 days ; the succeeding apyretic period of 12 days showed temperature at normal level. See Chart No. 10, Plate V.

By means of such additional paroxysms the specific attacks within ordinary limits become multiplied, and the intervening apyretic intervals sub-divided. It sometimes happens that without any other change, a relapse (usually the first) comes on prematurely 3, 4 or 5 days after the preceding crisis, and it may then be decidedly prolonged. Such vagaries in the manifestation of spirillar infection were certainly not commoner than variations of malarious fever : probably others of the kind will become known.

2. *Variations of Individual Relapses.*—Fifth, fourth and most third relapses are commonly represented by one or two brief paroxysms, differing only in prominence : less frequently such stand in place of second, and seldomer of first recurrences. An example is shown in Chart No. 8, Plate IV., of a short, isolated paroxysm appearing as first relapse :—

CASE XXXIII.—M., 30. Invasion of 7 days with pronounced crisis ; first apyretic interval of 7 days, temp. nearly level at normal line, but pulse declining at end : fever at 9 P.M. preceded by chills lasting two hours, next m. t. $101^{\circ}6$, headache, pains in legs, the spleen enlarged, projecting an inch below the costal margin, not tender, no hepatic implication : pyrexia was reported to leave at 1 A.M. with sweats, some headache, pains and splenic fulness still persisted ; t. at normal level ; the spirillum was found before acme.

Relapses of three or more days' duration, whether first or second, are sometimes so disintegrated as to present a series of separate paroxysms.

CASE XXXIV.—M., 14. First apyretic interval 7 days, level ; first relapse 6 days.

much pronounced and trifold, being cleft by two intermissions; second apyretic interval 9 days, low and level; the second recurrence had the form of two isolated paroxysms, separated by a non-febrile period of 34 hours, the first being pronounced (t. 105°), the second reduced (t. $100^{\circ}5$), crisis moderate: the spirillum seen throughout the three days, disappearing at crisis. The fever set in at 1 P.M. with chills lasting an hour, headache, thirst, spleen not enlarged, sweating at end of first paroxysm; during the non-febrile intermission, the symptoms were slight although the blood was visibly contaminated; the final paroxysm began at noon without chills and left with sweating: convalescence prompt. See Chart No. 7, Plate IV.

CASE XXXV.—M., 28, after a deeply pronounced invasion-crisis, an apyretic interval of 10 days at normal level until the end, when the t. declined to 97° . The first relapse of 5 days' duration consisted of four paroxysms separated by three intermissions, crisis decided and prolonged: additional temp. readings and blood-specimens were taken before and during the entire attack: they show the pyrexia to have been distinctly paroxysmal as indicated in the ordinary chart, the paroxysms differing in duration and intensity: spirilla present throughout, even at deepest intermission (t. $96^{\circ}2$), being few and not increasing with the pyrexial exacerbations. General symptoms slight, no chills, sweats with defervescences, early splenic enlargement and soon after tenderness, which persisted a day or two after fever left; also some epigastric uneasiness, and weakness: no subsequent return of fever for 22 days. See Chart No. 6, Plate IV.

These instances demonstrate the existence of a variety of specific pyrexia, which so strongly resembles intermittent fever in some of its aspects, that without some practical knowledge of relapsing fever and the use of the microscope, its misinterpretation was certainly possible. The *Quadruman*a did not display this disintegrated variety of pyrexia with continuous blood-infection; but they showed 6 times in 16 as the sole result of inoculation, a single brief paroxysm of 6–12 hours' duration, which under ordinary observation would be no more prominent than the relapse in CASE XXXIII. above, and might resemble the quasi-latent forms to be next described.

Variation by subsidence of Pyrexia: latent and suppressed Relapses.—It seldom happened that during a distinct relapse the temperature was uniformly depressed, without the attack being also abbreviated: but in some feeble subjects and fever of low type, this variety was seen.

When a relapse ceases to be distinct and is represented only by a slight rise of temperature, with very little else to attract attention, the process of suppression may be said to have affected all its dimensions; and the Chart record becomes more or less obscure, according to the degree of arrest of pyrexia. In extreme examples there is seen only one or two brief m. or e. perturbations, or an unusual depression, or finally none but pulse changes.

Instances are numerous of third and later recurrences being thus indicated, but no opportunity occurred of thoroughly testing their true character. As regards second relapses, however, I acquired the details of a case in which the common chart shows only a brief e. rise of $99^{\circ}2$, with a depression next m. of 95° ; some additional observations made, however, prove this record to be imperfect, for at 10 A.M. of the first day the temp. had risen to $104^{\circ}6$ and at 9 P.M. it had declined to $95^{\circ}2$. Microscopic examination demonstrated the presence of many spirilla in the blood, which also showed its infective property when inoculated in a monkey. This datum therefore becomes conclusive: here the chief sign of relapse was a very low m. temp. following a very slight e. rise.

Another instance of the quasi-latent variety, occurring as a first Relapse, is the following:—

CASE XXXVI.—F., 46, feeble, admitted with several relatives and friends from one locality, at end of invasion-attack; moderate critical fall on 7-8 day: a slight rebound (t. 100° , p. 84) two days later concurrent with some bronchitis, thenceforward very slightly raised but uniform body-heat for 8 days. On 19th day of disease e. t. $100^{\circ} \cdot 2$, p. 64: next day, m. t. $98^{\circ} \cdot 6$, p. 74; e. t. $97^{\circ} \cdot 8$, p. 64, and no further change for six days till discharge. Here the slight rise on e. of 19th day was the only visible indication of relapse in the ordinary chart of temperature; but it happened that minuter observations were taken, those of body-heat from four to eight times a day during 16-20 day of disease and those of the blood twice daily during the same period. Thus it was learnt that at 10 P.M. of last day the t. rose to $104^{\circ} \cdot 6$, declining again during the night; and I also found visible blood-contamination on the 17th and 18th day (with no rise of t.) as well as on the 19th, but not next day when the pyrexia left for good: this combined testimony being conclusive evidence that a veritable relapse had taken place, when upon bare inspection of the usual chart the occurrence might be considered at best as doubtful. The clinical notes are as follows, those regarding the blood being stated in the special chapter:—16th day (date of possible relapse) m. t. $98^{\circ} \cdot 6$, p. 64, skin dry, tongue florid moist, no thirst, headache, pains or cough, slept well, one stool, spleen barely felt and not tender; e. t. $98^{\circ} \cdot 8$, p. 66, feels better, though weak: blood unchanged: 10 P.M. t. $99^{\circ} \cdot 4$, p. 64. 17th day.—m. t. 98° , p. 60, skin dry, no advent of symptoms, no heaviness of the body, good appetite, spleen unchanged, e. t. $98^{\circ} \cdot 4$, p. 60, feels weak but no discomfort: 10 P.M. t. $99^{\circ} \cdot 6$, p. 64. Blood—no spirillum in the morning, a few seen in the e. blood. 18th day.—4 A.M. t. 99° , p. 68, 6 A.M. t. $99^{\circ} \cdot 2$, p. 66, skin dry, no change whatever, but increase of appetite and more food asked for: spirillum present: e. t. $98^{\circ} \cdot 6$, p. 62, respiration quiet, tongue whitish, moist, no headache, giddiness, thirst or discomfort; splenic dullness 2 sq. in. only, not projecting to costal margin, no tenderness; no hepatic change; abdomen relaxed; she has a haggard look but perhaps not more so than yesterday, and protests she is well; eats heartily: blood-spirillum present. 7 P.M. t. 99° , p. 66: 10 P.M. t. $99^{\circ} \cdot 6$, p. 66, no discomfort. 19th day.—1 A.M. $99^{\circ} \cdot 4$, p. 64, no complaint: 4 A.M. t. $99^{\circ} \cdot 4$, p. 60, no complaint: 7 A.M. t. $99^{\circ} \cdot 2$, p. 62, skin dry, no headache, thirst or pains, slept, one stool: spleen barely to be felt, not tender: no detectible local or general change. 10 A.M. t. $98^{\circ} \cdot 6$, p. 60: blood-spirillum present: noon, t. 99° , p. 60; 2 P.M. t. $99^{\circ} \cdot 4$, p. 64; 4 P.M. $100^{\circ} \cdot 2$, p. 64, no headache or thirst, feels very weak, no chills, spleen felt but not at all tender or hard, no epigastric uneasiness, protests she has no ailment (spirillum present): 7 P.M. t. $102^{\circ} \cdot 2$, p. 78, feels chilly (*i.e.* after t. began to rise), no headache or thirst: 10 P.M. t. $104^{\circ} \cdot 6$, p. 86, skin dry, feels chilly, some headache (acme of the relapse). 20th day.—1 A.M. t. 102° , p. 84, headache more, no chills, no thirst: 4 A.M. t. $99^{\circ} \cdot 4$, p. 88, no chills or thirst, headache less, no sweating at any time in this attack: 7 A.M. t. $98^{\circ} \cdot 6$, p. 74, skin dry, tongue florid moist, less headache, no thirst, or giddiness, or pains; to relieve the headache she induced vomiting by tickling the fauces, vomit a green liquid: she looks a little worn, but walks about, appetite is less, the spleen is unchanged, being neither enlarged nor tender: blood-spirillum not seen. The t. declined a little lower, at 1 P.M. being $97^{\circ} \cdot 8$, p. 66. For the next day, it was still taken frequently, but there was no sign of change; convalescence was slow for the remaining few days she stayed in hospital. *Vide* Charts 9 and 9A, Plate IV. at the end.

With evidence like the above, I am disposed to interpret afresh three or four cases seen at the beginning of my researches, which displayed the spirillum at periodic dates corresponding to those of first and second relapse, when no perceptible rise of temperature appeared at m. and e. thermometer readings. These instances were, at the time, regarded as showing a relapse might be truly 'latent'; but it now seems most probable that owing to the long intervals of observation, some pyrexial disturbance had been overlooked, and that the 'latency' was apparent only. Whether or not visible infection can ever pass without involving temperature-changes, has yet to be conclusively settled; but, at present, evidence tends to show that the specific recurrence in man is always

attended with some degree of pyrexia, or, in other words, that spirillar blood-contamination always culminates in pyrexial perturbation, however brief.

Another topic requires notice here: thus, at many occult late relapses and at some of earlier date, I have found in blood-specimens prepared by the Albrecht-method and staining, minute filaments having a close resemblance to immature spirilla, so far as the latter may be imagined to exist (*vide* Chapter on the Blood): and I gained the impression that a partial development of the spirillum may attend a partial development of relapse-phenomena.

Deferred Relapse.—It is uncertain whether or not a latent or suppressed relapse, is ever followed by a recurrence of unmistakeable character; and more difficulties surround this subject than might be supposed, on account of the wide limits of apyretic intervals or known incubation-periods. Thus, as regards the first apyretic period, its mean duration being 7–8 days, 11 or 12 days have occasionally been witnessed in hospital to lapse before the first febrile recurrence (not to mention the longer time mentioned by a few patients), this interval allowing of re-infection from without and the conversion of a possible abortive into a relapsing attack. Nor is this merely imaginary, for I know that one spirillar infection does not preclude another even at early date; and in the medical wards contagion may be said to have been always possible. I have not, however, been able to establish such an event as now supposed, nor am I at present acquainted with a method by which its reality could be demonstrated. Respecting the later and longer apyretic periods, their mean duration being even more variable, the difficulties of proof are not lessened. The following case much interested me:—

CASE XXXVII.—F., 30, one of a group admitted with specific disease, previously had fever for a month (?); there was high pyrexia (t. $106^{\circ}\cdot8$, p. 132) and critical fall 4 days later: for 7 days no febrile disturbance whatever, then a brief rise to 100° , p. 74, then a decline for 6 days and a second rise, prolonged, low (max. t. $101^{\circ}\cdot8$, p. 80) and gradually declining, when she left hospital. The temperature was taken several times daily during these two perturbations and the dried blood submitted to scrutiny by the acetic acid method; it remains doubtful if the spirillum as commonly recognised was ever present, only filaments resembling it were noted about the acme of first rise, and beginning of the second more pronounced perturbation: more I cannot say, from not knowing the significance of appearances seen. It should be stated that the woman suffered severely, at first; and that the spleen was distinctly implicated in the later temperature movements.

Febrile paroxysms occurring as late as 2 or 3 weeks after the first crisis, with an intervening apyretic period quite undisturbed, could not be regarded as relapses proper, for they were always non-spirillar.

Spurious relapses.—In course of enquiry the spirillum fever was found to be attended with febrile paroxysms, not apparently due to local irritation or malarious influence, which, though periodic in date and in other respects resembling brief relapses, yet did not display the spirillum. They were seen in about 15 p.c. of all cases and commonly in place of third or second relapse when of 1 day's duration, and when lasting longer as second or first relapses. As seeming to be 'spurious,' *i.e.* non-specific *quoad* recognisable blood-contamination, they attracted particular attention: most occurred before I was aware of improved methods of blood-

scrutiny, and I must now regard such instances as only showing the practical difficulties once met with in correctly interpreting the clinical phenomena of spirillum fever. It is almost certain such paroxysms were not spurious; but in justice to observations made with care, I am bound to remark that the fully-grown blood-parasite may not represent the sole infective agent of this disease.

Other occasional febrile phenomena.—In practice, the first or second specific attack sometimes appears to be preceded by one or more mild, isolated paroxysms of uncertain character: that the spirillum may not then be found is only a conditional argument against the specific nature of such preliminary paroxysms, since it is often not to be seen in fresh blood at the beginning of an indubitable relapse, or even of a clear invasion: and the real question is, whether or not the blood-scrutiny has been an adequate one.

Also at the close of invasion and relapse, a more or less detached febrile paroxysm may take place, which is open to varied interpretation as either belonging to the specific attack or supervening like residual fever: here, too, much depends on the skill and patience of the observer, it being understood that once the spirillum has disappeared at acme or crisis, it never reappears until date of next recurrence.

Varieties of Apyretic intervals.—An acquaintance with this subject becomes useful, when patients do not come early under notice. Variations concern the length, general level and course of inter-febrile periods, all of which differ considerably: rallying after critical fall whilst usually prompt, may be either delayed or gradually effected; mid-course presents either a quasi-normal temperature or one somewhat depressed, a sustained elevation above normal being indicative of local complication; at the end, when relapse is near, either no visible change occurs, or a slight depression contemporary with first advent of the spirillum, or a slight rise. These remarks apply to the aspect of ordinary Charts.¹

Repeated Attacks.—Before comparative experiment had proved that one infection does not either preclude from or predispose to a second, on even early date, I had at hospital seen instances of repeated attacks amongst men. Occasionally new comers gave a history of such earlier illnesses, but the opportunity of witnessing successive fresh attacks was necessarily rare: how many repetitions may happen in the same subject, is unknown. Second infections may or may not differ from the first, in their general manifestation.

CASE XXXVIII.—M., 30, Mussulman immigrant, was admitted 2 July, 1877, during the invasion-attack, which after 6 days was followed by a marked relapse of 5 days' duration (spirillum so rare as not to be seen in the fresh blood), the succeeding apyretic period was normal for 12 entire days seen. Five weeks later, he was admitted at end of a smart fresh invasion-attack; and there was no relapse during 17 days after crisis.

¹ Necessarily I have consulted Wunderlich (Syd. Soc. Transl. 1871) as a recognised authority on Pyrexias, and also later authors; the result being a reliance on my own data. Respecting these apyretic intervals, Dr. Wunderlich observes 'let the course of temperature be what it will, there almost always occurs about the middle (of the first interval) a brief, sharply-pointed elevation of temperature . . . freedom from fever soon recurs and very often it is complete only after this episodal elevation; this hardly accords with experience at Bombay of uncomplicated cases, but in connection with secondary fever and local inflammation, interruptions of apyrexia become frequent.

CASE XXXIX.—M., 22, native medical pupil, one of eleven infected in the wards of the same hospital, was admitted 10 June, 1877, on first day of first attack ; a sharp rebound followed in 3-4 days after crisis (no spirillum), and 5 days later another febrile perturbation of undetermined character : 17 days afterwards he left hospital. Nine weeks later he was readmitted with a second attack also caught in the wards, and the invasion was about as pronounced as before : after 4 days there occurred a febrile movement of undetermined character (no spirillum and no apparent complication), for 15 days long & the temperature was nearly normal, there being noted only a slight rise on the 9th day—blood not examined. In this instance a certain similarity is noticeable in the charts of the successive attacks : the lad never suffered again, though still exposed to infection.

The first attack which I incurred in December 1877, was of the Relapsing kind and tolerably severe (spirillum in the marked recurrence so sparse as not to be visible to myself in fresh blood) : the next illness in February, 1880, was milder and of the abortive form : both events followed inoculation at autopsies ; and I may add that a Native lad co-infected on the last occasion, underwent an abortive attack resembling my own.

CHAPTER V.

COMPLICATIONS OF SPIRILLUM FEVER.

SECONDARY FEVER, AFFECTIONS OF THE NERVOUS, RESPIRATORY, CIRCULATING AND DIGESTIVE SYSTEMS, OF THE URINARY ORGANS ; ABORTION.

THESE comprise the more unusual phenomena associated with febrile attacks, and they may be said to consist essentially of (*a*) intensified ordinary symptoms and states—*e.g.* delirium, hiccup, hepatic, splenic and renal changes ; jaundice, diarrhoea, sudamina (see also Chapter III. for the three last-named) ; (*b*) local inflammations also more or less directly arising from the infection : *e.g.* meningitis, ophthalmitis ; bronchitis, pneumonia ; parotitis, pharyngitis, gastritis, enteritis, colitis ; nephritis. (*c*) accidents in the circulation ; as hæmorrhages, cutaneous, mucous, serous and parenchymatous ; thrombosis : abortion. And lastly (*d*) post-critical febrile reaction. Other examples imperfectly recognisable during life, are mentioned in the description of Anatomical Lesions.

Almost all complications date from the acme of fever or ensuing crisis, when general symptoms become most acute and abrupt blood-changes occur : they are commonest at close of invasion, and tend to reappear with relapse : their severity is very diverse, and some, as cerebral hæmorrhage and pneumonia, are common causes of death.

As every pronounced illness displays some peculiar features due to the predominance of one or more symptoms, it may be said that complications are always imminent ; and their discrimination from the more ordinary clinical phenomena, is often matter of judgment. Also indicative of their common origin, is the usual concurrence of two or more of these phenomena, it rarely happening that any complication exists alone ; and to the varying combinations occurring, are referable many remarkable types of the spirillar disease. I do not here allude to the congeries of symptoms known as the typhoid state, or to the general and special modifications of spirillum fever described below in Chapter VII.

Combinations.—For convenience, each complication must be considered apart ; but it is clinically important to recognise the chief conjunctions of phenomena, as follows :—Ordinary severe cases ; besides hepatic, gastric and splenic derangement, delirium, bronchitis and diarrhoea occur as a frequent group of milder complications. Jaundice, hiccup, broncho-pneumonia, dysentery and parotitis ; splenitis, with bronchitis and diarrhoea at relapse. In CASE X. are named symptoms often concurrent ; and in a Hindoo subject about the same date, I noted jaundice with hepatitis, delirium, petechiæ, sudamina and hiccup, form-

ing a nearly similar type of fever ; this fact pointing to a prevailing epidemic character during 1878. I would also remark that groups of cases may have peculiar aspects : thus amongst Ward-servants, some striking combinations of symptoms occurred—such as vomiting, epistaxis, jaundice, irregular distribution of heat, delirium with twitchings, cough, splenic uneasiness ; and in another case, bronchitis, marked *perturbatio critica*, albuminuria, delirium, jaundice and transient pneumonia. Native medical pupils showed epistaxis, hepatitis, melæna, vomiting, diarrhœa ; and vomiting, delirium, jaundice, hepatitis, petechiæ, cough, pleurisy and burning sensation in the feet. Unusual events noted amongst hospital patients seized in the wards did not materially differ from the above, except on the few occasions when their original ailment was acute enough to intrude its own symptoms : dysentery was, however, more frequent or marked. Cases with evident local inflammation, and the casualties, are not included in these synopses ; and to other parts of this work, I must refer for detailed illustrations of the principle under comment : details are many and varied.

The combinations of main symptoms furnish the *insignia* of disease, and upon them as much as on the predominance of one or two special signs, must rest both diagnosis and prognosis. Synthesis as well as analysis of bed-side data, is needful for accurate conception (*imago*) of a specific fever ; and a chief object of the memoranda detailed below, and elsewhere, is to aid in such conception as is intuitively formed in the mind of the observer, and becomes his idea of the spirillum disease.

For purpose of description, complications are arranged in physiological order, but I begin with one of general character and especial interest.

1. **Secondary Fever.**—Clinical experience at Bombay has necessitated the separate consideration of this subject. By the term Secondary fever (*synonyms* :—consecutive or residual fever, and reaction or rebound of temperature), is meant non-specific pyrexia often supervening at acme and crisis of Invasion, seldomer of first Relapse.¹ At first sight, such sequelar pyrexia appeared to be strictly 'idiopathic' ; but there is reason to connect it with marked blood-changes, and with parenchymatous lesion of the larger abdominal glands.

Like several other complications, it arises by many gradations from ordinary symptoms ; and it was seen at two periods, *viz.* at acme (*a*), as practically continuous with specific fever ; and (*b*) following the crisis, as a form of febrile reaction.

(*a.*) *Sub-acmal Pyrexia.*—In ordinary cases fever begins to subside immediately after the acme of attack, when the general symptoms already described are most acute ; but occasionally it persists for a time, entailing increased risk of life and often causing death. Fever thus prolonging uninterruptedly the pyrexial state, is associated with the advent of

¹ It was perhaps owing to my habit of constantly examining the blood, that the present discrimination arose ; possibly, too, the phenomena here described were commoner than usual at Bombay, yet on looking over the numerous temperature-charts furnished within the last 10 years by recent observers, I now perceive that a similar feature attends the spirillum fever of Europe, although it does not seem to have been alike interpreted. At an earlier date, Prof. Wunderlich evidently referred to it as forming an 'amphibolic stage' such as is not peculiar to relapsing fever. See Chapter on Pathology below.

remarkable blood changes : see Chapter on the Blood, Section III., and for clinical illustration, CASES XV. and XVI. Chapter III. of this Section. It is not irrelevant to point out, that the declining pyrexia at 'lysis' sometimes appears to be of this character.

Generally, such post-acmal pyrexia does not last for more than a few hours ; and since it is practically continuous with the specific fever of illness, not being distinguishable therefrom except by means of microscopical blood-examination, the discrimination here attempted may be regarded as a needless refinement. But believing as I do, every marked change in the aspect of the blood to be of clinical significance, the present distinction becomes both valid and useful ; for it is founded in reality, and is also of prognostic import, in so far that experience teaches when after disappearance of the blood-spirillum high pyrexia persists, the patient's life is in imminent danger.

Two cases were seen at the close of first relapse, which displayed a distinct sinking of temperature corresponding to critical decline, prior to the final rise, and may therefore be regarded as transitional to the next group. That last seen (M., 46) presented symptoms like those of acme ; and after death, I found the lungs permeated with serum and fine air-bubbles, spleen 20 ozs. and in it several infarcts, liver large and flabby, congestion (? extravasation) at lower end of ileum.

(b.) *Secondary fever in form of Rebound temperature after the crisis.* The date of reaction varies:—1. Immediate rebound. After the invasion-attack, especially, and in milder form, not uncommon ; see an instance in the chart of CASE X. : of more pronounced degree, the following is an illustration :—

CASE XL.—M., 19, famine-immigrant though in fair condition ; admitted on 5th day of invasion, e. t. 105°, p. 124, spirilla many, no peculiar symptoms, the liver and spleen not appearing much implicated ; some cough ; dysentery at the acme two days later, and critical fall on estimated 8th day of attack, e. t. 96°·2, p. 80, sweating profuse. The same evening chills with return of pyrexia, and next m. t. 104°·8, p. 116, skin dry, headache, pains in loins and legs, bowels quiet, liver and spleen unaffected, cough continues, second day after crisis, m. t. 102°·6, p. 112, hepatic dullness upwards not increased, but some fulness and tenderness in the right hypochondrium, tongue moist, two stools ; third day, m. t. 99°·4, p. 82, skin dry, some jaundice, three loose stools ; fourth day, slight increase of fever, m. t. 101°, p. 106, tongue moist, cough less, four stools, hepatic uneasiness not increased ; during the next three days fever rose and then declined, being remittent in type, and not ending with sweats : the hepatic soreness was not attended with distinct enlargement, bowels now quiet, and no other local lesion appeared in connection with this sequelar pyrexia : notes and blood-examination by Mr. S. A. *Vide* Chart No. 20, PLATE VI. ; it will be found to resemble, at early course, some charts of more indubitable complications—*e.g.* that of pneumonia No. 26.

The following cases are examples of immediate rebound and death:—

CASE XLI.—M., 38, medical subordinate, stout habit but healthy, contracted fever at an autopsy : the attack was tolerably pronounced ; bilious vomiting and cough were present, liver and spleen not much implicated, no epigastric tenderness ; 6th and last day, 7 A.M. t. 104°, p. 124, some delirium in the night, urine passed involuntarily, no vomiting, headache or distress, tongue dry and brown ; 9 A.M., the crisis has taken place, t. 97°, p. extremely feeble and intermitting, about 100 ; heart's sounds nearly inaudible, impulse absent : yet collapse not entire, pupils normal, eyes bright and indicative of his hearing and partly understanding what is said to him : there were constant tremors of the limbs and the hands fumbled at the hot water

bottles ; some small livid spots now appeared on the arms and legs, and front of chest ; an hour later, reaction had begun and at 11.30 A.M. the t. had risen 8° or to $105^{\circ}6$, p. very rapid and small, skin moist, the unconsciousness and tremors increased, the face became livid and death took place in $\frac{3}{4}$ hr. ; hence a period of five hours included acme, crisis and rebound, all these events being witnessed because the patient was under special notice ; under ordinary circumstances, the later phenomena might have been overlooked. *Vide* Chart 18, PLATE V.

CASE XLII.—M., 35, admitted for malarious fever, and a few days after recovery appeared specific fever, caught in the ward : the attack was pronounced and lasted 5 days ; blood-contamination abundant, general and local symptoms not peculiar. On fifth day, m. t. $104^{\circ}6$, p. 130, countenance anxious, skin moistened and unusually sensitive all over, pains of the joints, no fresh spots, abdomen not distended, bowels relaxed, tenderness in both hypochondria preventing sleep : e. t. $105^{\circ}4$, p. 120, tongue moist, skin dry. Last day, m. t. 96° , p. 80, crisis in the night with sweats, skin now dry, breathing quiet, a few fresh spots of eruption, no pains, depression ; spirillum disappeared : e. t. 105° , p. 150, moderate volume and soft, respirations 60 ; fever returned with chills about noon, no headache, has pains of the loins and knees, much thirst, skin dry, vomited twice, no particular distress, no fresh spots ; blood free from spirillum ; it is stated he remained in this state during the night, quietly sinking next morning under persistent fever. At autopsy. no jaundice ; head—a little slightly-turbid serum in arachnoid, with gelatinous streaks on dura mater, no increased vascularity, or petechiæ ; heart appeared healthy ; lower lobe of both lungs much engorged with blood ; liver 62 ozs., termed healthy, spleen 19 ozs., dark, firm ; kidneys healthy looking ; mucous membrane of stomach of pink hue, no extravasation, a few small petechiæ in the j. junum. It happened that an hour or two later, I inspected some parts, and found on cut surface of spleen the large pale areas indicative of 'infarcts'—exposure to the air bringing out their different aspect to rest of spleen-pulp. Since the engorged state of the lungs may have been due mainly to 'fever,' the splenic degeneration seemed significant of fatal blood-deterioration.—*Vide* Chart 19, PLATE V.

During my comparative experiments on the Monkey, this prompt febrile rebound was recognised in both mild and severe form, its resemblance to the phenomenon in man being very close : when most pronounced, it ended in death 3 times out of 4. The temperature attained usually exceeded that of the prior specific fever, as was the case in the human subject ; the type of fever was somewhat remitted, and its course could be better traced by the more frequent thermometer readings practicable in the monkey ; these showing a prompt decline before death, not, I think, always occurring in man. At 3 autopsies I found the mucous membrane of stomach and small intestines to be congested or inflamed ; the spleen enlarged, infants being not noted ; such coarser changes are not peculiar. See Appendix on Experimental Pathology, No 1, with Chart.

Commonly the secondary fever does not arise until from 12 to 48 hours after crisis, and may be deferred to the 3rd day or later ; it then varies in intensity, though still sometimes exceeding the spirillar pyrexia it follows, and its consequences are then seldom serious. This kind of reaction happened in about 10 per cent. of ordinary surviving cases, seen at all periods of the epidemic, amongst all races and castes, and after both abortive and relapsing attacks ; being sometimes noted after invasion alone, but commonest and most pronounced when following the fully-developed first relapse : 2nd and 3rd recurrences were rarely thus attended, or the fever might in them remain undistinguishable. Its onset was abrupt, course more or less paroxysmal, and the decline more or less prompt, but rarely sinking below the

normal level of body-heat : the pyrexia was acuminate or remitting, maximum 101° - 105° or higher ; duration from a few hours to several days. The attendant symptoms were those of pyrexia, with oppression or weakness ; local derangement might remain unnoticed, and when observed in the longer cases particularly, abdominal or chest symptoms seemed rather to follow the fever as consequences of it : splenic, hepatic or bronchitic uneasiness prolonged beyond the crisis, were not necessarily exacerbated at this time ; with unusual debility, a quasi-typinous state was occasionally seen. The pulse seldom quickened proportionately to rise of temperature ; but with diarrhoea, delirium, and signs of local irritation, actual or imminent, the pulse was apt to be more excited ; initiating chills, and at the end sweats, were often wanting. Secondary fever was commonly preceded by a pronounced specific attack, and the mean t. at crisis in 27 cases was $96^{\circ} \cdot 7$; but as the rebound followed also less marked crises and was absent in the deepest ones known, its occurrence was not in strict proportion to the prior state of depression. Some of the most striking examples were seen in young subjects, though not limited to such ; almost all patients were males ; the general condition was fair. The attendant visible blood-changes were neither notable nor uniform, the blood-spirillum invariably being absent ; and this negative character sufficiently distinguishes the fever from that of specific attacks.

Repetitions of the rebound were rare ; and the interpretation of most uncomplicated febrile events after 2nd true relapse being difficult, such are apt to be confounded with later recurrences.

CASE XLIII.—M., 30, Mussulman weaver (family affected) admitted at close of invasion, at the decline of which there followed a level first apyretic interval of 9 days. The relapse was very pronounced, duration 5 days, crisis with fall of $100^{\circ} \cdot 8$, but little sweating, there was depression yet not collapse, t. 95° , p. 78, small, weak : rallying did not begin till after 48 hours, when the progress of the case was as follows :—second interval, second m. t. $98^{\circ} \cdot 4$, p. 96, no headache, much pain in loins and staggering gait, slept, one stool ; urine free, 1015, no sediment, no albumen, chlorides $\frac{1}{8}$ vol. : e. t. 101° , p. 84, no fresh symptoms with this rise. Third day, m. t. $104^{\circ} \cdot 4$, p. 102 : no such distress as attended the specific fever, or sign of local complication except some diarrhoea, skin moist, heart's action very feeble, no eruption, he slept well, pains in loins and knees ; urine copious, chlorides $\frac{1}{8}$ vol. : e. t. $105^{\circ} \cdot 8$, p. 102, full, soft, no spleen or liver tenderness, no stool, tongue moist, skin supple, much thirst, little headache, pains and much debility. Fourth day, m. t. 104° , p. 120, no distress, headache slight, skin dry, sudamina have appeared on chest and abdomen (front), tongue coated, moist, indented at margins by the teeth, he slept, pupils normal, urine plentiful and clear, no implication of liver or spleen apparent ; e. t. $104^{\circ} \cdot 4$, p. 120, he lies quiet, some headache, much aching in loins and knees, and cannot get up from weakness : spleen now somewhat enlarged, not tender ; liver unchanged, no other apparent complication ; considerable thirst, tongue moist, skin moist, urine free, 1015, chlorides $\frac{1}{8}$ vol., no albumen. Fifth day, decline of fever, m. t. $100^{\circ} \cdot 4$, p. 90, no stool, abdomen rather tympanitic, no distress, tongue moist, fresh sudamina, sleep indifferent, some headache and pains ; urine acid, 1010, chlorides $\frac{1}{8}$ vol., slight cloudiness on addition of nitric acid (cold test) : e. t. 101° , p. 96, feeble, no sweats, he is rallying. Sixth day, m. t. $99^{\circ} \cdot 8$, p. 84, slept only in latter part of night, less headache, fresh sudamina, the pains continue : urine free, 1005, chlorides $\frac{1}{8}$ vol., slight indications of albumen by cold test ; e. t. 101° , p. 96 ; last day, m. t. 98° , p. 72, no headache, sweats on forehead, he slept, some dry cough, much pain in loins and weakness : urine as before ; e. t. $98^{\circ} \cdot 2$, p. 90, some sweating on forehead. The temp. declined further to 97° , p. 84, no fresh symptoms appeared, but the pains in the loins (muscular) became severer for a time ; the urine at once lost all traces of albumen : sciatica, diarrhoea, increased appetite, attended the ensuing convalescence.

The blood was continuously and carefully scrutinised with the negative results shown in the chart : white corpuscles were plentiful. *Vide* Chart 21, PLATE VI.

This man's daughter of 12, admitted just after him, displayed a striking intermittent rebound of 8 days' duration, which was attended with delirium, splenic and hepatic implication, and some bronchitis ; but, as with her father, no fresh complication or lasting lesion was present : the specific attack seen in hospital was probably a first relapse, like his : the child's mother had lately died of fever.

CASE XLIV.—M., 12, admitted 3 days before the close of a well-marked specific attack, which seemed to be first Relapse : liver and spleen implication not unusually marked and not persistent after the crisis ; 3rd day of post-critical apyrexia, m. t. $97^{\circ}8$, p. 86 ; the same night secondary fever set in sharply, and next e. t. $105^{\circ}4$, p. 140 (t. higher than ever before seen), yet no distress ; some constipation, headache, thirst, general aching pains ; 4th day, vomiting with slight enlargement again of liver and spleen ; then increased thirst, want of sleep, slight hepatic and splenic tenderness, indisposition for food, vomiting, deafness, tendency to diarrhoea ; on the 7th day, pyrexia, remittent all along, declined to 99° , p. 102, he was weak but less oppressed ; fever, however, at once returned, though less pronounced, remitting daily, and on the 9th morning t. declining to $98^{\circ}4$, with some sweats. During this period there were no fresh symptoms, but rather a subsidence of those previously existing : a second and final recurrence lasting 6 or 7 days, of nightly, ague-like paroxysms, now took place ; at the end subsidence to $97^{\circ}5$, p. 76 ; no symptoms of urgency : two subsidiary rises followed and 3 weeks' unchecked convalescence. No blood-spirillum throughout the first 15 days of this sequelar pyrexia, or at the last (Mr. S. A) : no obvious cause of the fever in any local lesion. *Vide* Chart 22, PLATE VI.

Several cases of anomalous fever seen after 7 or 10 days' illness outside hospital, doubtless had their correct explanation in examples like above, the symptoms present being best or only understood on such interpretation : often there was a history of association with relatives or friends, known to be affected with relapsing fever.

This subject is of great practical importance ; and hardly less interesting were instances like the following, which indicates the possibility of deferred febrile results of infection, and forms a link connecting such results with ordinary sequelæ.

CASE XLV.—M., 16, whose chart shows a well-defined febrile attack of negative blood-character, coming on in hospital 21 days after the crisis of demonstrated spirillum fever ; it lasted 9 days, being pronounced and fairly sustained. No local lesion was detected beyond some early splenic and hepatic fulness and tenderness, bowels rather relaxed, no pains, a general soreness of the body at first ; afterwards some numbness of the legs and feet, also deafness ; there was slight cough : final convalescence. The connection of this attack with the original spirillar infection may be dubious, yet I note that the youth continued to be low and delirious long after the specific crisis, or until 5 or 6 days before this new event, with which a similar kind of delirium at once returned.

Amongst complicated and fatal cases to be hereafter discussed, there were examples of prompt rebound attended with cerebral hæmorrhage, enteritis and, oftener, pneumonia. An instance before me shows that the pleuro-pneumonia and splenic enlargement found after death, did not probably supervene until ten days, at soonest, after secondary fever had set in. As even frequently, this consecutive pyrexia seemed to become the cause of such local signs as might appear, its occasional origin in a faulty state of the blood can hardly be doubtful.

Summary.—Non-spirillar fever immediately consecutive to the specific pyrexia of Invasion or Relapse, when sustained and prolonged (for even a few hours) was seen only in fatal cases.

It is probable some instances of defervescence by lysis were examples of this consecutive pyrexia, which was not sustained but declined gradually : they, too, were of serious import.

A decline of temperature resembling brief crisis, may intervene between specific acme and this secondary fever ; and CASE XLI. above quoted, indicates the likelihood of such intermediate event being more frequent than is stated.

Then follows the series of more distinctly separated and longer deferred secondary fever, which though not seldom prominent, were seldom known to prove fatal ; and, lastly, there are instances which might be regarded as sequelar.

Diagnosis of Secondary Fever.—This rests upon the history and previous known course of the case, on the absence of local symptoms and lesions at all proportionate with the promptness and height of the pyrexia ; and lastly, upon the absence of spirillar blood-contamination. I should add that the fact alone of such consecutive fever occurring in the lower animal—a true *fera naturæ*—is proof of its not being, in man, an accidental conjunction of a civilisation fever (such as typhus or enteric) ; and evidence is wanting that the monkey is liable to ordinary malarial infection.

Complications affecting the Nervous System.—These are important, because of the office of the structures involved ; and they are frequent, because of the unusual vascularity of the brain and its membranes.

2. **Delirium.**—The term is employed here to mean temporary mental derangement, attended with either excitement or depression of the frame. This symptom was noted in over 16 per cent. of observations made upon a series of 140 detailed cases seen throughout the epidemic, in both survivors and the dying ; it was associated solely with the febrile event, as either accompaniment or sequel. It was never remarked prior to the commencement of specific fever, or until the 4th or 5th day of the severer first attack, thenceforward daily becoming more common (mean frequency now 13 p. c.) ; at the acme and immediately ensuing critical fall, delirium was noted in 28 p. c. of cases ; and during the succeeding interval amongst survivors it became uncommon, being also limited to the first day or two after the crisis (about 5 p. c. of cases) : amongst the casualties at this last period (complications usually present), it was noted in 20 per cent. During the first relapse, and more so during later recurrences in which, too, the fever was much briefer, delirium was rarely seen, and then almost solely at the close of attack ; in cases fatal now, it appeared in 14 p. c. of instances. At all later non-specific periods this symptom was extremely rare, being present only with febrile complications. Generally, the first signs and milder degrees of delirium being liable to oversight, it is probable my notes should sometimes be both ante-dated and supplemented, especially as the entries were mostly made only twice a day ; and it is within my personal experience that transient delirium at the acme, was such as a hospital attendant might not regard.

Clinical forms.—For precision I would arrange these as follows :—

a. Febrile delirium: varieties—1. During high specific fever. 2. At secondary fever or the rebound. 3. With symptomatic pyrexia. 4. With the typhoid state at lytic decline of specific fever. *b. Non-febrile delirium* supervening during, at the end of, or shortly after the crisis of specific fever. It is to be understood that some of these varieties may, in the same case, be seen severally, or in chronological continuity during illness; and also that in successive attacks the delirium may not be of the same form.

Clinical characters.—*a.* I did not perceive any constant difference in cases where the fever was specific (*i.e.* attended by spirillar blood-contamination), and where it was non-specific or due to co-existent local inflammation: under all conditions the typhoid state was threatening, imminent, or actually present, when delirium became noticeable. The symptom here resembled that in other 'fevers'; it earliest appeared and was most marked at night, when pyrexia usually exacerbates: at first active in character it tended to become low and muttering: pure sleeplessness often preceded, and drowsiness or stupor might follow this cerebral manifestation. Usually, it promptly subsided with the fall; but might persist longer, especially if complications were present.

b. Post-febrile delirium is attendant on the depression which ensues during or soon after, the critical fall of spirillum fever: it is a remarkable phenomenon, in its more pronounced form coming on abruptly day or night, and resembling either a brief maniacal impulse, or a more sustained state of active and continuous delirium of 1, 2 or 3 days' duration: in both cases the predominating idea seemed to be fear, leading to corresponding aspect and acts; there was sleeplessness, involuntary evacuations and obstinate refusal of food and medicine: recovery usually ensued upon repose, and might be prompt and permanent. The milder forms were commonest (possibly not infrequent), displaying a tractable, chattering, busy delirium coincident with decline of temperature and pulse, and not lasting more than a few hours: about 10 p. c. of my last series of cases showed this kind of mental derangement at crisis, and usually at night-time, first or only.

Other details may be seen in the cases below; the association of headache with delirium was oftener than not recorded, their conjunction varying: the pulse may not be rapid with active febrile delirium, its relationship also being unfixed.

Febrile delirium was of highly variable degree in the same or similar cases, the active state being seldom pronounced or prolonged. That of collapse may be very violent (even furious) and sustained, as in mania; or restless, vagarious, sleepless, liable to sudden exacerbation; or no more than mere incoherence and restlessness: destruction of clothing, bedding, and seeming intolerance of interference or confinement (? due to terror) were common expressive acts; the general aspect and state being such as not witnessed in any 'fevers' in India, I am acquainted with.

Among the mildest and incipient degrees of delirium, may be mentioned the causeless sleeplessness or terrifying dreams occasionally named by patients (old as well as young).

Both forms were liable to remission (even intermission) and exacer-

bation: the first changing with the febrile state and general condition of the patient; the second altering less, with the less variable depression and increasing debility.

In neither form occurred any proper sequel, the febrile ending in recovery or death; the post-febrile in prompt recovery, or death (rarely), and lastly, in mania or mental imbecility more or less persistent.

Age.—Young persons or adults were the usual subjects: of 9 youths with mean age of 22, 4 had delirium, and of 13 persons with mean age 28, 6 showed this symptom, whilst of 14 cases of mean age over 30, 5 only had it: all these instances belonged to the hospital contagion series of 1877.

Sex.—Disproportionately oftenest in males.

General condition.—Febrile delirium was commonest in originally weak subjects, or those enfeebled by want and fever: the non-febrile form occurred in even robust males, yet no rule appeared here.

Type of disease.—Low or prolonged fever with lysis-tendency was especially attended with ordinary delirium; high and pronounced fever with marked crisis, was oftener the precedent of the maniacal form.

From my notes it seems that amongst Hindoo immigrants in 1877, maniacal excitement was commonest; whilst in 1878 and afterwards, among Mussulman weavers, the febrile delirium proper.

Correlated symptoms.—Febrile delirium in the sthenic seemed to bear some relationship to the intensity of pyrexia, in the asthenic to frequency and feebleness of pulse, with other signs of low febrile state.

The post-febrile form was seen only when the body-heat and pulse were greatly reduced, at and after the critical fall. I was, however, much impressed with its absence in some instances of extreme depression, as narrated elsewhere; and it holds no fixed relationship, when present, with degrees of bodily weakness. It is also to be noted that marked icteroid aspect, with even copious eruptions as of typhus, may not be attended with delirium, when 'fever' is mild, brief or uncomplicated. Severe and even fatal local complications may not be attended with delirium; once it was slight with meningitis in an old man. See also the cases below.

Diagnosis.—Febrile delirium occurring so early as the 5th, 6th or 7th day, even when complications are threatening, should lead to careful microscopic examination of the blood; the history and circumstances of the case also claiming attention: in itself, the delirium is not peculiar.

In Bombay I never saw post-febrile mania after 'agues' or 'remittents,' at all resembling that above described, as occurring in subjects liable to the contagion of famine-fever: it is true that in one instance of suicidal delirium, the blood had furnished negative data, yet to my mind the case was not clearly not one of this fever, for many circumstances had to be considered, which only a practical acquaintance with spirillum fever could, in absence of adequate blood-examinations, interpret aright.

The resemblance of specific febrile collapse to the algide stage of true cholera, was more apparent than real; one point of distinction being its occasional complication with a form of active delirium never seen in cholera, so far as known at Bombay.

Prognostic import.—Febrile delirium does not begin earlier in specific attacks which end fatally, either soon or later on; but it is decidedly commoner in such attacks; thus, amongst survivors the ratio showing delirium during the last 3 or 4 days of invasion being 13 or 14 p. c., it was 23 p. c. amongst fatal cases: all these patients being chiefly Hindoos seen in 1877 and '79. During 1878, however, among Mussulman weavers, febrile delirium was as frequent in survivors as in the dying, the general type of fever here being 'low'; hence the bad augury of this symptom will depend partly upon the general character and tendency of epidemic fever. Cases fatal in 1877 were usually attended with local complication (pneumonia oftenest), and this circumstance it is important to remember.

All 6 patients seen dying at end of first relapse had been delirious at close of invasion-attack; but only 3 of them showed delirium just before death; these died from exhaustion, the brain being found pale twice; 3 had no delirium in the relapse, who died from cerebral hæmorrhage, pneumonia and pericarditis: in these last the previous mental disturbance was slighter than in the others.

Notwithstanding the intensity of spirillum fever, its brief duration and tendency to complete subsidence render the prognosis of uncomplicated cases marked by delirium, less unfavourable than in continued fevers, or even remittents.

As a rule, post-febrile mania is even strikingly free from bad augury, due care being taken to prevent the patient injuring himself: some instances of quick rallying excited the most welcome surprise. When prolonged, however, it becomes a sign of organic degeneration, leading to final exhaustion.

Following delirium may be noted deafness, anæsthesia, or nerve-irritation evidenced by morbid sensations in the limbs; pains may be severe: yet all these symptoms come on also without preceding mental disturbance.

Illustrative cases.¹—These will be limited to instances of delirium either with co-existing spirillum fever, or immediately following it: the connection of this symptom with non-specific pyrexia, whether of reaction or symptomatic of local lesion, presenting no peculiarity of date and character. Febrile delirium:—

CASE XLVI.—M., 34, admitted on reputed 6th day of invasion, apparently at acme of attack, t. 104°·6, much enlargement and tenderness of liver and spleen, respiration hurried, but no lung complication detected; much exhaustion, and low, muttering delirium; a marked critical fall ensued on the same night, with little sweating; delirium subsided, headache and depression persisting 24 hours longer; rallying slow,

¹ Two sad instances met with prior to recognition of the blood-spirillum and therefore not included in the text, were the following: they happened within three days of each other.—M., 25, admitted with high fever of 4 days' duration, on 7th morning the critical fall of 6°·6 to 97°·6, much depression, pupils contracted, slight delirium the previous evening and at crisis; an eruption of pink spots (compared to typhoid spots); second morning after fall, t. 98°·4, no sleep after midnight and some headache; at about 9 A.M. he went to the latrine and passing by a staircase reached the hospital verandah, and threw himself over, being killed on the spot. M., 40, admitted at the close of fever, much depressed; temp. next morning 97°·6 (lysis decline), some pink spots on trunk; he was lying quietly on his cot at 8½ A.M. when I passed, and a few minutes later suddenly got up, ran to the verandah and threw himself over, meeting instant death. I had all fever-patients at once removed to the ground-floor wards, for this experience was peculiar; it confirmed my opinion that famine-fever was present in Bombay.

no eruption. After 7 days a well marked relapse occurred, lasting 5 days and ending with a great fall of temperature, the depression at its end continuing 48 hours; in the last 12 hours of this time delirium came on at night, the man wandering about, with no memory of places, t. 97° - 8° , p. 76-86, and respirations declining to 15 p. min.: this mental debility slowly subsided in 2 days, and convalescence was fair: a second relapse followed, with further rises of temperature, but no morbid delirium.

CASE XLVII.—M., 25, admitted on reputed 12th day of invasion. depressed; distress and dyspnoea, t. 102° .4, p. 110, respirations 70. there was active but tractable delirium in the night: at 4 P.M. the *perturbatio critica* with rigors during which his cot shook, crisis in the night, and next morning only some drowsiness left. At the close of apyretic interval and succeeding pronounced relapse, there was again a distinct critical exacerbation, during which time he became delirious and semi-conscious, being very low and in much distress (I suspected clots in the heart), yet these symptoms all passed away with the crisis. in a few hours.

Like the last, this case strongly indicated the connection of delirium with the peculiar systemic disturbance occurring at close of spirillum fever, when blood-changes occur.

CASE XLVIII.—M., 15, admitted on 4th day of invasion with high fever; next day there was active delirium increasing at night, eyes not injected, no headache; 10 gr. doses of Chloral and Potass. Brom. had no effect; with decline of t. prior to the acme, delirium ceased and there were sweats; it returned with the acme and persisted with the crisis, until near its end, although chloral in larger doses was given. The lad soon rallied, and 8 days after a pronounced relapse of 4 days' duration set in; there was again mental disturbance and excitement at midnight, during the acme, ceasing with the great fall of temperature. Another lad of 15, showed violent delirium at the close and rather gradual decline of specific fever, which was uncontrolled by morphia injections: so two young men of 18 and 22, who had pulmonic congestion at the same time, entire relief ensuing with crisis: and so a man of 25, in the course of whose single attack, epistaxis repeatedly occurred, on 7th morning t. 102° , p. 130, dicrotic; depression, restlessness, delirium, body hot, limbs cold and convulsively twitching; at 3 P.M. sweats, with crisis and prompt relief.

The morbid anatomy of febrile delirium is, as yet, but imperfectly made out; brain-lesions of coarser aspect are sometimes wanting altogether, or are slight (*vide* CASE XIII.) after death with febrile delirium; and with much lesion, functional disturbance may not be active.

CASES.—F., 23, was admitted in a state of active delirium, moaning, semi-conscious; t. 105° .6, p. 140, full and firm; became quite insensible and died in 24 hours. There was cerebral hæmorrhage (arachnoidal), with lobular congestion of the lungs.

M., 32, admitted near acme, when t. 105° , p. 120, eyes injected, headache, skin dry; epistaxis followed and next morning t. 100° , p. 100, small and feeble, no headache; delirium in the night, with continued decline of temp. next day, t. 98° , p. 100, feeble; when he became unconscious, restless and dyspnoic, dying shortly. There were hæmorrhagic petechiæ in the heart, lungs not collapsed, brain pale.

M., 14, admitted near end of invasion, being prostrate and delirious; pupils dilated and sluggish, urine passed in bed, death in 5 hours. The brain was pale, wet and soft.

Typhoid state in connection with spirillar infection.—M., 30, brought to hospital with a history of previous attack: is depressed, incoherent, at times semi-conscious and passing into the state of coma-vigil: eyes sunken, sordes on lips and teeth, tongue dry, brown, red at sides and tip; no abnormal sounds in chest: tenderness over the liver, spleen and r. iliac region: evacuations passed involuntarily, the stools semi-consistent, t. 100° - 101° , rising on the 3rd and last morning to 103° .6 when he became unconscious and died. The blood then contained swarms of large spirilla, many large pale cells, some of which were vacuolated; there were also large endothelial cells with fatty granules

in them. Cerebral meningeal hæmorrhage, petechiæ elsewhere and congestion of lower end of ileum, peculiar changes in liver and spleen; the heart and lungs unchanged.

Mental perturbation first appearing with critical subsidence of fever, may be brief and limited to the beginning of crisis (of which several examples are before me), or it might come on with, or last until, the end of decline; and, finally, sometimes it supervened still later.

CASE XLIX.—F., 40, admitted at close of invasion-attack, crisis the following night with a decline of $10^{\circ} \cdot 4$ F.; on first day of fall, m. t. $93^{\circ} \cdot 6$, p. 68; continued decline on second day to 93° (estimated), p. 72; she had now become incoherent and restless, and on the next night was very turbulent: on third day a slight rise to 96° , p. 74, delirium persisting; on fourth day t. $94^{\circ} \cdot 6$ to $95^{\circ} \cdot 4$, p. 72; on fifth day t. $96^{\circ} \cdot 2$, p. 82, she had slept until 2 A.M., the delirium then reappearing; on the following day the temp. rose to 97° , p. 80, and there had not been further disturbance. During this time Bromide Potass. and Chloral Hydr. had been freely given, with stimulants local and general.

CASE L.—M., 25. For upwards of 10 days had high fever outside hospital which left him with profuse sweating, and so weak that on admission 3 days later, he could not stand; some jaundice and several pink spots on the body: he then began to improve. Four days after entering hospital, the relapse set in with chills; it was pronounced (t. on three days 105° , p. 130-140) and lasted 6 days: fresh spots appeared: on the last day there was a slight remission prior to a. exacerbation, vomiting, no delirium: in the following night crisis occurred and delirium came on, with a decline of $8^{\circ} \cdot 8$, a. d. pulsations 44. First day of fall m. t. 96° , p. 96, much exhaustion, skin clammy, tongue pale, shrunk, tremulous, moist; pupils sluggish, of normal dimensions; stools passed in bed, urine retained; no fresh eruption: 'he lies in a peculiar condition of collapse with tremors and mental derangement, mouth open, eyes bright; is now quiet, but during the night when the fever left (*i.e.* after 9 P.M.) he was sleepless, restless and attempting to run away; respirations 30, rather gasping; he cannot speak above a whisper, and he picks at his teeth, as if feeling something there' (MS. notes on case). Urine drawn by catheter 20 ozs., 1011, high-coloured, slightly acid, clear and no sediment on standing: after a few hours' rest in conical vessel, there were no particles visible under microscope except a few epithelial scales and bacteria. Stimulants given. Vesp. t. 95° , p. 110, very feeble and small; skin moist, has rallied a little though temp. lower (pulse not slower); delirium less, and he takes food; much thirst, no urine passed: catheter ordered. Second day—m. t. 96° , p. 108, very small; was excited during the night running about the ward: now still prostrate, pallid, but not sinking into the bed; pupils normal, skin dry, sordes on the teeth, tongue dryish, and not protruded fully, evacuations passed in bed, no eruption; the liver and spleen rather full and tender. Urine drawn—sp. gr. 1011, acid, high-coloured, clouded, but not depositing sediment, chlorides reduced to $\frac{1}{6}$, bile-pigment much, albumen none by heat and acid, slight traces (?) by acid alone: after standing, under microscope, some epithelial, mucus, and blood corpuscles (due to friction of catheter), many small bacteria moving and quiescent, some large, clouded or granular cells, nucleated, tint yellowish, nature uncertain, some granular masses like zooglæa; nothing more. Vesp. t. 95° , p. 120, skin clammy, he is restless, getting up and trying to run away, tears bed-clothes and chatters incoherently, pupils of normal aspect, slight injection of conjunctiva, no sensible heat of head, expression of face vacant and brows contracted: pulse and heart's systole extremely feeble, he cannot stand erect: sordes, shallow breathing, involuntary evacuation of urine, slight œdema of feet: petechial spots have appeared on abdomen, r. shoulder, l. chest, bright red, slightly raised, and not quite effaced on pressure. Chloral in addition ordered. Third day—m. t. 98° , p. 114, small, soft, flickering: somewhat less delirious, but still talks indistinctly and incoherently; skin soft, dry; tongue brown, but inclined to be moist; abdomen distended, some tenderness over spleen, none over the liver; pupils normal, eyes slightly congested, craves for water; no stool, urine passed in bed and drawn off; he takes nourishment, slept until 11 P.M., after then lay awake but quieter than before; still can hardly stand: fresh spots on the wrists, none on back or lower limbs, those of yesterday are fainter, less raised and not affected by pressure. Urine—

sp. gr. 1015, acid, chlorides a trace, bile acids and pigment present, no albumen or sugar: no sediment. Vesp.—t. 99°, p. 114, less feeble, still mutters to himself but understands when spoken to: takes food and medicine; tongue becoming moist, passed urine voluntarily. Next day—m. t. 98°·4, p. 102, fair volume, slept without the sedative, appears dull but is more rational, urine free: after this date his recovery was fairly progressive. no return of fever, the œdema of the feet had not subsided in the fortnight he remained in hospital. This case received particular attention, and was regarded as showing that mental disturbance is not necessarily due to visible urinary derangement.

Delirium may not supervene until after the crisis has passed and the minimum temperature recorded.

CASE LI.—M., 40, also Mussulman, a weak subject, on admission gave a history of invasion-attack, with a first relapse then present; 3 days afterwards the critical fall to 94° F. (estimated), p. 60, with extreme depression, no urine for 24 hours, then found to be clear, 1022, free from albumen; pupils rather contracted, intellect clear: a few pink spots: he then rallied promptly and completely as usual. On 5th day of second apyretic interval intermitting fever appeared, ushering in the second relapse (no spirillum), and on 7th day continuous pyrexia began (spirillum present) which lasted 4 days, ending with a *perturbatio critica* (t. 104°·2) and followed with a decline next m.; t. then 94° (estimate), p. 102: sweating occurred, and a few fresh spots appeared; vesp. t. 94°·4, p. 92, some headache. Second day of fall—m. t. 94°, p. 108; slept, still some headache and pains; vesp.—t. 96°·5, p. 72, much headache, with pains in loins and knees: at 7 P.M. he became delirious and ran away from the ward, was brought back and placed in a side room. Third day—m. t. 98°, p. 90, said to have slept after being locked up, is now (7 A.M.) silent and morose, but when approached shouts and become excited as if from fear: very weak, pupils somewhat dilated, no injection of eyes or heat of scalp: vesp.—sweating; is in the same maniacal state, refusing food and medicine, and not replying to questions, mutters incoherently to himself, and resists the least attempt at control, becoming very violent: Potass. Brom. and Chloral were administered. Fourth day—has slept and is rational, t. 98°·5, p. 88 (sitting), pupils normal, some deafness, still headache and pains in loins: urine also normal, 1010, chlorides $\frac{1}{2}$ volume, no albumen present; from this date he rallied quickly and completely, the only complaints for a time being slight headache, buzzing in the ears, deafness and debility: in 10 days he was convalescent, and for a longer period had no return of fever.

CASE LII.—F., 30, admitted 5th day of invasion-attack, which was pronounced and marked with daily febrile exacerbations and remissions: crisis on 9th day was prompt but not sudden or extreme; e. t. 95°·2, p. 88, resp. 26 (in m. 22 only), skin dry, tongue coated white, no headache or delirium, or thirst or pains, is very weak, much abdominal tenderness, spleen going down, much cough, sputum bronchitic, stools natural. First day of apyretic interval, m. t. 98°·2, p. 80, resp. 24, skin dry, tongue white and dry, no headache, thirst, no appetite, bowels costive, no pains anywhere, spleen felt but not tender; was delirious at night, but now seems tranquil: vesp.—t. 98°, p. 80, resp. 24, skin dry, bowels moved; wanders in talk and manner, occasionally becomes excited and has to be placed in confinement. Next day, she had slept after Chloral, and the mental state was quieter; t. and p. natural: next day slept without Chloral, now had pains in all the limbs but no headache, the delirium henceforward ceased.

CASE LIII.—M., 28, admitted on 5th day of invasion, symptoms pronounced, marked crisis on 7th morning, t. 96°·4, p. 92, much sweating last night, no headache and great relief to the symptoms generally; vesp.—t. 101°·6, p. 108, a rebound of fever preceded by chills at 3 P.M., no headache, much thirst, hepatic pain gone, spleen still enlarged and painful on coughing; no spirillum in the blood. First day of apyretic interval: the secondary fever has subsided, m. t. 97°, p. 88, no headache, slight thirst slight cough, no pains, spleen tender still, no sweats: vesp.—t. 97°·4, p. 66. Second day after crisis: m. t. 97°·8, p. 88, no headache or thirst, tongue moist and coated at the back, no cough or pain in chest, no pains in limbs, spleen tender; bowels moved; he became delirious last night at 10, and did not sleep; now exhibits a busy, active state, picking at the bed clothes and getting up, though he is tractable; pupils rather dilated, a watery discharge from the eyes, pulse weak, small and

irregular, systole feeble: the spleen is tender on firm pressure only. Vesp. t. 96° , p. 74, skin moist, he slept after Chloral and Digitalis. Next day there was still some depression, t. $97^{\circ}6$, p. 70, but no further delirium: the splenic enlargement was probably old.

The full morbid anatomy of this form of delirium has yet to be revealed.

CASE LIV.—M., 40, admitted towards close of invasion which subsided by lysis to t. $97^{\circ}2$, p. 88, there were pink spots, cough, hiccup, and no other unusual symptoms: day after completed fall, t. $97^{\circ}6$, p. 88, little change noted; second day—t. still $97^{\circ}6$, p. 102, and now some delirium with restlessness, respirations 24 shallow, hæmoptysis (scanty) and moist sounds only heard on auscultation of chest: death. Brain congealed only: pneumonia of part of one lung: characteristic changes in liver, spleen and kidney. Here delirium at fall was associated with latent pneumonia; and there is reason to suppose similar complication to be not unusual, with both febrile and post-febrile mental disturbance: see also some of the cases above.

3. **Mental Hebetude, Mania, Dementia.**—It was not unusual after specific infection to see a certain degree of mental feebleness, not explicable by the general debility alone; various prior conditions doubtless contributing to this result, but not always in apparent connection with head symptoms. One case (M., 22, after first relapse) displayed with mental depression and feebleness, marked frontal headache preventing sleep, a feeble circulation and tendency to diarrhoea not controlled by treatment. Another instance of prolonged low delirium tending to become chronic and indicating a connection with more than functional disturbance, was the following:—M., 16, before and after crisis (? of relapse) delirium was the most prominent symptom, in diminished degree it persisted for seventeen days, with temp. and pulse nearly at normal; at first deafness (not of extrinsic origin), numbness of legs and toes and a burning sensation in the palms and soles, which lasted for a few days only: on twenty-first day after crisis, a smart and sustained febrile attack, apparently due to secondary fever, return of delirium: convalescence slow. There were many spirilla in the blood on his admission. Some further stages of changes possibly due to a common lesion, are illustrated by the instances following.

CASE LV.—M., 30, a weak subject, admitted at end of invasion probably terminating by lysis (many spirilla with t. $99^{\circ}2$): next day, t. 97° , p. 80, restless delirium came on in the night, with depression, pupils active, evacuations involuntary; e. t. 100° , p. 103, a slight rebound of fever, no enlargement of liver or spleen. First day of interval, t. 99° , p. 100, no sleep, delirium at night, tremors, eyes injected, the typhoid state threatens: second day, indifferent sleep, no delirium in morning, still debility; so third and fourth days, the same low state with incoherency and but slight evening febrile exacerbations (not of pulse); so till the 11th day after first fall, m. t. $98^{\circ}6$, p. 110 (quickened now), slept, took food, scalp said to be warm, is dull and listless; vesp. t. $101^{\circ}8$, p. 120—the relapse beginning. Second day—m. t. $99^{\circ}4$, p. 120, slept, is more alert than yesterday (the fever seeming to act as a stimulus): vesp. t. $103^{\circ}4$, p. 120, feeble and irritable, no sleep: third day—A. M. t. 104° , p. 150, skin moist, slept a little (sedatives given), eyes injected, head hot, much depression, sordes about the mouth; is again chattering and delirious: urine free: vesp. t. $103^{\circ}2$, p. 154, sweats, no stools, takes food, spleen enlarged and tender: delirium active—many spirilla in the blood. Fall—m. t. $97^{\circ}6$, p. barely perceptible; collapse present: vesp. t. $100^{\circ}2$, p. 128, copious sweating, a rebound of fever. The second apyretic stage now followed, lasting 8 days till death: temp. level, commonly below normal (extremes $98^{\circ}4$ and $96^{\circ}4$), pulse above normal (92 to 108) and very feeble: his mental state was that of depression, amounting even to dementia, and ending in

unconsciousness ; his bodily condition prostrate and emaciating day by day. At the autopsy—some opacity of arachnoid, pale and collapsed lungs, pallid heart, disseminated fatty patches in the liver, spleen unaltered, so the kidneys ; congestion of the cæcum.

CASE LVI.—The following case was regarded as one of brain lesion after spirillum fever : family history good : no bad habits. M., 18, brought in a very weak state with a history of 15 days' fever, delirium having come on during its course : t. $103^{\circ}6$, p. 130, feeble, organs in chest and abdomen apparently normal ; there is a state of passive delirium, with rational intervals when the patient understands what is said, pointing to the forehead as seat of pain. I found the blood-plasma clouded, fibrillation indistinct, free protoplasm in clumps, several active blood-spirilla : also in the sputum a spirillum sluggish and a little larger, with other parasitic growths ; some salivary corpuscles contained dividing nuclei and swarms of mobile granules. The fever declined by lysis on the 3rd day ; t. $96^{\circ}5$, p. 100, and thenceforward the temp. rising to normal fluctuated but little for the 12 weeks he stayed in hospital : the pulse declined after crisis, as usual, and remained slow and weak. The lad's mental condition after improving a little, soon passed into that of imbecility ; pupils normal, hearing good, so sleep and appetite ; but taciturnity and inattention to calls of nature, with much emaciation and profound anæmia : final prospects of the worst.

4. **Deafness, Noises in the Ear, Earache.**—None of these symptoms were common, or appeared of special significance. Deafness was noted chiefly at the critical fall, and oftenest at the end of first relapse (one-seventh of cases), then attending other signs of exhaustion and feebleness of the circulation : it seldom lasted longer than two or three days : an illustration is given in Case No. IX. It also sometimes came on at the acme of fever, with heaviness of the head and tendency to drowsiness : and it was occasionally noted in connection with typhus-like symptoms amongst the Mussulman weavers, yet not often or in proportion to the apparent cerebral exhaustion or oppression ; in such instances it might be one-sided, and persist for some time. It was not most frequent in the fatal cases. The symptom was also noted later after crisis, in connection with increasing debility, giddiness, slow-pulse, exhaustion after delirium and secondary fever. It was sometimes present during the complication of sore throat, tonsillitis and parotitis. Extreme or permanent deafness was not seen ; and from this summary the usual conditions of its presence will be evident : deafness due to treatment by the cinchona alkaloids is here excluded.

Tinnitus aurium ; buzzing in the ears was, in all cases, associated with weak circulations and functional derangements of the other special senses.

Otitis and otorrhœa were noted in a few women and children, and had probably existed prior to febrile attack : in a lad of 8, left earache was a premonitory sign of the brief relapse, not persisting : in a woman of 25 admitted at close of invasion, there was a peculiar nodding, tremulous movement of the head in possible connection with pain and then watery discharge from the left ear ; after crisis a slight febrile movement in apparent connection with the ear symptom ; and with a striking paroxysmal relapse, brief return of the tremors ; convalescence was slow. Earache has been known to come on after a second relapse, together with boils and pustules on the legs.

5. **Numbness and Soreness of Hands and Feet.**—The subjective sensations complained of by patients, pertained rarely to diminished sensibility of the soles and palms, but rather to exalted or perverted feeling—

variously defined as tingling, formication, pins-and-needles, or commonest of all, a burning sensation, much increased on pressure being applied to the skin.

Perhaps 3-4 per cent. of subjects were thus affected, or more, since modified hyperæsthesia sometimes long eluded notice, becoming evident only when the feet were accidentally placed to the ground, or the hands made to grasp firmly : it was seldom noted in the fatal cases, being usually a phenomenon sequelar to fever. Perverted sensations were rarely detected (if ever) during invasion-attack, seldom earlier than a week after first crisis (or about the time of first relapse), and 9-12 days after second crisis (about the date of second relapse) : when a febrile recurrence occurred they were neither suspended nor increased thereby ; and in the absence of a distinct first or second relapse, they still might be well marked : they have been noted towards the close of both apyretic periods, in the same subject.

Their intensity seemed to vary much ; patients sometimes complaining bitterly of the discomfort, loss of sleep, and crippling entailed : no exact data, however, are available, and I am unable to estimate the variety of common sensation, most or oftenest affected : numbness was indicated seldom at beginning, and never as a sequel of the hyperæsthesia, but precise information is needed here. Duration—usually a few days only ; sometimes 3 weeks, or as long as the patient could be induced to stay in hospital : site—the soles of the feet always ; often (perhaps generally) the palms of the hands also, either first or for a briefer period, and seldom so severely as the feet, the hands always recovering first : it is probable the hyperæsthesia extended to the digits, sometimes the dorsum, and also included deeper-seated parts ; some patients distinctly naming pains shooting upwards, along nerve-trunks (?) : no visible or temperature changes were remarked in the parts. The morbid sensations were always increased on pressure or movement, and usually increased for a time before subsiding, a diurnal exacerbation after noon being noted, and more or less distinct remission towards early morning.

The majority of 12 cases analysed were young men : paupers ; all the subjects were weakly, the circulation especially being feeble and heart's action sometimes irregular : cases were seen at all periods of the epidemic, and after fever mild or (oftener) pronounced, abortive or relapsing. Other complications were rarely present ; the state of the blood not visibly peculiar : signs of contemporary defective nerve-action were usual, such as pains in the limbs then or previously, giddiness, swimming or flashes before the eyes, *muscæ*, *tinnitus aurium*, neuralgic pains in the head ; dilated pupils.

Diagnosis.—Suspicion of some connection with the cerebro-spinal centres, was not verified, the nerve-lesion seeming evidently transitory ; though patients sometimes felt the ground as 'velvety,' yet they distinguished the shape of objects with closed eyes, and could walk fairly : in two cases I noted the gait was striking and peculiar ; for the men not daring to put the whole sole to the ground, walked on their heels, as if the ground were on fire.

Prognosis.—Was not unfavourable, so far as known : but the final result in some severer examples could not be witnessed.

The anatomical characters and intimate cause of these phenomena being unknown, their real value can be only surmised : inference from

common site (oftenest the lower limbs) and observed occasional direct nervous connection, pointed to analogy with the characteristic febrile and post-febrile 'pains': a peculiarity was the frequent occurrence of these perverted sensations about the time of either latent or expected relapse, first and second.

Other details are mentioned in the following illustrative memoranda:—

M., 40, weak subject, œdema of the feet, soreness of palms and soles on admission, near close of first (or second) attack, and persisting; that in hands ceased after crisis in a few days; crippled by the foot-soreness, treading on a pebble would throw him down; no starting of the limbs or pain in loins: ineffectual treatment by ergot, arsenic, strychnia: the heel gait for the 3 weeks he stayed in hospital.

M., 17, very weak, severe relapsing attack, and on 12th day of second post-febrile period œdema of feet, starting of the limbs, soreness of the feet, especially, which could not be put to the ground without pain, and then also involuntary contractions of the toes, augmenting the suffering: this experiment showed local hyperæsthesia, at least, and I thought there might be increased irritability of the spinal centres: the whole foot was complained of: there had been very severe aches of the usual kind before this came on: much relief in course of a fortnight from local use of aconite and administration of strychnia.

M., 35, weak; burning sensation came on during the relapse (blood full of very active spirilla), it also affected the eyeballs; the ordinary pains were very severe: defervesence gradual, and delirium supervened: day after crisis, deafness and numbness of the head, aching pains traceable along back of lower limbs (probably sciatic nerves); the same evening burning of the soles came on (and of palms also): next day dysentery.

M., 22, numbness is named as preceding the soreness: hemicrania followed 8 days after the first relapse, and it would seem also increased sensitiveness of some mucous surfaces, the urethra being the seat of burning when urine passed, and the mouth when food was introduced: diarrhoea was present: the heart's action was feeble and irregular, sometimes intermitting; no relief from nerve tonics.

A general soreness of the surface of the body has been noted during specific fever; the most striking instances I recollect, occurring in two patients who died at close of the attack, one of cerebral hæmorrhage and the other, at rebound after crisis.

Local Palsy.—Weakness and wasting of the deltoid muscle, has sometimes been noted. Instances were those of a stalwart man, whose right deltoid became enfeebled 4 days after a first relapse; the injury did promise to be permanent: once the left muscle was affected, pain in the shoulder referred to the back of the joint and increasing towards night came on 10 days after an attack of fever supposed to be the first, a brief relapse followed; relief was afforded by counter-irritation and strychnia. A patient with a history of relapsing fever, had the right deltoid so wasted as to render the arm almost useless; only slight relief ensued on treatment. It is probable other muscles are liable to similar implication.

6. Cerebral Hæmorrhage.—The frequency of this serious lesion in all its degrees is not precisely known; because whilst the larger extravasations could not be overlooked, the minuter, or incipient, forms need for their detection closer search than was usually practised.

In 54 autopsies there were 8 examples of copious hæmorrhage ($\frac{1}{6}$ of the whole) which are described below; and, besides, in several other autopsies of pauper 'fever' subjects admitted in a moribund state, similar lesion was found; these instances, though generally concordant with the rest, being unverified by the microscope, are here excluded.

1. M., æt. 29, died 4th day (?) of first attack. Brain surface pallid above, venous congestion behind, with subarachnoid serous distension: hæmorrhage (scarlet blood) into the cavity of the arachnoid, the clot compressed in a thin layer over the posterior lobes as far as the fossæ at the base of skull, its margin in front being well defined opposite the coronal suture; there were scantier clots also in the anterior and middle fossæ. Brain-substance everywhere firm and healthy; no internal congestion or unusual effusion: cranial nerves (including the *vagi*) unaltered; so vessels and membranes, there being no sign of cerebral inflammation or embolism. Pupils much contracted, restless delirium, rigors or spasms, and partial insensibility some hours before death; last noted temp. 101° , probably rising; p. 112. Spirilla in the blood.

2. F., 23, d. 9th day of invasion. Great congestion of scalp: effusion of fluid blood into cavity of arachnoid, coagulating immediately on withdrawal; also along the course of middle cerebral arteries, beneath the arachnoid, in the Rolandian fissures. Intense congestion of veins at vertex of brain: brain-substance firm and dry, puncta vacuolosa many; no internal hæmorrhage, vena Galeni congested: autopsy 2 hours after death. Previously restless delirium and deepening insensibility, moaning without stertor: last noted t. $105^{\circ}\cdot6$, skin dry; p. 140, full and firm. Large granule-cells in the blood. Pronounced jaundice.

3. M., 35, died at critical fall of invasion, a few hours after admission; t. 97° , p. imperceptible. Vascular turgescence and slight opacity of membranes; reddish serum in arachnoid; subarachnoid effusion of blood opposite left parietal eminence and at base of middle and posterior lobes of brain, most on right side. No laceration of brain-substance, which everywhere seemed healthy. The typhoid state and restlessness prior to death: pupils contracted. Spirilla in the blood.

4. M., 55, d. 5th day of first apyretic interval, pyæmia (?) having set in: there was hæmorrhage with suppuration beneath the arachnoid and clots in brain-substance: purulent foci in one lung.

5. M., 30, d. 5th day (? acme) of first relapse. Scalp much congested, and so pericranium and endocranium: diffuse meningeal hæmorrhage over lateral and inferior surfaces of right hemisphere chiefly, in the pia-mater and traceable between the convolutions along the middle cerebral artery. Elsewhere brain and membranes healthy: the arterial circle at base of brain was everywhere pervious. The temp. rose to $103^{\circ}\cdot6$ before death, and the spirillum then disappeared.

6. F., 20, dying after acme of relapse, suddenly. No congestion of scalp or brain membranes; a large clot filled the vault over right hemisphere, reaching below to middle fossa and both posterior fossæ, and in front nearly to the anterior fossa; it was situated in the arachnoidal cavity, being somewhat adherent to the parietal dura-mater and above covered by a thin fibrinous layer towards the serous cavity, but not below where a fresher effusion of brighter red colour was seen, the aspect of the whole was recent, though not equally so. Left hemisphere alone intact. Brain-substance everywhere firm and healthy: *velum interpositum* pallid, ventricles empty: cranial nerves unaffected. Spinal cord healthy throughout, only a thin clot at upper part (for 3 inches) in direct continuation with that in the posterior cranial fossæ. There was an old injury to the skull on the left side unconnected, to all appearance, with this hæmorrhage. Temp. $106^{\circ}\cdot2$, and p. 140 the previous evening, falling next morning with decease, and the blood then became non-spirillar.

7. M., 30, death at critical fall of relapse. Much dark grumous fluid blood was found in the sac of the arachnoid, chiefly on the left side: brain congested, substance firm; no sign of inflammation or of arterial obstruction beyond a small pale nodule in one of the posterior communicating branches, not obstructing its channel. Pons and medulla healthy. Last temp. noted 95° , p. imperceptible: there was hæmorrhagic pericarditis; endocardium seemingly free: deep jaundice and intestinal blood-stasis. No spirillum at last, but previously abundant.

8. F., 35, d. at undetermined stage of fever, moribund on admission, t. $101^{\circ}\cdot6$. Congestion of membranes: a thin layer of blood in the subarachnoid space, and in the sac over the upper surface of both hemispheres; no serous effusion; brain-substance moist; a small quantity of tinged serum in the lateral ventricles. Deep jaundice and many spirilla in the blood.

Remarks on the above series.—The amount of blood extravasated may have varied from 2 to 8 ounces: its site was always outside and mainly at the vertex, of the hemispheres; 5 times on both sides, 2 on

right chiefly and 1 on the left, the effusion doubtless spreading or gravitating where least resistance : it was usually found both under and within the arachnoid, and since it might be present in the visceral subarachnoid space alone, it must have been poured out in the meshes of the pia-mater, afterwards forcing its way by laceration into the serous sac. The rarity of large extravasations within the brain-substance is noteworthy, only one instance being seen and that accompanied by inflammatory softening (*vide* No. 4 above). As to source of the blood, whilst various cerebral vessels at the base might be implicated, it was oftener possible to trace the effusion alongside the middle cerebral artery (here of the right side), the vertically ascending branches of which upon reaching their highest point at the upper surface of the hemispheres and there changing their direction to the transverse or descending, whilst comparatively unsupported in the loose meshes of the pia-mater, seemed to be specially liable to impaction and rupture ; and inasmuch as both larger trunks and minute interior vessels commonly escaped, it may be that those of a certain calibre or direction alone were predisposed to injury. Either only one effusion was indicated, or if there were several, one so large as clearly to account for the sudden death common to these cases.

The aspect of the extravasated blood was usually bright as if arterial ; once it was darker when suppuration co-existed ; generally coagulated once the blood was still fluid, and as autopsies were made as soon as practicable after death (mean interval 3 hours) sufficient time may not usually have elapsed for separation of the blood-fibrine.

Additional particulars are furnished in the summaries of cases above, and such as relate to the blood itself are mentioned in Chapter I., Sec. III.

Symptoms.—During fever, cerebral compression from the clot always caused insensibility more or less complete ; yet even when established, the coma was not of the usual apoplectic character. In one typical instance the state was thus described—3 hours after sudden onset of insensibility and 2 before death : she lies supine and tranquil, breathing 50 per minute and very shallow ; no stertor ; no convulsions or rigidity of limbs, or spasm of the calm features ; sphincters relaxed ; pupils dilated, fixed and equal ; temp. declining, now $102^{\circ}\cdot6$, p. 144, full and bounding. (*Vide* Case No. 6, above.)

In other instances unconsciousness was less active, and rather resembled that of shock with partial retention of irritability, as evinced on attempting to force open the mouth, or on firm pressure over the liver, spleen or epigastrium ; audible moaning was then a common symptom ; the pupils might be much contracted, even when hæmorrhage had been considerable, the abdomen tympanitic and generally tender, the breathing hurried, the body-heat above natural (mean 103°) and pulse quick and small (mean 120) ; twice the critical fall was probably complete before death, mean t. 96° , pulse 100 and feeble, or imperceptible.

Special symptoms noted were lachrymation, ecchymosis of the conjunctiva, limited to one side ; alleged diarrhœa.

At first the insensibility might not be continuous, alternating with delirium, and the state has also been compared with coma-vigil : eventually deep coma or exhaustion supervened and decease in a few hours or not for a day or more, according to the amount and rapidity of bleeding. It would seem that copious hæmorrhage, once and finally, was the rule ;

yet there were also signs of small effusions repeated at intervals, the brain symptoms being obscure or not perceived until by cumulation or a larger efflux, or superinduced inflammation, the lesion became insupportable. Five of the eight cases under analysis being admitted after attack, the premonitory signs of hæmorrhage were imperfectly learnt; in three of the five delirium was noted at first, but not in the instances (better protected and nourished) seen in hospital throughout; nor was headache present in the latter, and the coma is said to have come on suddenly in the night. Twitchings of the limbs was reported twice prior to attack, but once was possibly confounded with rigors of the *perturbatio critica*: particular injection of the eyeball or flushing of the face was seldom noted, and epistaxis not once. Symptoms truly antecedent were not ascertained.

The date of hæmorrhage was always about the termination of specific fever: 3 times in 7 this pyrexia belonged to a relapse (probably the first) and it may have been oftener so; on such occasions the invasion-attack ended by lysis; the recurrent pyrexia was pronounced or prolonged, yet not peculiar. At the time of cerebral lesion the typhoid state had sometimes supervened, but not necessarily; in 3 of 8 cases there was pronounced jaundice: in 7 the spleen was found to be large, infarcted 1, softened 5; it was small and exsanguine 1: the liver was termed normal 4, large and pale 4; the kidneys congested, mottled (fatty) or spotted with hæmorrhage, never granular; the state of the urine was ascertained too seldom, but evidence of uræmia might certainly be wanting, and so of heart-implication (once only pericarditis): the lungs showed lobular congestion or apoplexy twice, inflammatory nodules once, oftener a collapsed state: there were concurrent hæmorrhagic petechiæ in the sub-mucous intestinal surface 5 times, subserous (abdominal) 2, substernal 1, and doubtless elsewhere unnoted: 1 was a case of hæmorrhagic pericarditis and enteritis: scurvy was rare.

The diagnosis of this complication is determined by the rules applicable to all similar lesions; more particular stress being laid upon the rapid onset of cerebral compression, the speedily deepening coma, and the previous absence of head symptoms or marked toxæmia: the contracted state of the pupils, when present, would indicate surface cerebral irritation; the character of the coma and its attendant symptoms, are also noteworthy. When the hæmorrhagic effusion is small, the symptoms of compression may be almost replaced by those of irritation; and in milder degree, these could hardly be distinguished from the effects of toxæmia.

When meningitis supervenes on the hæmorrhage, diagnosis may be very difficult: spontaneous meningitis was, however, extremely rare in spirillum fever; and the remarkable limitation as to date (with attendant blood-changes) in cases of hæmorrhage, should be borne in mind, as well as the frequency of lysis in the preceding or actual febrile attack. The correspondence of symptoms generally to those of acme stage of fever is noteworthy, and upon these the apoplexy supervenes as if by unforeseen accident.

Prognosis: highly unfavourable, no instance of recovery being known; and, I may add, no example of old arachnoidal effusion being found amongst the numerous autopsies of fever cases made during the epidemic

period. This statement does not, however, exclude the probability of rare sequela symptoms (*e.g.* of mental or motor derangement) in survivors, being due to minute vascular lesion of the cerebral surface.

Instances of minute cerebral hæmorrhage are the following :—

1. F., æt. 30 (healthy hospital matron, infected in her ward), minute spots over left parietal and right occipital convolutions and also in Sylvian fissure of r. side : pia-mater congested and loose, convolutions pale, wet and shrunken, slight opacity of arachnoid at base and some turbid serum : blood in cerebral arteries.

2. M., æt. 30, congestion of membrane and slight opacity of arachnoid, effusion of turbid serum and rather softened brain-substance ; minute extravasations in some of the convolutions at the vertex and a *Filaria* more stunted than that of the blood found in one specimen (seemingly not accidental).

3. M., æt. 34, brain pale and wet, spots of punctate hæmorrhage in convolutions along the two sides of the superior longitudinal fissure : no hæmorrhage in the membranes, subarachnoid fluid at base pinkish and arteries there distended : copious black vomit prior to death.

4. M., æt. 23, congestion on surface, arteries at the base empty, but ramifications of the middle cerebral distended, substance of brain pale and softened ; in the crus cerebri above the locus niger a hæmorrhagic spot as large as a pea.

5. M., æt. 24, congestion on surface with tinged serous effusion : on left side subarachnoid hæmorrhage on under surface of posterior lobe ; none in Sylvian fissure : in the substance of the right superior peduncle of the cerebellum hæmorrhagic specks : convolutions shrunken and easily unfolded, brain-substance wet and flabby.

6. M., æt. 25, minute extravasations beneath the dura-mater at the base of the skull ; some congestion of membranes and clear serous effusions : convolutions pale, wet, soft and shrunken.

Remarks.—The above are but samples of a lesion, which doubtless was frequent in the large typhus-like class to which the cases belonged. The cerebral extravasations were small or minute, often numerous and scattered ; their seat not only in pia-mater on the superficies, but also, or alone, within brain-substance, grey or white, when others like them may have been overlooked, in the same or similar cases. They were clearly due to rupture of small blood-vessels (*vide* Chapter on Morbid Histology).

The spirillar-infection was at all times abundantly manifest ; external and internal petechiæ, or other blood-effusions were co-present ; death was never attributable to these minor lesions, yet it always occurred at the time when larger hæmorrhage was common, namely during the later days of the febrile attack, or immediately after its close ; no patient in this series survived until the relapse. Mean age of subjects 27 years ; one female ; general condition variable, and instances commonest amongst Mussulmans at later periods of the epidemic.

Symptoms likely to follow these lesions could seldom be precisely dated, or dissociated from the typhoid or exhausted state supervening at the last : in 3 at least there were no head-symptoms beyond the drowsiness and delirium, not uncommon in severe cases of spirillum fever ; in 2 the patients became unconscious and restless a few hours before death, with hurried breathing (40 to 50 per minute and no stertor) features pallid, pupils once of normal size, once equally contracted ; and in 1 case carefully watched (No. 1 above) head-symptoms were noted in the following order :—vomiting after the 3rd day, restless and delirious after the 5th, pupils contracted on 6th, petechial spots in skin, screaming, dilated and fixed pupils, abdomen tympanitic, eyes heavy and conjunctivæ injected ;

head not hot on the 7th day : on 8th or last day, ecchymosis in upper half of ocular conjunctiva, right side, pupils much contracted, spasms of face and limbs, quick breathing (50, shallow, jerky), feeble action of heart, partial unconsciousness only ; probably coma at the end. When death in this series took place at the acme of fever, the mean temp. was $104^{\circ}2$, p. 125 : it happened twice during the fall.

From the above summary it seems that whilst the cerebral lesion may not have been concerned in the production of prominent symptoms, its possible occurrence is worthy of recollection, since when present it must add to the dangers of the case or interfere with complete recovery : its effects will depend upon the site as well as degree : the pupils were normal twice (Nos. 2 and 3), finally more or less contracted thrice, state unknown once (No. 4), their state therefore being no positive guide to diagnosis of the injury. There was jaundice 5 times, 2 of deep hue (*typhus biliosus*) ; the general character of the symptoms approached to typhus in early prostration, feeble heart, eruption ; and, as regards the cerebrum, in the pale and flabby aspect of the convolutions : a similar state being found in other cases when petechiæ were present elsewhere than in the brain. The local changes in other organs were not peculiar, but like those seen in all severe examples of spirillum fever.

7. Epistaxis.—Is rarer than might be expected considering the abruptness and force of the pyrexial changes, the frequently impoverished state of the blood, and the manifest troubles of the circulation : on the other hand, I note that epistaxis rarely occurs in agues, that the fever-patients were not usually scorbutic, and that circulation difficulties might be looked for on the arterial side of the capillaries.

Bleeding from the anterior nares of a venous character was noted in 5 or 6 p. c. of survivors, and in about 3 p. c. of casualties : it may have occurred somewhat more frequently. It was commonest during first or invasion-attacks (7.5 per cent.), less common in relapses (2.75 p. c.), and seen only once with complication (pleuro-pneumonia) during the first interval of specific attacks : of 12 selected instances, epistaxis was noted once on third day of invasion, 6 times at or near the close, and twice at critical fall ; once in the succeeding non-specific interval ; and twice during first relapse from third to fifth day : it occurred rather oftener during the day than at night. The bleeding was probably always venous, coming by oozing or quick welling from one or both nostrils, and soon setting : its estimated amount each time was a few drops to 6 or 8 ounces ; its recurrence not usual or more than two or three times in the whole day, upon, at most, as many successive days : its source doubtless was the sub-mucous venous plexuses of the nares. Epistaxis was usually seen in young male adults, once in a child of $2\frac{1}{2}$ years, once (in 12) in a woman of 30 yrs. : the subjects were the strong or weak, residents or immigrants, Hindoo or Mussulman ; none were particularly scorbutic. Instances were seen throughout the epidemic, yet oftenest amongst the ill-fed weavers with fever of a low type ; the specific pyrexia was usually pronounced.

It did not appear that epistaxis was necessarily associated with particular head, chest or abdominal symptoms : only severe headache was noted in more than half the cases, and sometimes it was distinctly

relieved by the hæmorrhage: delirium was present with or soon after the bleeding in 5 of 12 cases, as often it was noted that the eyes (conjunctivæ) were injected or unusually suffused, or the seat of burning sensation, before or with the flux: thirst was sometimes excessive. It is noteworthy that epistaxis happened generally just before or at the acme of attack; if coming on previously, it ceased then; and more rarely was it seen during the *perturbatio critica* or actual crisis: the congestion of collapse did not lead to nasal hæmorrhage: urgent vomiting was noted in some cases only. Epistaxis was not present during secondary fever, and once it attended a pulmonary complication after crisis (3rd day of 1st interval): jaundice of unusual intensity was never present: splenic or hepatic enlargement not unusually great.

The diagnostic value of this symptom is but slight, yet Remittents and other common pyrexia were very rarely accompanied by bleeding from the nose. As to its prognostic import the occurrence of epistaxis with marked perturbations of temperature, would point to the final stage or acme of fever being at hand; though the cases were pronounced, yet the fatality being little beyond the mean, this symptom cannot be regarded as of bad augury. The bleeding either ceased spontaneously or was readily checked by cold; the tampon was used but once in 12 cases. Anatomical conditions—not ascertained: the attendant physiological state was doubtless excessive vascular turgescence of the Schneiderian membrane; the anastomotic connection with veins of the cerebrum or orbit, would account for occasional modification of head-symptoms with epistaxis. Involuntary swallowing of the blood, with its subsequent reappearance, was borne in mind. As to conjunction with other passive hæmorrhages, nasal bleeding was rarely associated with skin petechiæ, never with hæmatemesis or hæmoptysis, and only once in this series with ecchymosis of the conjunctiva. In one of two fatal cases it took place at the acme of invasion, lasting four hours; death at the crisis, and at autopsy small hæmorrhagic spots on the heart and not elsewhere. It would be easy to speculate on the significance of this symptom in particular cases, but when all instances are compared, it does not appear to pertain to a common state of the system, seeming rather incidental in its occurrence: an illustrative case is the following:—

CASE LVII.—M., 25, hospital servant in good condition, an abortive attack pronounced but uncomplicated; seen throughout; temp. moderate, pulse not unusually quick till the end, then losing strength, headache and ocular congestion not excessive: repeated and rather copious bleeding from fourth to last day, but not at acme: Warburg's Tincture and Digitalis freely given: convalescence uninterrupted: eruption not noted. More significant, however, were instances like the following; and especially CASE LXXIX. below, which was overlooked in the above analysis. M., 27, had copious epistaxis at close of invasion outside hospital: on 3rd and 4th day of a well-marked relapse there was very free bleeding, just before and after a febrile exacerbation which either was itself, or foreshadowed, the acme, when the state of the blood undergoes a change: much headache before, not after, though the head was hot and temp. still high: no injection of eyes, or delirium: scanty eruption after crisis.

8. Inflammation and Ecchymosis of Conjunctiva: Keratitis: Inflammation of the deeper Tissues of the Eyeball.—Coming near to these more palpable lesions, were seen also excessive lachrymation after crisis; injection of the conjunctiva of a dull, passive hue; altered condition of

the pupils, usually dilatation, sometimes contraction, of undefined significance ; and the subjective sensations and functional impairments of ocular vertigo, dark vision, flashes, *muscæ*, night-blindness, diplopia, and oscillation of the globes : together with these symptoms, debility predominating.

A sense of burning in the eyeballs has been noted at the height of fever, and also as a post-critical phenomenon ; in conjunction with head-symptoms and nerve-irritation.

Conjunctivitis.—Not uncommon and not always indicative of deeper-seated lesion, as would appear.

Ecchymosis of ocular conjunctiva.—Was noted in about 6 per cent. of fatal cases and may have overlooked sometimes ; in three examples selected, the site was right eyeball twice—once in a woman the upper half, occurring at seeming acme just before death ; at autopsy there being found small specks of cerebral hæmorrhage in both hemispheres and many petechiæ in various parts of the body : in the other case the right eyelid also was ecchymosed, probably about similar date of invasion, and after death copious cerebral hæmorrhage was seen chiefly at base on r. side. The third instance was that of a lad (hospital servant), ecchymosis occurring at acme of severe primary illness, followed by double pneumonia, pharyngitis and death in 6 days : autopsy not available.

That this symptom is contingent, like most other localised attendants, on impeded circulation, is shown by the following memoranda :—A young apothecary at my hospital caught relapsing fever, and at the first crisis both conjunctivæ became blood-shot, purple spots also coming out on the skin ; with first and second relapses (unusually pronounced) the ecchymoses were not increased or repeated, but their absorption was gradual, traces remaining after a month. A native lad also infected in hospital, after the first fall showed large deep red patches in both eyes, which were not augmented at the severe relapse (with greater febrile changes than at invasion), and in course of absorption displayed a yellow tinge somewhat resembling jaundice. A woman was admitted at undetermined stage of fever in a very low condition and displaying ecchymosis on the right side only : she was removed before the end could be known. So far as appeared, the cause of these extravasations was not excessive vomiting, or like muscular effort.

Corneal affections.—These being chronic and sequelar, were seldom seen in hospital : ulceration was noted in connection with irido-choroiditis at late post-febrile periods : and there is reason to suppose defective corneal nutrition may have been not uncommon after this fever, in conjunction with general emaciation.

Deep-seated inflammation of the Eyeball.—Post-febrile ophthalmia amongst survivors was probably not very rare ; near 5 per cent. of all cases may have been affected ; notes of several instances are before me, occurring at all periods of the epidemic and all in adult males. The right eye was commonly implicated, and in this series oftenest a few days after the invasion-attack ; occasionally after the first relapse : subjects usually, but not invariably, much debilitated.

Selected instances of ophthalmitis were the following :—

CASE LVIII.—M., 25, suffered severely during invasion and relapse; 2 days after second crisis a bed sore threatened over the sacrum, next day he complained of much pain in the left eye, which had become affected with iritis; under treatment mitigation ensued, but an exacerbation 5 days later, opacity of both corneæ (deepest on the left side) ensuing; again slow amelioration interrupted by a brief second relapse; ultimate result unknown: the spleen had been latterly much affected in this case.

CASE LIX.—M., 25, admitted at end of invasion, the blood-plasma then clear, fibrillation distinct, some free protoplasm present and numerous active spirilla: crisis very pronounced and rallying gradual: 10 days after, diffused pain, redness, lachrymation, intolerance of light in left eye, hazy cornea, pupil rather contracted, iris unchanged in aspect: mercury (with opium) was administered to salivation, and relief in 3 days ensued with seeming subsidence a week later; then the ophthalmia returned, and was similarly treated; discharged convalescent 9 days later: quinine had been given during the interval. The ophthalmoscopic appearances were as follows—a shallow ulcer on the cornea, no deep-seated changes, and the dimness of vision complained of is accounted for by the artificially dilated state of the pupil.

CASE LX.—M., 50, European, about 13 days after an abortive attack, the right eye became inflamed, vision being attended with distinctly granular cloudiness: the ophthalmoscope revealed only changes in the vitreous body, which very slowly cleared away: no other sign of relapse; much general debility. The left eye was slightly implicated, in so far that vision was impaired by muscæ: it had been habitually less strained than the right.

9. **Skin Eruptions**—In the brown skin of Native patients, occupying wards screened from sunlight, red spots were seldom prominent and, I doubt not, were often overlooked. At the better lighted G. T. Hospital, I found them quite at the beginning of the epidemic, and continuously afterwards, though not constantly. The commoner eruption consisted of minute pink (rose) spots, raised, readily effaced, and either fading forthwith, or changing into purplish, more persistent stains. Occasionally true petechiæ began at once: more rarely a diffused mottling was perceptible, and at times vibices.

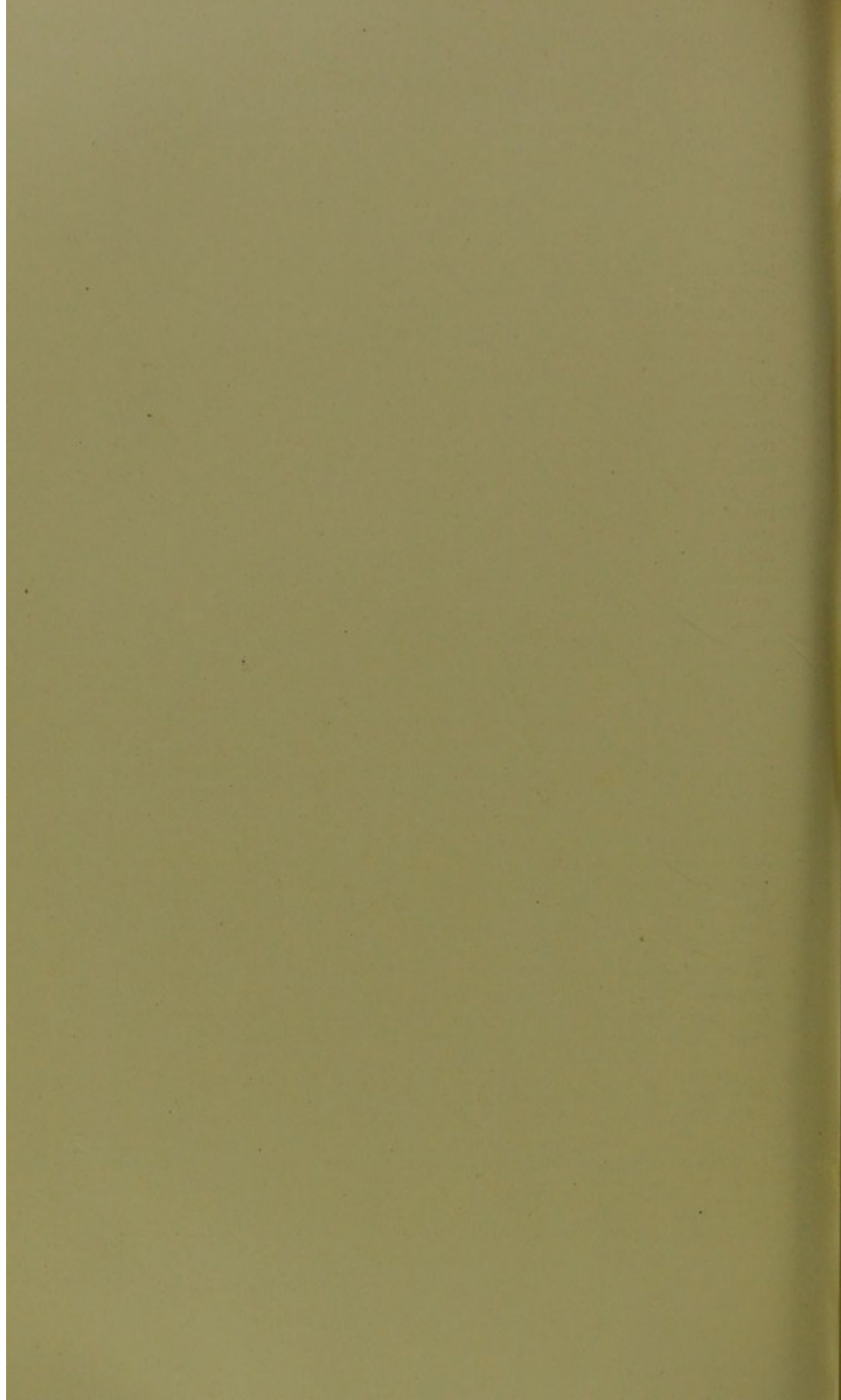
The pink spots were noted in 10 per cent. of later cases, and were possibly commoner earlier in the epidemic; some groups of subjects showed them oftener than others; thus, families, patients from one house, or otherwise connected, as the 9 vernacular students who caught disease in hospital during 1877, of whom 5 displayed distinct spots. This form occurs in crops with the fever, and may continue to appear longer: it is equally frequent in first attacks and recurrences, being seen in both events, or in either alone; it is rarely visible so soon as 2nd or 3rd day (5 p. c. of cases), usually not till the acme of attack (12 p. c.), or at the critical fall (20 per cent.); and 1, 2 or 3 days after the crisis, it may persist, or even then first be found: the spots seem to come out at night, being generally noticed in the morning. Site—earliest or chief on the front (infra-clavicular region) and sides of the chest (below axilla, or at line of costal cartilage synarthrosis); then on front of abdomen, front and inner side of forearms and arms, less often on lower limbs; on the back they were often present; seldom above on the neck or face; exceptionally at root of neck, sides of sternum, wrists. On or near old sites fresh spots sometimes re-appeared. Aspect.—At the beginning some difference may be noted; commonly they resembled small rose-tinted papules, quite effaced on pressure, without central dot, rounded, undefined; quickly appearing and fading in 2 or 3 days; or changing to the next more persistent form. Frequently, the earliest aspect was



H. V. C. Homberg, 1877.

West, Newman & Co. ch. 10

THE ERUPTION OF SPIRILLUM FEVER.



that of minute red blotches or stains, of breadth varying from a pin's head to a split pea, hardly raised, or if so flattened, shape circular or irregular, edges defined, not effaced on pressure; apt to come out abruptly in clusters; sometimes fading speedily, oftener turning dark purple, more visible, and lasting several days: the transition from these spots to the petechial was not rare. The total numbers varied from half-a-dozen, to two or three dozens or commonly more: the successive crops were few and irregular; both forms may concur; the more persistent lasting from one attack until or beyond the next recurrence, when new spots appear: I have detected the still fading maculæ 3 weeks after their outbreak, and sometimes they became raised before disappearing. It happens the spots were not noted in infants; at all other ages they were seen, and in both sexes: they were not attendant especially on cachexia, poverty, scurvy; or on typhus-like attacks (altogether) or the ordinary mild or severe: nor were they connected with special clinical symptoms, or state of blood or urine: other hæmorrhages might be present without these spots during life, or found after death. Morbid anatomy.—The pink papules disappear with disease: the purplish stains remain more or less apparent, and on division of the skin show a dark speck in the cutis, limited above by the pupillary layer, in other directions fading gradually, sometimes reaching the subjacent connective tissue. Translucent sections display one or more foci of vascular congestion or stasis, with exuded blood or colouring matter around: the presence or character of any embolus was not ascertained in my interrupted examinations, yet it seemed clear that the changes were all of one kind, from the pink spot to purple blotch or true petechia. Diagnostic and prognostic import.—This eruption was first noted in connection with spirillum-fever during 1876-7, yet as it eventually proved not to be peculiar to the new disease, its significance becomes modified: in fatal cases of specific fever, the spots were only slightly more frequent than usual in those dying during invasion-attack, and less so in those dying later; of 12 instances dying with first relapse, none showed them. Though not diagnostic, yet when occurring in fever possibly specific, they should engage attention: thus one of the vernacular students in 1877 with pyrexia at first remittent, had the blood examined with negative results; but upon seeing a few purplish flattened spots, I tested the blood myself and found in it several active spirilla; these were not again detected two days later, when the lad was at the point of death; yet the diagnosis was valid. A hospital nurse caught the disease in the same ward, and at the same date as this student clerk; she, too, had a few spots, and also died: in neither of these cases was the temperature-chart characteristic of spirillum-fever as commonly conceived of.

CASE LXI.—M., 22, vernacular student, on 7th day of invasion-attack showed some pink spots on the chest and forearms, some of which faded four days later and others were still visible after seven days as dark, very slight elevations: with the relapse fresh spots were detected in 2, 3 and 5 days, on chest, shoulders and arms, front and back; the ones first noticed had now a measles-like aspect and purplish tint: the patient was not in a typhous state and rallied quickly: there was some diarrhœa afterwards, seemingly quite independent of the eruption.

CASE LXII.—M., 20, vernacular student, displayed a large ecchymosis on lower half of both eyeballs, sub-conjunctival, nearly symmetrical and simultaneously appearing on day after crisis of first attack: no skin eruption and the ecchymosed blood

was gradually absorbed in the course of twelve days, leaving a spurious jaundiced aspect: on 2nd day of the relapse a few pink spots made their appearance on the chest; there was no return of the conjunctival hæmorrhage: symptoms marked, but not typhus-like or extremely severe. I have seen another instance of ocular ecchymosis with spots on the trunk simultaneously.

CASE LXIII.—M., passed through a marked first attack, and on 2nd day after crisis a crop of pink spots was seen on the chest, which disappeared on pressure; next day, a few others some of which were of brief duration (3 days), but others on the shoulders and back became persistent, being slightly raised, of purplish hue, and lasting till the relapse and ensuing fall: during this recurrent attack fresh spots were not seen: fever of low type.

CASE LXIV.—M., 33, on 8th day of invasion-attack there were a few small pink spots on the chest and abdomen, which increased in depth of tint next day and were not wholly effaced on pressure; they had not the dark aspect of confirmed petechiæ, yet some of them lasted for upwards of three weeks, the patient meanwhile undergoing an attack of suppurating parotitis, which supervened 8 days after the crisis (*i.e.* about the time of relapse), and on that date there was an outbreak of fresh spots. A weak subject, whose wife had the fever, and whose child died of it.

CASE LXV.—A man, 35, showed a few pink spots on the chest at close of first attack, not effaced on pressure and gradually fading in the course of four or five days: 6 days after the first crisis, there was headache and sweating with a very slight rise of temperature ($99^{\circ}\cdot 2$) about then and a distinct outbreak of pink spots on the front of chest and abdomen, which did not fade for three days.

CASE LXVI.—Eruption in a case of Typhus icterodes.—M., 35, Muss. weaver, from Lucknow, in Bombay a fortnight, admitted from a factory sending other cases to hospital: Jan. 1880. Fever of 7 days' duration; t. $102^{\circ}\cdot 2$, p. 128, deep jaundice and much debility—numerous red spots in 1 ft infra-clavicular fossa, level, purplish, size and form irregular (some being as large as a pea), clustered and ineffaceable on pressure: a few on the back and arms: on the back also some large hyperæmic patches: no mosquito bites: many spirilla in the blood. Acme and crisis within the next 24 hours; with the fall the skin becoming rather pallid, the blotches were more distinct, some now resembling true petechiæ; a few fresh spots on the chest: red patches on the back gone. With a slight rebound was much depression, with hiccup (? petechiæ in stomach), and also a copious eruption of new purplish spots, varied, papular: next day decline of temp., some more fresh spots; there was sore throat with vascularity, and apparently purple discolorations on the fauces and tonsils. In the ensuing apyretic interval, all the eruptions began to fade, and after 3 days the older purple blotches had nearly disappeared (to my surprise), the later pink spots now become purplish and puffed (not inflamed) as if from irritation of the extravasated blood, or some new local growth: axillary glands not enlarged or tender. The man rallied quickly; a brief intercalated paroxysm now occurred: and probably a few fresh spots came out: after a second apyretic interval another pronounced relapse took place, at the crisis of which, in the place of ordinary eruption, I noted some minute specks of cutaneous hæmorrhage in the upper intercostal spaces close to the sternum, where the intercostal arteries terminated by dividing and then come forward: the patient rallied remarkably, and 14 days after his admission the eruptions had quite gone. His son occupied the next bed, and with his specific attack, also showed the eruption, but without jaundice and typhus-aspect. *Vide* Chart No 10, Plate II., and the coloured illustration.

It appears to me that these spots partake of a common origin and character: the lighter-tinted papular form being due to slight or temporary blood-stasis in the derma, whilst the less transient is a real blood-stain, due to actual extravasation; and obviously an inter-transition is possible here, as well as termination in true petechiæ. In general aspect, their resemblance is mostly to the eruption of *typhus exanthematicus*; but otherwise to that of *typhus abdominalis*, as regards scanty numbers and advent in successive crops: so that, supposing the ordinary distinction of English typhus and enteric eruptions to be insisted on (a

point to myself seeming hardly tenable), then these spots would be regarded as partaking of a mixed character.

Having detected a precisely similar eruption in non-specific fever of the remittent (typho-remittent) character, and also in pneumonic and hectic (?), at the same epoch in Bombay, I consider the pathological significance of this clinical sign may be more extensive than is yet recognised.

It is necessary to state that the appearances above described may be closely simulated by the bites of the common small mosquito, or, less palpably, of the ordinary flea: thus, the mosquito-bite may not show a central prick, and in vascular cutis of the face and thinner cutis at the wrist, it may be attended with defined blood-stain or extravasation: its duration, too, varies like that of the spots, the nocturnal advent of which further favoured this idea of their occasional origin from the insect pest of some wards.

On the other hand, these incidental punctures are more regular in form and size, found only upon exposed parts, are very seldom clustered; and do not last a week or more, or ever change into purple stains, or true petechiæ. In a strict sense, the production of all such spots is referable to a common origin, namely injury or rupture of minute blood-vessels in the *cutis vera* or deeper down. I found, on some trials, that application of the cupping glass did not bring out the spots, but rather rendered them less distinct, owing to the local hyperæmia resulting from diminished air-pressure.

10. *Petechiæ*.—Spots manifestly hæmorrhagic and indelible from the first, were less usual than the above; but into such definite form, the pink blotches sometimes passed before fading. Therefore, it would appear there are several grades of sanguineous effusion regulated by the amount or rapidity of extravasation, and possibly by the character of the exudation. According to my experience the more evident petechiæ were to be found in remittent and other ill-defined fevers, at the late epoch; and hence they had no real diagnostic value: their site is always alike, whenever found. Perhaps they were most abundant, however, in the worst form of spirillum fever, or that attended with deep jaundice and a typhus aspect. Diagnosis—black pigment-spots in native skins, may closely simulate petechiæ.

CASE LXVII.—K. S., 24, Muss. weaver, admitted on 8th day of invasion, dying next day (? at acme), t. 105°·8, the blood containing many large spirilla and fat-granule cells, coagulating imperfectly, and being of thin, brown aspect. Besides a few minute vesicles on front of chest, there were many petechiæ about the elbows, in axillæ, on front of shoulders and on the back, with some larger purple stains on front of abdomen and chest; these were true hæmorrhages, some being bright red, others becoming purple, and acquiring a tinted halo: in the axillæ were several dozens of them. There were some ecchymoses in the injected conjunctivæ: pupils decidedly contracted and equal: internal hæmorrhages were found after death, the man dying of cerebral effusion and enteritis. In another case dying of pneumonia after the invasion, petechiæ appeared on the reputed 7th day of disease on the chest and back, arms, and lower limbs (also it is said on the face); those I saw beneath the clavicle were very dark, numerous, and clustered, yet they are reported as disappearing 5 days later or just before death: patient a Hindoo labourer, æt. 30: date 2 July, 1877. Such brief duration of spots decidedly resembling blood-effusion, was not rare: it is striking.

11. *Rashes*.—Purplish mottling of the skin, in patches, was very

rarely noticed ; obviously, it might have been overlooked in the dark skins of native patients, yet it has been distinctly seen, usually with the rose-tinted or purplish eruption (*vide* CASE LIV.) ; and I perceived some hyperæmic patches in my own skin at first attack. On the application of a cupping-glass, such rashes were not intensified.

Vesicles, pustules and boils, were occasional attendants and early sequels of specific pyrexia. Once I noted at the onset of first relapse (spirilla in the blood) a large darkish vesicle on the thigh, looking like 'Charbon,' the liquid contents of which did not coagulate spontaneously ; large granule-cells were present, some filled with actively moving granules, also moving free granules which I compared to *micrococci* ; no spirillum here. Next day a slough had formed, the pus around which contained, beside pus-corpuscles, an immense number of micrococci, identical with those in the swarm-cells : date May, 1877. The patient had epistaxis at the time, and thrice afterwards, with active delirium at crisis : the fever was of low type, eyes injected and jaundice, with enlarged spleen ; convalescence tolerably prompt. Commonly pustules appear later : M., 35, with low fever, shortly after a brief relapse, a pustular eruption on the left knee, and earache : boils were generally sequelar.

12. *Desquamation of the Cuticle.*—This was quite unusual, except in the form of minute branny scales following the drying up of sudamina : after even profuse sweating and maceration of the epidermis, apparent exfoliation did not take place. Once it was noted on the limbs after invasion of low type (lysis) with œdema and a few pink spots, hypogastric tenderness, diarrhœa ; urine retained, sp. gr. 1010, pale, clouded, a little albumen : there were signs of a latent relapse at the time. It was seen on the neck of a man on last day of relapse, before critical sweats : and has been noticed on the corpse. Exfoliating cuticle was then in comparatively small flakes. For instances of desquamation at crisis with little or no sweating (perhaps in consequence thereof) see above CASES XVIII. and XXI.

13. *Odour emitted from the Skin.*—In several cases, both surviving and fatal, from the body of patients, a sour, musty, or offensive smell was given off, especially about the time of critical sweats : sometimes this was so evident as to be complained of by the sick man himself, and in the instances I met with the skin was not in a particularly foul state : it is not the odour of dysentery or colliquative diarrhœa.

14. *Spontaneous Gangrene.*—The lividity, coldness and shrinking of nose, ears and extremities of limbs, however noticeable at acme and fall of fever, did not persist and were not followed by local gangrene.

At height of the epidemic, I once saw gangrene of the cheek in an adult male, contemporary with the presence of granule-cells in the blood large enough to block the skin capillaries : further particulars of this case (which much impressed me at the time of its occurrence) have been mislaid.

15. *Bed-sores.*—Sloughing from pressure. The occurrence was rare, although many patients had been much reduced by want : the following instances show the varying conditions under which it was seen, primary

fever being amongst these. The local effect of involuntary faecal and urinary evacuation did not seem to be concerned here.

M., 45, butler, habits possibly not temperate, and affected with elephantiasis of r. leg: exhibited pronounced symptoms of spirillar infection (first attack), and on reputed 8th day, or acme, redness appeared on the back, which with continuation of fever (now attending pneumonia) ended in bed-sores, the scrotum also becoming excoriated; death on second day after apparent critical blood-change. This early supervention of decubitus is noteworthy.

M., 25, weaver, weak subject; at end of pronounced first relapse was much reduced, and showed diffused inflammation over lower end of sacrum and coccyx; which, however, under his rallying did not pass into wide sloughing: recovery after a second relapse.

M., 50, destitute, 30 days after end of specific infection attended with moderate pyrexia, but abundant visible blood-contamination, acquired bed-sores and lingered in a feeble state for four weeks longer.

Complications affecting the Respiratory Organs.—The principal and most frequent of lung-changes found after death being that of pneumonitis (present in nearly $\frac{1}{3}$ of autopsies), in the remainder of cases the lungs were in an opposite condition of pallor and collapse, or only congested, or of quasi-normal aspect. Presuming these varied conditions, after excluding degrees of pneumonia, to represent the state of the organs amongst surviving fever-patients, it may be said that the common symptoms of hurried breathing and slight cough, with scanty mucoid sputa, do not imply more than slight or non-persistent lung-changes; whilst severer dyspnoea, cough, râles and hardly altered percussion-note, attend the state terminating in paleness and inflation or collapse. Congestion of the organs, diffused, lobular or hypostatic, did not give rise to special symptoms in addition to those already well known, but it often happened that owing to extreme feebleness or depression of the patients, close physical examination was impracticable. I am unable, also, to add many clinical details of the rarer lung-lesions mentioned in the Chapter on Morbid Anatomy (some of which may have supervened shortly before death); and it must suffice to invite attention to these several lung-complications, as affecting both diagnosis and prognosis.

16. *Laryngitis*.—Two or three instances were met with of sudden implication of the larynx, resembling acute oedema or transient inflammation: the symptoms were of mild character, and not persistent. Implication of this part in extension of pharyngitis, I do not recollect seeing.

17. *Bronchitis* as a separate affection, seemed to be practically unknown at necropsy: and however frequent it was as an attendant upon the pneumonia usually causing death, amongst survivors bronchitis of definite presence was by no means common. When occurring, its tendency was obviously towards deeper lesion; and I regard it as probable, that most of the examples of bronchitis as a complication of spirillum fever, were instances of mild catarrhal pneumonia; this opinion being founded on the scrutiny of cases showing non-specific pyrexia either continuous with the spirillar attack, or appearing as secondary fever after crisis. The fatality of pneumonia in this fever would appear unusually high, unless it be supposed that some of its milder forms were so blended with bronchial inflammation as to become liable to

oversight or misinterpretation ; just as is known to happen in other fevers, and also independently of specific infection.

Acute bronchitis seen during spirillum fever may be regarded as a quasi-exaggeration of the lung congestion invariably present in some degree ; and where it supervenes after the crisis with secondary pyrexia, there will be almost always found present other local derangements (as of liver, spleen, bowels), with a tendency to depression, or the typhoid state, or delirium.

Instances of this kind were not uncommon, especially amongst the Mussulmans from N. India : they were seen in adults and children of both sexes ; and though rather more frequent after primary invasion-attack, yet also happened after the first relapse ; it being noteworthy that the secondary fever (judged to be non-specific) with its lung and other complications, sometimes appeared about the time of, and as it were in the place of, a simple recurrent attack. The severity of the phenomena varied, but not seldom it was obvious that if a little prompter in occurrence (*i.e.* nearer the critical depression going before), or more pronounced in degree, the complication might have proved fatal ; the main determining consideration, in my mind, here referring to the presence or extent of actual pneumonic lesion.

The relationship of pronounced bronchitis (so-called) to pneumonia, and the great frequency of these complications in severe attacks, were evident amongst a group of 15 hospital servants infected in their wards : 6 of 9 survivors presenting them in marked degree, and 3 of the 6 deaths being mainly attributed to lung inflammation.

Such milder degrees of bronchial congestion and inflammation as promptly subside with specific fever, or if lingering do not lead to manifest perturbation of the system, were the most frequent of all complications : they shade off towards the above-named severer forms, or, on the other hand, to the 'cough' already alluded to as a nearly invariable symptom.

In the last series of cases analysed, decided bronchial congestion during the invasion-attack, was noted towards its close in 54 p. c., at the acme in 66 p. c., and at the crisis in 35 p. c. of instances ; these ratios indicating a progress towards maximum with the final perturbation, when, there is reason to believe, pneumonia eventually fatal often was initiated. Amongst all casualties, the tendency of bronchitis was to augment till the crisis or end of attack.

During beginning of the first apyretic interval this system persisted in 20 p. c. of cases, entirely subsiding, however, by the 4th or 5th day : it occasionally first appears at this stage on 2nd, 3rd, 1st, or 5th day, and at all these dates pneumonia was known to begin, when the lung complication became severe. I also noted bronchitis with some pyrexia, about the date of a regular recurrent attack.

With first relapse the symptom appeared or re-appeared, chiefly towards the close in 17 p. c. of cases, at acme in 24 p. c. and at fall in 22 p. c. : its diminished frequency at the recurrence being in correspondence with less evident blood-contamination and briefer pyrexia ; then, too, the severer pneumonic lesion becoming comparatively uncommon. In casualties, bronchitis was rare, except along with pneumonia.

During the second apyretic interval it was noted in only 9 p. c. of cases, being early, mild and brief.

It did not appear to me that in the uniform, tropical climate of insular Bombay, season of the year exerted any decided influence on the prevalence of the lung-complications; the majority of cases being seen between April and September (when famine-fever attained its maximum), and not during the cooler winter season.

18. *Pneumonia*.—This complication was both frequent and serious; and its forms and degrees were as varied here, as under combination with remittent or other fevers.

The notes of 21 autopsies show consolidation of an entire lung 4 times (3 on right side), there being also engorgement of the opposite organ, and marks of pleurisy: the lower lobes only were solidified 8 times, viz. 4 on both sides and 4 on one side (the right and left equally often): the upper and middle lobes alone were condensed 5 times, and in 4 instances there was disseminated induration in one or both lungs, partially or throughout. Pleurisy co-existed, in all, 13 times.

At decease the inflammation had not passed the first stage 3 times, had reached the second stage 12 times, and the third 5 times; whilst in 1, consolidation seemed to be chiefly from lobular hæmorrhage.

In 4 deaths at close of invasion-attack, the pneumonia engaged a whole lung or the lower lobes of one or both lungs, and it had already attained the second stage: once the form was that of disseminated patches of induration, and twice, at least, there was some pleurisy. As in 2 other instances of death on second and third day after the crisis, there was similar evidence of advanced disease, it seems clear that pneumonia may come on very early in a first spirillar attack, and its own symptomatic fever be lost in the specific pyrexia.

Most deaths from this complication took place within a week or ten days after the first crisis, a non-febrile interval, more or less distinct, being interposed prior to secondary fever; and between the two groups of completely blended pyrexias and the clearly sequelar pneumonic, there were seen gradations of concurrent fever not easy to interpret without repeated scrutiny of the blood. In 3 deaths from 5th to 7th day, the upper lobes of the lung were inflamed; in 3 others dying from 9th to 10th day, the lower lobes or whole lung; and I mention these data, because it was possible the later difference might be connected with latent spirillar infection. In another instance, pneumonia commencing at first interval became continuous or confounded, with the specific relapse; and such intermingling (here carefully unravelled) might be regarded as indicative of a further series of cases wherein the whole apyretic interval became occupied with extraneous fever, and so the spirillar pyrexia entirely hidden; several examples of such incidentally 'continued' fever were met with, in both living and dying.

During the second apyretic interval, pneumonia again appeared, though less often, the milder relapse probably less predisposing to it; there was one death on the fourth day (upper and middle lobes involved of left lung), and one on the fifteenth (disseminated patches of induration in right lung).

Pneumonia might set in 3 or 4 weeks, or longer, after the close of

specific pyrexia, primary or secondary ; it had then no special characters, engaging either whole lung or both lower lobes.

This autopsic series and a few other fatal examples, together furnish 27 instances of pneumonia in 97 casualties, or 37·3 per cent., which may be rather under the truth, as the pneumonic complication is apt to be overlooked ; this proportion exceeds, however, the scanty ratio of pneumonia amongst survivors, for death seems to be the ordinary termination of undoubted lung-lesion of this kind. My notes contain few instances of recovery after decided pneumonic inflammation, at any stage of spirillum fever ; whilst there are several of recovery after marked bronchitis, coincident with ordinary pneumonic dates of end of invasion and early post-crisis period : as before intimated, the comparison here is strictly between fully-developed and quasi-latent pneumonia, results depending partly on the amount of parenchymatous lung-lesion involved.

As to the nature of the secondary pneumonia, it is open to question if it were always the same ; and a correct discrimination here, may be the first step to preciser knowledge.

From the evident connection of this complication with the close of the first and severest attack of spirillum fever, it might well be supposed that the contemporary overloading of the blood with leucocytes and granule-cells was some way concerned in its production : yet I have shown that in several typical instances of death at close of invasion, the lungs were pallid and sometimes partly collapsed ; and, besides, pneumonia may come on several days after the fall when the blood has regained its normal aspect. When, however, lobular collapse takes place about this time, inflammation may promptly supervene in these shrunken parts and thus give rise to disseminated indurations.

According to current pathology, such partial collapse of lung tissue is apt to follow on inflammation of the finer bronchial tubes ; and I have before stated that bronchitis is one of the commonest attendants on spirillum fever. Two autopsies have been described where the base or the entire lung was permeated by fine frothy serum, death happening at invasion-fall or end of relapse ; here, too, localised collapse might be looked for. That acute capillary bronchitis also terminates in lung solidification (here included under the head of Pneumonia) was distinctly proved by another case dying on ninth day after crisis, with the back part only of left upper lobe in a state of splenisation, the rest of the lungs displaying the signs of acute bronchial catarrh.

For some of the examples of pneumonia seated in the lower lobes of one or, especially, of both lungs, the term hypostatic might be regarded as correct.

There were two or three instances of highly localised indurations of tissue at the surface of the lungs, in different parts, when pleurisy also was present ; and these I should regard as examples of pyæmic, or toxæmic, pneumonia. The concurrence of bubonic and diphtheritic inflammation would also be evidence of blood-contamination.

There still remains to consider the cases of croupous or lobar pneumonia, and as possible explanation of them, individual susceptibility, malarious influence, incidental exposure to cold or some unknown blood-contamination, may be named. Reference should here be made to the Chapter on Morbid Anatomy.

The mortality must in general be considered high, and the prognosis as bad.

Nothing peculiar appears in the clinical characters of specific pyrexia attended with, or followed by inflammation of the lung; an unselected series of 12 fatal examples giving $8\frac{3}{4}$ days as the mean duration of the attack (primary); the last-noted temperature and pulse as t. $104^{\circ}5$, and p. 128, or only a little in excess of the means in survivors; the critical fall when seen was direct in 8, lytic in 3, and once only partially witnessed, mean minima t. $96^{\circ}6$, and p. 95, which is higher than the general mean, and therefore a quick pulse at the fall should engage particular attention. There was a prompt rebound of temperature 7 times, with early initiation of lung symptoms; 3 times this rise was deferred, and in 2 cases there was no rallying after crisis, although then the lungs were much inflamed—an important datum. Death partly or wholly from pneumonia occurred on any day after the crisis from the first to the thirty-fourth; mean date ten days. The apparent mean duration of symptoms was for death at the first or second stages, about 4 days; and at the third stage, 9 days.

According as the pneumonic fever is present alone or blended with the specific, so will its own features be more or less apparent; in it generally the mean temperature was lower and pulse slower (though firmer); the headache, thirst, and aching pains decidedly less pronounced, and the hepatic, gastric, or splenic implication altogether wanting. On the other hand, the frequent cough with viscid, bloody sputa, pain in the side and marked dyspnoea, together with the usual physical signs of inflammation of lung or pleura, belong solely to this complication. As to the varying facility with which the local disease may be made out, I need only remark that minor degrees of catarrhal, pyæmic and hypostatic pneumonia were apt to be overlooked under the ordinary conditions of enquiry at Bombay, which seldom permitted thorough clinical scrutiny of the chest, day by day; this experience is not peculiar. Valuable as a thorough investigation might prove, the observations it was possible to make did not reveal any novel positive data, as to the nature and causes of inflammatory lung-implication.

In the series analysed, there were no cases of infants and proportionately few of women. Practically, nothing could be learnt of pre-existing states of the lung in most cases; but old bronchitis, asthma and phthisis were sometimes known to be present, without pneumonia supervening with the specific pyrexia.

Influence of Season.—During the years under review pneumonia, in general, was commonest at the close of the rains and in the cold season; in connection with spirillum fever, however, such distribution was not so apparent: thus in the larger J. J. H. series, of 29 fever-deaths from March to May (hot season) there were 8 with pneumonia; of 25 from June to August (wet season) there were 7; of 22 from September to November (malarious season) there were 4; and of 6 from December to February (cold season) there were 3. In 15 other deaths there were 4 with lung-inflammation, of which 2 occurred in the hot season; and as characteristic examples of all forms of pneumonia were seen throughout the year, I infer that this complication was not essentially connected with atmospheric states.

More importance would seem to attach to the influence of specific contamination, aided by privation, checked excreta, mental anxiety and prior malarious cachexia, upon the blood and system in general of the subjects implicated; which of these co-agencies were most effective, the data do not enable me to say.

The clinical cases supporting the above remarks also show, that whilst the physical signs of pneumonia may be tolerably constant, the accompanying pyrexia was highly variable. Perfect lung-consolidation not being found prior to the critical fall of invasion, two instances were met with of death at this time with confirmed double pneumonia; unfortunately they were not seen till 8th day of illness, and hence I am able to state only that the crisis was not excessive or peculiar; death took place one or two days later. One of these cases is copied here:—

CASE LXVIII.—M., 40, m. after admission t. $103^{\circ} \cdot 2$, p. 110, breathing hurried, countenance dull, some jaundice, tongue and skin dry, headache, thirst, pains, constipation for 8 days, abdomen tender and tympanitic; chest reported normal to auscultation and percussion; the blood full of spirilla. Defervescence now began without sweats, and continued by lysis, the min. being reached in $2\frac{1}{2}$ days, t. $97^{\circ} \cdot 2$, p. 88; purple spots appeared, hiccup, slight cough, pulse extremely feeble, respirations 24 per minute, some sputum tinged with blood, delirium, coldness of the limbs and death in the aspect of collapse 36 hours later. The whole of the lower lobe of the r. lung was consolidated and softened; lower lobe of l. lung deeply engorged; parenchymatous inflammation of the liver, spleen large and soft, kidneys pale and soft, vascular patches in the intestinal mucous membrane: so far as known here, pneumonia had come on during the gradual defervescence, whilst the t. was falling, and the instance may further be regarded as pointing to the complications liable during lysis of spirillum fever.

Two other selected instances in youths of 16 and 20, show pneumonia supervening immediately after crisis, concurrently with smart rebound of t.: in one case death happened 9 days afterwards, the fever becoming remittent, pulse variable, typhoid symptoms before the close: the r. lung was emphysematous, dry and bloodless; the left inflamed throughout and its lower lobe suppurating: changes elsewhere not striking. The other instance is reproduced:—

CASE LXIX.—M., 20, emaciated, gave a history of fever, but none seen for 10 days after admission, when a smart attack set in suddenly, and lasted 3 days; spirillum present, and many usual symptoms; at the crisis a descent of $10^{\circ} \cdot 5$, p. sank to 80 from 144; drowsiness and some delirium at the end; an immediate rebound took place and on second day t. $105^{\circ} \cdot 2$, p. 128, no delirium but great depression, no cough seen or heard, no expectoration, yet dulness at l. apex, in front with tubular breathing on third day; increasing weakness and death two days later, without permanent defervescence. At autopsy—r. lung healthy, upper and part of lower lobe of l. lung in first stage of inflammation, with reddish serum in the pleura; splenic infarcts and old colitis. Chart 26, Plate VI. This was an example of pneumonia directly following spirillar infection, and the chart shows how closely pneumonic fever may resemble pyrexia of 'rebound.'

The following case shows more deferred lung-disease, and especially the difficulty there may be of correct diagnosis, when pneumonic and spirillar fever become blended:—

CASE LXX.—M., 22, scorbutic, admitted on 4th day of pronounced invasion, with most of the usual signs of infection: *perturbatio critica* and crisis with relief; slight cough only and no sputum: mild secondary fever ensued with pains in the

joints, which subsided in two days, and my note was that the patient now looks convalescent for the first time ; fever, however, promptly returned and it persisted without evident complication for three days, the man rapidly becoming prostrated : then the blood on close scrutiny by the Albrecht process displayed a very few spirilla, there was slight mitigation of the symptoms, with no alteration of temperature. and some consolidation of the r. lung above was noted : pyrexia continuing, the typhoid state supervened, with splenic and hepatic fulness and tenderness, delirium, looseness of the bowels, a dicrotic and very feeble pulse, dyspnoea at the last. After death—apex of r. lung consolidated, on its surface some marks of pleurisy, and commencing gangrene ; also were seen converging white lines, as of lymphatics containing fatty matter or pus, and within the lung-parenchyma the larger vessels around were filled with pale clots : liver enlarged, with pale patches in it ; mucous membrane at mid-region of stomach highly injected ; spleen little changed ; kidneys much congested. In this instance, also, the pneumonia seemed to be of blood-origin ; source of poisoning unknown. Chart 24, Plate VI. : date of relapse indicated only by blood-spirillum appearing.

Pneumonia causing death at later periods of the fever was comparatively rare: an instance was the following :—

CASE LXXI.—M., 30, scorbutic subject, gave a history of first attack, the end of a relapse being witnessed in hospital : then ensued a series of mild daily febrile perturbations which continued for a fortnight, the t. never rising above 101° , but p. seldom below 100 ; finally a sudden rise to 103° , p. 118, and death next day, probably from syncope. During this period, the blood was frequently examined with negative results ; no definite complication was noted by the medical officer in charge, debility being the main symptom all along. At the autopsy—disseminated lobular pneumonia at advanced stage in r. lung ; left lung healthy ; clots in the heart, liver very large (fatty?), and so the spleen (softened), kidneys healthy-looking ; subcutaneous effusion of blood and serum in the lower limbs. This was another example of the modified lung-inflammation found after spirillar infection.

Respecting the cases recovering from pneumonia after fever, the amount of lung-consolidation seems to have been always slight, although fever might be high ; bronchitis had usually preceded and other complications were often present, the pneumonic not having the predominance noted in fatal cases. Excluding the more probable examples of blood-deterioration, it might, however, be said that in all essential particulars, except that of intensity, no difference obtained between cases surviving and the dying. The usual symptoms were not noted, and the whole tendency of evidence was to show that lung-solidification might certainly exist without its presence being suspected.

There are no other data bearing on recurrence of pneumonia, but it is not improbable the examples seen of lung-inflammation supervening a fortnight, or longer, after specific crisis, were connected with the infection through dormant lesion earlier incurred ; and in some subjects, it is likely that a predisposition to acute pneumonia was then established ; for best on such hypotheses, can be understood the sudden recurrence of severe local symptoms after a long interval of quasi-convalescence.

19. **Pleurisy.**—Not seldom seen in connection with pneumonia, this complication was sometimes the more striking indication of blood-poisoning ; as appears in the following case :—

CASE LXXII.—F., 12, admitted with high fever and dysenteric symptoms said to be of several days' standing : the blood contained spirilla ; there was no cough, breathing quickened, frequent muco-sanguineous stools with tenesmus, *lumbrici*

passed; after a final marked paroxysm the fever declined, becoming mild and irregular, purging continued, much emaciation ensuing and death 8 days later. At autopsy—mucous membrane of large intestine studded throughout with round, raised ulcers, largest in cæcum and rectum; lungs non-adherent, mostly crepitant, the right pleura widely inflamed including its diaphragmatic layer, with streaked vascular lymph on the parietes, contents turbid, flaky serum; at base of r. lung was some consolidation apparently due to fibrinous infiltration in the form of nodules, one of which was as large as a nut; the heart, liver, spleen and kidneys seemed healthy; there was some reddish serum at base of brain.

Pleuritis also occurred in conjunction with febrile turgescence of liver or spleen, as indicated in another case:—

CASE LXXIII.—M., 20, admitted on 7th day of invasion, symptoms pronounced; after marked critical perturbation and defervescence, there ensued secondary fever, which from the 3rd day assumed the form of daily remittent paroxysms till decline in 8 days more, max. t. 103° , p. 120; on admission I noted unusual quickness of the breathing, which at acme was 48 per minute; no dullness of chest, some dry cough, as usual; heart's systole loud, much abdominal fulness and tenderness, right rectus abdom. muscle tense, tongue red, shrunk and dry; much distress at the acme, with delirium, involuntary evacuation of fæces, jaundice, the urine free from albumen: after crisis depression and continuance of splenic pain especially, quick breathing, dry cough and on 4th day acute pain in left side of chest, where a loud friction sound was audible: hepatic tenderness diminished; the man was now reduced in flesh, and sleepless from the pain and cough, but did not become worse; pain in the l. shoulder followed, most at night, the spleen had gone down, urine continued free from albumen; with decline of fever convalescence after 3 weeks. Here I associated the pleurisy with peripheral splenitis.

20. **Asthma.**—That amongst other chest complications, pre-existing asthma may become greatly, perhaps especially, aggravated during an attack of relapsing fever, was shown by the following case:—

CASE LXXIV.—M., 40, spare, admitted at close of a febrile attack attended with bronchitis (seemingly chronic); no blood examination: soon rallying, he remained well for 10 days, when fever returned with renewal of the chest symptoms, the dyspnoea becoming urgent; t. daily reached 104° , p. mounted to 150, and soon after this the blood-spirillum at first not seen (? overlooked) became visible: judging that the respiratory troubles were disproportionate to existing signs and other symptoms, enquiry was made and it was learnt that the man had been subject to asthma for 5 years. The ensuing interval was now occupied by febrile paroxysms (non-specific) and without subsiding the dyspnoea became lessened: after 8 days spirillar pyrexia recurred, lasting 5 days and ending abruptly; a brief yet smart rebound followed (anal abscess now), after which the normal level: during this relapse the man's sufferings were very great; with the signs of chronic bronchitis, there was no dullness of the chest and rallying was prompt on cessation of fever: mild dysentery alone checked an almost unlooked-for convalescence. Without the aid of the microscope this long attack would, I doubt not, have been regarded as one of remittent fever with bronchitis: see Chart 12, Plate V. It is probable contagion occurred in hospital.

The dyspnoea at acme of uncomplicated spirillum fever often being remarkable, in certain cases it may simulate asthma; then, at least, trenching on the narrow limit between functional and lesional chest derangement.

CASE LXXV.—M., 37, hospital servant, caught fever while on duty in a medical ward; the attack lasted 7 days, being pronounced, and declining slowly, some irregular perturbations of t. followed, but no distinct relapse; the pulse, not unusually frequent at first, at acme and fall much quickened, together with the chest symptoms; and it afterwards remained irritable. Until the 6th and last day of fever, there was

no complaint of breathing; previous e. t. $104^{\circ}\cdot6$, p. 100, wet sheet packing applied: this m. t. $102^{\circ}\cdot6$, p. 120, full, bowels relaxed, no sleep, cough and oppression on the chest have supervened, sputum scanty; e. t. $103^{\circ}\cdot8$, p. 128, some sweats during day, less headache, the sense of suffocation remains: 7th day, m. t. declined to 101° , p. 120, soft, is not able to lie down from dyspnoea, chest resonant, respiratory sounds distinct, heart sounds feeble only, eyes heavy and suffused, general aching pains, skin moist, tongue dryish and brown; some oedema of the insteps: bowels relaxed: e. t. $98^{\circ}\cdot4$, p. 116, skin clammy, the dyspnoea not less: 8th day, m. t. $97^{\circ}\cdot4$, p. 140, small and soft, he cannot lie down, no cough or expectoration; left side of chest posteriorly is slightly dull on percussion, breathing there tubular, voice resonant, moist râles heard around; he vomits upon eating (hiccups next day); urine high-coloured, acid, 1013, contains $\frac{1}{2}$ vol. of albumen; 3 stools semi-consistent: e. t. 96° , p. 112, better developed (lowest critical fall of t.), the difficulty of breathing is less; remedies had been actively employed. On the following day, m. t. $97^{\circ}\cdot2$, p. 96, chest symptoms subsiding, tongue dry, hiccup; e. t. $97^{\circ}\cdot6$, p. 100, hiccup ceased, he can lie down and manifests tendency to sleep, but dreams ensue; giddiness continues, the urine free from albumen, bowels quiet; no cough and convalescence in a fortnight. It need not be supposed the wet packing brought on transient pneumonia, for it was well known that frequently lung-lesion threatens at the acme and lytic decline of spirillum fever, not rarely, indeed, supervening of itself: the chart of this interesting case presents no unusual features whatever, beyond the pulse range at fall, above alluded to: the concurrence of lysis-tendency and suffering, is also evident.

21. *Phthisis Pulmonalis*.—From the known frequency of this disease amongst the inhabitants of Bombay, and from the liability of subjects under spirillar infection to pulmonary inflammations, it might be supposed that the conjunction of spirillum fever and hectic would be seen sometimes in hospital; and in fact 5 or 6, at least, of such instances were recognised. Occasionally it seemed as if the lung wasting were hastened by the specific infection, at other times not so; nor was it ascertained that a predisposition to phthisis might be aroused by relapsing fever. Tubercular disease of the lungs was not detected in 74 consecutive fever autopsies, mostly of recent illness.

Two of the earliest cases seen were of Indo-Portuguese youths (a class liable to phthisis), who showed in succession bronchitis, pneumonia and phthisis, with relapsing fever and much subsequent exhaustion; one also caught chicken-pox in hospital before leaving. The following instance is quoted as illustrating blended hectic (pyæmia) and spirillum fever, with consequent obscurity of symptoms and need of special diagnostic means:—

CASE LXXVI.—F., 30, thin subject (son here with fever), admitted after a week's illness at home, pyrexia, low delirium, bronchitis and some consolidation of right lung, jaundice, enlarged spleen, dry tongue, costive bowels: the fever remitted and also intermitted, for 7 days, during which time the blood was frequently examined with negative results; some general improvement was taking place, when during a fresh exacerbation I detected a few spirilla in the blood (e. t. 104° , p. 108, or not much quicker than before), delirium now returned, giddiness and lachrymation without pain or redness of the eyes; next day also the blood contained a few organisms noted as not so active as usual, e. t. $101^{\circ}\cdot4$, p. 108; the following m. t. $98^{\circ}\cdot2$, p. 100, decline representing the crisis of this brief relapse, the blood now containing some large nucleated, granular cells like fatty endothelium. After two days' sub-normal temperature, pyrexia again returned, being of similar character and rather more pronounced, cough grew worse, the sputa became nummular, and the physical signs of tuberculosis (sub-acute) were detected in both lungs; the strength diminishing, aphthæ appeared in the mouth; the blood showed only some free protoplasmic masses of large size; after a few days more the patient was removed by her friends. Chart appended, No. 13. Plate V.

Affections of the Circulating System.—Some much-needed information is here wanting, from my having to give up enquiry when the materials had been collected.

22. *Heart.*—Respecting the significance of post-mortem clots in the heart, a pathological rather than clinical import seemed to be indicated.

The coarser changes of the heart-muscle, such as pallor and softness, being noted chiefly in deaths during invasion and second post-febrile periods, may be associated with corresponding granular and fatty changes of liver, spleen and kidney; and I here note the main symptom of feebleness with rapidity of the heart's action during primary fever, whilst later on feebleness with slowness, irritability, and tendency to irregular or intermittent action. It is conceivable that death at either stage was often hastened by unusual implication of the heart; and a disqualification for physical exertion was not seldom evident in patients, who insisted on returning to their avocations from restlessness or family needs: the future of such individuals remained unknown.

The smaller cardiac hæmorrhages elsewhere described, were not attended with peculiar local signs, so far as I am aware; nor could they, alone, have exerted considerable influence upon general symptoms.

Inflammation of the endocardium was not proved to be due to spirillar infection, early or remotely: nor was it apparent that pre-existing disease is necessarily aggravated thereby.

Inflammation of the Pericardium.—This outer, looser serous investment was the commoner seat of both hæmorrhagic and inflammatory changes, the local signs still being subordinate: thus, with the striking hæmorrhagic pericarditis described in the Chapter on 'Morbid Anatomy,' neither temperature nor pulse-course was extraordinary; feebleness of the heart was extreme, but in the record no allusion is made to dyspnœa or palpitation.

CASE LXXVII.—M., 30, washerman, said to have been ill 3 days, was brought in a state of low delirium and deep jaundice, t. $100^{\circ} \cdot 2$, p. 120; I found the blood to contain numerous spirilla, which after 18 hours on the slide were noted as being unusually large and sluggish: next day pyrexia declined, subsiding by lysis, delirium persisted, urine and stools passed in bed, hiccup came on, min. t. $94^{\circ} \cdot 6$, p. 98: slow rallying then began, and 8 days later the man, though weak, was in aspect convalescent. The pulse had risen with the supervention of hiccup and diarrhœa (?) just after the lowest temperature; but at last declined to 72, with t. $98^{\circ} \cdot 4$. Now the relapse came on, and lasted 6 days; temp. sustained (max. 103°), decline slow: the pulse gradually rose, max. 120, always feeble, imperceptible 2 days before death: the liver and spleen seemed unchanged, vomiting came on at close of relapse, jaundice was again pronounced; throughout exhaustion, and death with defervescence: spirillar blood-contamination was noted by Mr. S. A. as being constant and abundant. After death, was found copious cerebral hæmorrhage, extensive hæmorrhagic pericarditis, infarcts of the spleen, and inflammation with hæmorrhage at lower end of ileum; lungs collapsed, healthy; liver congested; kidneys blood-stained: this evidence pointing to a blood-change of which the cardiac disease was only one indication. I did not think the kidneys especially implicated: no dropsy; and no signs of acute rheumatism, which is extremely rarely seen or heard of in the class of patients to which this subject belonged.

CASE LXXVIII.—Another fatal case was that of a young woman, age 18, admitted for chronic diarrhœa, weak and anæmic; she displayed spirillar infection 20 days after her entering the ward (contagion in hospital), undergoing a pronounced

febrile attack of 9 days, and dying 3 days later with secondary febrile complications. Besides the characteristic signs of this fever, there came on palpitation, a systolic bruit (? friction sound also), oedema, delirium, jaundice, pharyngitis and epigastric symptoms: my impression was that both pericarditis and endocarditis had been enkindled. The detailed notes being mislaid, I am able to quote this summary only; autopsy not available.

23. *Thrombosis of Veins.*—A single instance was seen: such must be extremely rare during remittent fever, but the like is known to attend typhus and enteric. Its connection in the following case with prior great loss of blood (and resulting alterations in blood-quality), was very significant; yet hardly less so, its occurrence soon after crisis, when specific blood-changes are known to occur: perhaps both these conditions were concerned in production of the local arrest of circulation, and the instance of the child's father will indicate which may have been most influential.

CASE LXXIX.—F., 8, a thin, weak subject; admitted in a state of semi-collapse, t. 95° , p. 85 (? crisis after invasion), rallying was prompt and complete, t. becoming normal and p. at last 80-6: 8 days after admission the relapse suddenly set in, first m. t. 104° , p. 112, high fever continued for 4 entire days, the t. ranging 103° - 104° 8, p. 130-146, and resp. quickened, she was exhausted, but not distressed, there was some splenic enlargement, liver not so much affected, no jaundice: at acme of fever e. t. 104° 8, p. 140, resp. 40, skin dry, tongue white and moist, much headache, no eruptions, no delirium but does not sleep, abdominal organs not more tender, copious blood-contamination continues: grs. x. of chloral were gi en at bed-time. Next m. t. 102° , p. 138, on awakening and being taken in her mother's lap, profuse epistaxis came on, almost inducing collapse, both nostrils bled, upwards of $\frac{1}{2}$ pint of blood certainly lost, which swarmed with active parasites: under suitable treatment she rallied a little, e. t. 101° 4, p. 132; crisis now came on, or became complete, and m. t. 98° 6, p. 120, resp. 26, still in depressed state, pupils normal, spleen much reduced in size; e. t. 99° , p. 128. First day after crisis m. t. 98° 6, p. 120, though very weak, she had slept and was decidedly improving, spleen still going down; e. t. 100° 8, p. 120, and this rise probably continued throughout the night. Second day, m. t. 104° 6, p. 132, headache, furred tongue, dry skin, spleen more enlarged, no hepatic uneasiness, no delirium, no eruption; I found her quite conscious, lying on the side, with head thrown back; the spirillum had disappeared from the blood, which now displayed clouded plasma and increase of white corpuscles: this rebound of t. persisting, e. temp. at 4 was 103° , p. 136, notwithstanding frequent spunging of the body and free use of iced water; skin and tongue dry, no delirium, she had taken some liquid food; two hours later, t. 103° 4, she moans, screams and tosses about, the right limbs seemingly more than the left, is still conscious, pupils in mid-state, pulse fluttering and rapid; 8-10 P.M. the left thigh has become swollen, depression following the excitement, and death in an hour.

Autopsy 10½ hours afterwards; froth at mouth; l. thigh swollen, of bluish tinge, flexed and everted, the pubes swollen and red. Head—pupils of normal dimensions, brain very pale, all vessels empty, no effusion seen, and no other alteration in substances or membranes. Chest—lungs very pale, inflated, no disease; heart contracted, scanty pale clots on r. side. Abdomen—pallor general, intestines inflated; liver bloodless, otherwise normal; spleen very large, not otherwise diseased in aspect, but has become permeated by small air-vesicles: kidneys pale and firm, capsules rather adherent; some stellate vascularity of surface, a double ureter on r. side in connection with a narrow, elongated organ; bladder distended with urine, which contained flocculent shreds (? from mucous membrane), no albumen, bile-pigment present: other pelvic organs unchanged so far as examined. Left thigh—the swelling is due to serous infiltration and the evolution of gas; muscles and connective tissue equally involved, the former being red in hue, streaked with dark blood, and very soft: femoral artery pervious, so the femoral vein excepting the presence of loose clots in the ham: the long saphenous vein firmly closed with black clot, and so other veins on inner side of thigh, even the deep-seated; the corresponding internal and common

iliac veins on both sides of the pelvis, contained blood. This diffused thrombosis of the l. thigh was mostly superficial : embolic masses not seen in vena cava or r. side of heart : the clots in the saphenous veins were of soft consistence : arteries everywhere seemingly healthy.

A healthy young man while assisting at this autopsy, cut his finger and 3½ days later was seized with relapsing fever, suffering considerably from two relapses.

The father and mother of the above child accompanied her to hospital, the three composing a family of pauper-immigrants from the neighbouring province of Kattia-war ; date of admission July 2, 1879. The mother gave an account of late fever sickness ; the old man (a thorough famine-subject) was actually ill with fever, and his blood swarmed with the spirillum ; in him the pyrexia (? of first attack) subsided by lysis ; a rebound immediately followed, on 4th day t. 104°·4, p. 120, and during this post-critical fever no visible specific contamination of the blood : two days later death in a comatose state, there being found at the autopsy meningeal cerebral hæmorrhage, and suppurative inflammation probably thence arising. My impression was that here as in the girl, his daughter, the circulation of the blood had become impeded about the time of 'crisis,' the fatal effect being alike in both cases, though differently brought about. No more characteristic instances than these of spirillum fever, were seen by me during the previous two years, or when the main epidemic was at its height.

The temperature chart shows only the unusual feature of a sharp rebound on second day after crisis of relapse ; it is not peculiar.

Complications affecting the Digestive System.—These were numerous, and including those of the spleen, both frequent and marked.

24. *Inflammation and suppuration in the neighbourhood of the Parotid gland.*—This event, to some degree, was noted in 2 or 3 per cent. of all cases ; and nearly as often amongst survivors as in the casualties.

It rarely supervened prior to close of specific fever, usually following at a distinct interval the crisis of invasion or relapse. I have known it appear at the time when a first or second specific recurrence was to be anticipated, seemingly taking its place ; but there is no rule in date for its advent.

It was much the commonest after the first attack.

Oftenest the right side was implicated, or both sides together or in succession.

Similar swellings were not noticed of superficial glands elsewhere ; but in the most serious cases, deeper seated parts, as tonsils and fauces, were also implicated, either before or after the parotid swelling became apparent.

Nothing peculiar was perceived in the preceding specific illness, though usually the subjects were weak and oftenest Mussulman weavers, in whom spirillum fever tended to a low type.

Alone, this complication, single or double, was not necessarily of bad augury, and in some subjects convalescence, if delayed in completion, seemed unusually prompt to begin : other local lesion conjoined, prognosis became decidedly unfavourable.

Its diagnosis was direct : the form of attendant pyrexia was usually low and remitting, seldom sustained ; of 3–5 days' duration, or more when both glands were successively involved and suppuration prolonged, the pyrexia not then assuming any particular form amongst the many varieties of consecutive fever. The pulse was decidedly quickened : the blood free from spirillar contamination.

Before the formation of pus, the local suffering and intumescence might be considerable ; with it relief ensued, and the matter escaped spontaneously through the auditory meatus, or by rupture behind the auricle, if not let out artificially : there was little tendency to burrow in the cases seen, or to extend by sloughing. Suppuration sometimes became visible 4 or 5 days after fever and pain had subsided, and tenseness of swelling diminished ; the quantity of matter might then be small : apparent non-suppuration was very rare, and it seemed likely that scanty and deep-seated collections of matter were sometimes absorbed. Local induration may persist for many days.

In one very bad case (hospital servant), inflammation around the ear supervened on acute pharyngitis ; in the equally serious though less acute instance quoted below, it preceded throat affections ; but not always was parotitis intensified when followed by renewed fever, as the case of a younger man showed, in whom copious suppuration and relief were not checked by the relapse. The anatomical relations of the parotid at its deeper surface and through Steno's duct, sufficiently account for occasional extension of the inflammation : yet I do not recollect seeing the front of the mouth and submaxillary glands implicated, or the nasal cavities : the middle ear, however, might be involved, and the larynx.

CASE LXXX.—M., 35, Hindoo famine-immigrant from the Deccan, condition fair ; was admitted on 8th day of invasion, t. 104° , p. 116, there were no urgent symptoms but the right side of the face was swollen and hard, and the mouth could not be opened : the blood was found to contain several active spirilla of the usual aspect. Next day a remission ensued, the blood then containing also large granule-cells which seemed to have burst, displaying a large nucleus and swarms of minute granules in active movement : another exacerbation and the crisis followed, the local pain subsiding but not the hard tumefaction. The temp. now remained nearly at normal level, the pulse range 60 80, for 8 days, when a relapse suddenly set in (spirilla seen on the first day) which lasted 3 days, terminating critically and being followed immediately by prompt rebound and high fever of deeply remittent type (no spirillum in the blood) : this secondary pyrexia continued 5 days, and ended in extreme depression. During this time the local pain and swelling had increased but did not advance to suppuration ; great debility and the typhoid state supervening, with constant and uncontrollable hiccup, and finally inflammation of the tonsils and fauces : some jaundice had also appeared, but the abdominal symptoms were subordinate : no swellings elsewhere noted. The patient becoming insensible, was then removed by his friends.

25. *Pharyngitis*, Sore-throat, Dysphagia, Tonsillitis. — In varying degrees were seen in 3-4 per cent. of all cases, and rather oftener amongst casualties : sore-throat was commonest just after close of invasion, or at its end ; and nearly as frequent towards close of first relapse, or shortly afterwards. Its intensity ranged from painful deglutition with little visible change to distinct faucial inflammation (including also the tonsils and soft palate) : I do not remember seeing a diphtheritic membrane, but it happened that the severer cases could not be examined closely, and amongst those dying autopsy was not available. When mild, the affection lasted only 2-3 days, subsiding spontaneously ; the severer were always further complicated, and ended fatally in little longer time : most of the patients were young male adults, and Mussulman weavers ; 3 casualties occurred at the ages of 16, 35 and 55 : general condition decidedly low, and type of fever

tending to typhus or the icteroid form ; examples were seen chiefly at later periods of the epidemic. The preceding or attendant specific pyrexia was not unusually high, or crisis much marked ; some degree of secondary fever (varying, yet remittent and mild) accompanied the symptoms, when supervening on the fall ; and the pulse was quickened, usually weak. Diagnosis was direct ; dysphagia might be more considerable than explained by local congestion, paralysis was not noted distinctly : on ordinary occasions prognosis was not unfavourable, the fatalities always showing other local signs of systemic disturbance ; examples of extreme typhus tendency might not, however, entail this complication : the spirillar blood-contamination was always absent with non-periodic pyrexia : anatomical changes not ascertainable. The connection of sore-throat with earache and deafness was distinctly noted in some milder cases.

In illustration, reference should be made to the detailed CASE of H. A., No. X., 2nd day, 1st apyretic interval : another is that of CASE XXXII. M., 35, a marked instance of *typhus icterodes* : after the little pronounced invasion, a rebound of temp. not preceded by chills, slight in degree, but attended with depression, hiccup and a copious petechial eruption on skin ; there was also dryness and soreness of the throat, with much vascularity of the mucous membrane of fauces and tonsils, and (as it seemed) some petechiæ there : on febrile decline these local changes did not proceed further, and they did not recur with the subsequent relapse.

A fatal case was alluded to under the heading of Parotitis (at its termination) ; 2 others were hospital servants : viz. one a ward attendant, æt. 55, not seen until 7th day of invasion, t. 104°, p. 130, the local inflammation was less pronounced than the general depression ; liver and spleen not especially implicated : crisis moderate, delirium, vomiting, epistaxis, signs of pneumonia, hardness and tenderness of the abdomen, restlessness, moaning and finally unconsciousness, with death on 4th day after crisis : the secondary fever did not rise above 100°, p. 100, feeble, small and intermitting. The other instance offered a similar combination of serious symptoms illustrating the worse results of spirillar infection :—M., 16, attendant also in a fever ward, J. J. Hospital, general condition fair, but gums discoloured : the specific pyrexia was pronounced, t. 105°·2, p. 136, some epistaxis at acme, a moderate fall (t. 99°·4, p. 116) and interrupted, the copious blood-contamination persisting till near its end : secondary fever at once ensued, some previous bronchitis passed into pneumonia first on the left, then on the right side, the liver tenderness and enlargement persisting (not splenic) ; for 6 days fever was high and continuous when death ensued, which was preceded by typhoid symptoms and great tumefaction of the throat and neck on both sides : it was noted that the physical signs of the pneumonia were not developed, as usual, posteriorly and below, nor proportionately to the respiratory and general distress. Some instances of sloughing sore throat were seen at the G. T. Hospital which might have belonged to this series, although the blood seemed free from contamination ; for this form of disease is extremely rare in remittent fever, and diphtheria in native paupers seldom witnessed.

26. *Hiccup*.—Spasm of the diaphragm (with closure of the glottis) was noted in about 10 p. c. of cases, and almost solely in the severer instances or those ending by lysis, or attended with deep jaundice or secondary fever : I have however seen it troublesome when no such superadded conditions were present.

An example of minor degree is given in CASE IX. at fall of relapse : in other instances, also, it was seen at the period of defervescence, persisting sometimes a few days longer : the concurrence of vomiting was not invariable, nausea and puking might be present ; meteorism was rare ; extreme epigastric uneasiness, and either splenic or hepatic implication, though usual, were not constant : the state of the bowels

was not uniformly the same, or necessarily abnormal. In a characteristic instance of 'black vomit,' hiccup was not noted until gastric hæmorrhage had probably begun.

Corresponding to the undefined origin and accompaniments of this troublesome symptom, was the uncertainty of its relief by ordinary measures, local or general: sometimes many remedies were tried in vain.

CASE LXXXI.—M., 30, admitted at close of severe and characteristic primary fever, delirium and jaundice being present: crisis moderate, and at once followed by a compact non-specific febrile attack; mid-temp. 103° , p. rising to 120 ; subsidence prompt, but not critical. With the advent of this secondary fever hiccup came on and it lasted till the end, then ceasing of itself: general symptoms not increased; there was debility, also constipation, yet no local complication was detected, and the blood (frequently examined) showed no other peculiarity than unusual hæmoglobin forms. Appetite then returned and convalescence set in with the resumption of normal temp., the pulse remaining somewhat above normal. I was unable to associate this striking febrile rebound with such abdominal derangement, as the persistent hiccup seemed to point to.

In CASE XXXII. hiccup appeared at the close of invasion with rebound of temperature, deep jaundice, sore throat and an eruption of red and purple spots on the skin and fauces. I noted that petechiæ might also have been present on the gastric mucous membrane, at this time.

Another instance was M., 25; at marked crisis of invasion hiccup came on, some bronchitis and sputum tinged with blood; no skin-eruption: type of fever low, yet as in the above examples, convalescence was fairly complete.

27. *Gastric Hæmorrhage*.—Congestion, probably inflammation, and minute extravasations of blood in the mucous membrane of stomach and adjoining tracts being known to occur, more copious hæmorrhage occasionally showed itself (seemingly at identical epoch) either during life by the 'black vomit,' or at an autopsy without the previous emesis of blood: whether or not the alvine evacuations in any degree owed their frequent dark hue at and after crisis, to the presence of blood, I am not able to state.

The two following cases are characteristic examples of this complication: in both the occurrence took place with critical decline of fever, and was serious enough promptly to cause death.

Fatal Hæmatemesis.

CASE LXXXII.—34, Mussulman, resident in Bombay, ship labourer, a spare subject, not starved or scorbutic, was admitted with 5 days' fever, t. 102° and the usual symptoms: a few active spirilla in the blood: 6th day, m. t. $101^{\circ}6$, p. 100, weak, regular, no sweats, much headache and thirst, tongue dry, there is some bronchitis and pain on the right side of the chest, and considerable pain and tenderness in the upper abdominal zone, intensest at the epigastrium: no jaundice: the spleen is enlarged; no eruption: he is low and keeps his bed; one stool. The blood-parasite is now very abundant and active. Ordered diaphoretics, saline laxatives and poultice to r. side. Vesp. t. $103^{\circ}2$, p. 116, much headache and thirst, the local signs and state of blood unchanged; one stool. 7th day, m. t. $101^{\circ}2$, p. 100, little change, but spleen probably larger; there is much mid-epigastric tenderness, no hiccup, much thirst; tongue shrunken, its surface white and dry, edges red; no sweats; two pink spots on the left shoulder seen: the blood contains vast numbers of spirilla in its clouded plasma, a few white cells, little free protoplasm and no free granules. Vesp. t. 102° , p. 96, no sweats, much headache and thirst, slight cough and no sputum or pain in chest, epigastric tenderness rather less, no fresh eruption; he has become somewhat delirious: one stool (character not noted): blood-spirilla fewer but active,

they cluster and are sluggish : some quiescent filaments seem to become developed in the plasma at this time ; there is granular and vacuolated protoplasm, but no endothelial cells : is it near the end of invasion ? (MS. note). 8th day, m. t. $98^{\circ}6$, p. 96, no sweats with this fall, headache now slight, thirst still much, cough as before ; is slightly delirious and has some epigastric tenderness ; there is drowsiness and prostration, yet not complete crisis : he vomited once in the night (said to have ejected black matter, not kept) and now has hiccup, the stomach is distended and epigastrium tender : he slept (chloral given), no fresh spots. Blood-plasma tolerably clear, little free protoplasm, some large granule-cells, some free granules and short rods, many clusters of spirilla very large and active ; red discs shrunken and dispersed in this specimen. (Hence here was no real crisis, but rather defervescence by lysis, with persistence of blood-contamination. H. V. C.) Vesp. t. 98° , p. 100, no sweats, slight headache, much thirst, tongue dry, there is rather more epigastric tenderness, hiccup persists, vomiting once in the day (matters thrown away), two stools (not seen), depression and wandering of speech ; blood-plasma clouded, fibrillation not seen, some white cells and large protoplasmic masses, a very few free granules, red discs hardly changed ; all is quiet and not a trace of the spirillum to be seen. 9th day, continued decline of temp., m. 95° , p. 96, very soft, regular ; respirations 40, shallow ; skin clammy, no headache, slight thirst, he slept ; tongue moist, no stool ; urine very scanty, high-coloured, tolerably clear, albumen $\frac{1}{12}$ vol., urinary bladder empty ; there is abdominal fulness and uneasiness, and the stomach seems to be distended, incessant hiccup, great depression, no delirium but occasional restlessness, pupils normal, he vomited early this morning some black liquid : vesp. t. $96^{\circ}4$, p. hardly perceptible, has vomited three times since morning and is now moribund (death in 2 hours) : state of blood as last evening, with the addition of a few fatty endothelial cells. The black vomited matter of the morning was thus described - near a pint in quantity, no smell, thick, viscid, uniform consistence, tint in small quantities of clear, greenish hue : displays the presence of minute granules, of irregular form, dark green colour, also a few round cells probably altered blood of bluish tint ; many bacteria and some torulæ seen after 6 hours' rest : a few oil globules and no other peculiarities made visible by the microscope. Tested by contact with strong nitric acid on a white porcelain, surface no play of colours seen after two minutes, beyond a faint yellow tinge at the line of junction : conclusion that but little bile was present and much blood. Autopsy 1 hour after death ; stomach and small intestines filled with a similar black liquid, their mucous membrane pallid : the gall-bladder contained only 2 or 3 ozs. of glairy, yellow-tinted bile quite diverse to the black vomit : no splenic infarcts, or kidney disease : a very few petechiæ on both layers of pericardium, and in cerebral convolutions at one spot close to superior longitudinal fissure : colon and rectum empty.

In the preceding case there was visible proof of gastric hæmorrhage ; not so in the following instance, which, like the other, was not brought to hospital until near the end of life : their temperature-charts are without definite character.

Gastric Hæmorrhage.

CASE LXXXIII.—M., 34, also Mussulman resident, working weaver from the same loom-factory whence other fever cases (specific) came at same time (February 1880) : said to have been ill a fortnight ; on admission, t. $100^{\circ}4$, p. 128, very feeble and small, tongue dry and brown, much headache (frontal) pains and thirst ; liver and spleen enlarged and tender ; he is restless and distressed : vesp. t. $99^{\circ}2$, p. 106, cough and pain in r. mammary region, some dullness on percussion at r. apex, and no moist sounds audible : no jaundice. The low temperature and reputed duration of fever, did not seem to confirm the suspicion of this case being one of spirillum fever ; and in the midst of other engagements, I did not examine the blood whilst fresh ; a dried specimen, however, being taken, which after treatment with acetic acid, showed abundance of mature and immature spirillar organisms. During the following night the t. fell, being $97^{\circ}4$ at 7 A.M., pulse imperceptible ; it was stated that the man became worse in the night, keeping other patients awake by loud moaning and restlessness until the end ; there was no vomiting. At autopsy 4 hours after death, the stomach was found to contain, besides air, about a pint of black, grumous

liquid ; blackish mucus filled the small intestine, a little feculence in the large: in the gall-bladder much dark-brown bile (not like the pitchy liquid), liver pale, spleen large and infarcted, kidneys seemingly normal : old pleuritic adhesions on r. side. Urine in the bladder, clear, pale, 1016, no albumen, bile present. No other hæmorrhage detected.

For other details, see the Chapter on Morbid Anatomy.

28. *Enteritis*.—The aspect and comparative frequency of congestion, inflammation and petechial extravasations in the mucous membrane of the small intestine at its successive portions, being described in the Chapter on Morbid Anatomy, I have here to state that the symptoms noted during life, which might be supposed to correspond with these changes, were not so apparent, or constant, as to permit of special detail; and so far as I am aware, none of the local signs noted were peculiar or characteristic. It may be the clinical records are defective, yet this vagueness of symptoms is not really incomprehensible, since in the more marked cases, there was either general febrile excitement with several functional derangements, or prostration considerable enough to entail not less obscurity of intestinal signs. Thus, during secondary fever, the symptoms were those of general depression rather than of local suffering; and during specific pyrexia, enteric signs might be overpowered by the hepatic, gastric or splenic; or, at most, such indefinite marks were noted, as central and supra-pubic abdominal uneasiness, pain or tenderness; distension or retraction of the abdominal walls, and occasionally mucous diarrhœa (often green-tinted).

If symptoms of this kind be regarded as presumptive evidence of enteritis, that complication may be frequent; but passing over the slighter and more transitory lesions as being, at present, unrecognisable with certainty during life, I have selected for illustration 3 or 4 examples of the severer lesions capable of affording some useful information.

a. Inflammation with granular exudation at end of ileum; death during primary spirillar infection: deep jaundice.

CASE LXXXIV.—M., 25, not emaciated, brought in by the police (who found him lying on a roadside), day of fever reputed to be the 5th, much depression, t. 99°, p. 126, tongue dry and brown: next m. t. 99°·2, no sweating, p. 140, regular, full and very compressible, resp. 48, very shallow, in addition to much headache and thirst, general aching pains, slight cough, were noted great distension with air and acute tenderness of the abdomen, decubitus on the back with knees drawn up, eyes closed, jaws rather dropped, yet with this depression not the aspect of typhus proper; he was restless at times, turning on the side; moaning constantly, quite rational, pupils rather contracted, heart's action tumultuous though regular, impulse not felt; no chest dullness: no vomiting, he had slept a little, 3 faint pink spots were seen on r. shoulder and clavicle; urine procured after death high-coloured, clouded, 1014, albumen a trace, chlorides $\frac{1}{8}$ vol., urea gr. 1·46 per oz. I suspected acute congestion of the mucous membrane of stomach, ileum, colon or abdominal walls; but omitting fresh blood-examination, overlooking the rapid pulse, and (after so much experience) forgetting that low temp. did not necessarily exclude spirillar infection, I entered the remark—'severe remittent with jaundice, very rare typhous form:' the man died after a few hours; reported no change and rational to the last. Upon examining the dried specimen of blood taken on admission, abundant spirilla were found, and thus the correct pathology of the case made clear. Details of the enteritis are furnished in Chapter IX.; and it was pronounced and limited to the end of the ileum: the patient passed no stool during the short time he was seen alive.

CASE LXXXV.—M., 24, one of a group of admissions from a common locality, was seen on reputed 8th day of illness; t. 103°·2, p. 120, small and soft; several

active spirilla in the blood, which had the usual aspect and coagulated firmly; delirium and a condition approaching the typhoid, pupils contracted, conjunctivæ vascular, deep jaundice, many petechial spots on body, also sudamina; skin harsh, hepatic and splenic tenderness and hepatic enlargement; next day, the temp. rose to $105^{\circ}\cdot8$, p. 110, small, soft, regular; resp. 40, chiefly abdominal; bowels opened once only, in bed: abdominal symptoms subordinated by chest symptoms, and finally unconsciousness. At autopsy—cerebral hæmorrhage and extreme pulmonic congestion, in addition to intense vascularity, with extravasation, at end of ileum and beginning of large intestine, where the mucous membrane was also covered with granular exudation: see Chapter IX.

The condition presented in the above cases was probably never recovered from, and it may not have been approached except in casualties: the following instance doubtless belongs to a similar series.

b. Inflammation at termination of ileum; death at crisis of first relapse from other complications: deep jaundice.

CASE LXXXVI.—M., 30, admitted towards close of invasion (t. 104° , p. 120) which declined by lysis (t. $94^{\circ}\cdot6$, p. 88), general condition the typhoid, much tenderness and fulness in both hypochondria, urine and stools passed in bed; hiccup, florid tongue, looseness of bowels (?): yet rallying was so prompt, that after 8 days he became convalescent in appearance. Then the relapse came on, somewhat gradually, and though prolonged for 6 or 7 days, the pyrexia was remittent and never exceeded 103° , p. 120: the blood-contamination being again abundant: at its beginning there was some headache, tongue moist, bowels regular, liver and spleen hardly affected; then the man became depressed, jaundice increased, no local urgent symptoms, lumbrici vomited; the pulse was rather low and very feeble, becoming imperceptible near the close, when looseness of the bowels again appeared, with intense depression, and death at critical fall. At autopsy besides splenic infarcts, hæmorrhages in the arachnoid, pericardium and elsewhere, there was seen congestion and inflammation of the ileum, concentrated around Peyer's patches, especially at one place about a foot from the ileo-cæcal valve: higher up the valvulæ conniventes being of deep blood gradually diminishing as the jejunum was approached: I noted that Peyer's patches were raised, granular in aspect, defined, of deep blood hue, the mucous membrane around being highly congested and blood-stained, over a space of several inches.

It is worthy of notice that the above case, being long enough under observation, manifested all the chief characters of true relapsing fever, and this without unusual pyrexial irregularity or complication: a similar remark applies to the following instance, excepting that the enteritis was more deferred, being also attended with a febrile perturbation, which represented one of the many forms of 'secondary,' non-specific fever witnessed in both man and the quadrumana.

Two animals dying of such fever three days after the spirillar crisis, presented widely diffused enteritis as the chief anatomical lesion.

CASE LXXXVII.—M., 30, a cachectic subject, with bleeding gums, admitted towards close of invasion in a very low state: blood-contamination abundant: t. 100° , no stool for 5 days, some cough, liver enlarged and tender; after the acme, defervescence by moderate crisis, slightly delayed reaction and then a normal level for 7 days, pulse rather quick and rheumatic pains being troublesome, but rallying was fair and the appetite became good. Relapse of spirillar fever then came on: it was pronounced and prolonged for 6 days, max. t. $105^{\circ}\cdot4$, p. 128, a decided critical termination; the usual symptoms were present—vomiting, considerable pains in the thighs and joints, thirst, headache, fulness but not pain in hepatic and splenic regions, bowels regular. Secondary fever immediately succeeded to crisis attaining its max. on 2nd day, $105^{\circ}\cdot2$, p. 142, then declining yet persisting at a lower level (t. $101^{\circ}\cdot2$, p. 120–130) for 6 days longer, when death took place by exhaustion: during this

time the blood-spirillum was absent ; at first bowels costive as at the crisis, thirst ; then on 3rd day, very low and pale, t. 101° , p. 130, feeble and running, liver and spleen enlarged, tongue dry in centre, rough, red, shrunken, several petechial spots on the chest ; the typhoid state threatening : so next day, sordes around the teeth, one stool at night, heart's sounds feeble, yet distinct : 5th day, some of the spots have disappeared, 4 stools passed in bed at night, said to be thin ; 6th day, is losing strength, fulness and tenderness in l. hypochondrium where the firm spleen is to be felt, one thin stool passed in bed, breathing slow, corneæ dull and conjunctivæ wet, skin covered with clammy crust ; the blood now showed no fibrillation, some large granule-cells were present, ordinary white corpuscles very few (Mr. S. A.) ; I had on the first day of this rebound noted the red discs also to be scattered and shrunken, the blood seeming to be 'in a dying state' : 7th day, one thin, greenish stool passed in bed, abdomen retracted, pulse feeble though regular ; breathing chiefly abdominal and shallow, corneæ bright, pupils normal ; the body emitted an offensive odour : 8th day, m. t. $100^{\circ}\cdot6$, p. very feeble and irregular, some tenderness in hepatic and splenic regions, sudamina seen in axillæ and at elbows, two scanty, thin, and greenish stools passed in bed ; he is too weak to turn on the side, some coarse respiratory sounds heard on r. side of chest : death soon followed. At autopsy—corpse emaciated, tissues slightly tinged yellow, some lobular collapse at edges of both lungs, at their lower lobes especially ; heart flabby, substance rather pale : liver 47 ozs., aspect not unhealthy, permeated by tar-like blood : gall-bladder contains a thin, pale liquid resembling scrofulous pus ; spleen 12 ozs., pulp not abnormal in aspect, milk-white patches on surface behind : kidneys 4 and 6 ozs., cortical substance pale, brownish in aspect : the brain was wet, but firm, moderately congested ; the mucous membrane of duodenum and beginning of jejunum deeply stained yellow : the ileum presented several areas of intense vascularity (see Chapter on Morbid Anatomy, *sub loco*) : the colon thickened granular and vascular. *Vide* Chart 23, Plate VI.

29. *Diarrhœa*.—Was regarded as a complication where of unusual severity or incidence, as sometimes happened during pronounced specific pyrexia, or just after the crisis ; and though seldom excessive or persistent, its weakening or depressing effect was then often apparent.

Native patients coming in late occasionally gave a history of diarrhœa, which was not verified by symptoms actually seen : women, weak subjects, the intemperate, opium-eaters, those troubled with worms, cases ending by lysis and the other severer forms known as 'icteroid,' were commonest so affected : and it has been noted with hiccup, parotitis, lung inflammations, and marked splenic and hepatic implication. Most fatal cases at the end showed involuntary signs of quasi-diarrhœal flux, and sometimes this might be termed colliquative.

In general, the connection of diarrhœa with a tender and either distended or flat abdomen, was a point for remark ; but the data are not uniform in their indications, and this might be anticipated on considering the several possible causes of excessive alvine flux. Such were mentioned in the clinical analysis (Chapter III.), and here I allude to the association particularly with one or other of the intestinal lesions described in the section on Morbid Anatomy : even in the severe cases, however, the evidence varied—thus, a hospital matron seized and dying during first attack at the onset of moderate specific pyrexia had purging and vomiting (reported as bilious) with a distended abdomen ; rest was disturbed and in the snatches of sleep there was noted grinding of the teeth ; santonine being administered no worms appeared and much depression followed, without checking of the fever ; death 3 days later, and at autopsy were found petechial extravasations in some cerebral convolutions, with disseminated congestive patches and extravasations in mucous membrane of stomach and intestines ; the

combined morbid changes sufficiently accounting for the symptoms mentioned. On the other hand, a young man simultaneously infected in hospital and dying under similar conditions, had diarrhoea as a prominent early symptom ; abdominal fulness and tenderness were noted towards the end ; at autopsy the stomachal membrane alone was found congested : and, again, in a man of 35 (contagion in hospital) there was copious diarrhoea before the acme of invasion with much tenderness in the upper abdominal zone, yet no general distension ; death took place with rebound after a marked crisis, and on section but little morbid change was apparent in stomach and intestines.

Discrimination of simple diarrhoea from the enteric discharges of typhoid fever, was not needed during the epidemic ; it might, however, be difficult from local signs only : the transitions to dysenteric forms were many and gradual, doubtless representing a real assimilation. Once, after a latent second relapse brief diarrhoea of choleraic form came on, which besides observing a certain periodicity was associated with neuralgia and abdominal uneasiness, resembling the occasional relics of spirillar infection.

30. *Dysentery*.—Tropical dysentery in all* forms and degree being (like 'fever') very common in Bombay, its occasional concurrence with spirillum fever might be anticipated, without the supposition of direct causal relationship : and as acute specific infection often passed through all its stages in malarious subjects without any symptom of dysentery arising, such instances afforded clear proof of its essential independence. Nor could it be said that the new fever predisposed to 'colitis,' more than equally pronounced malarious pyrexias ; for example, in 66 autopsies of fatal cases, inflammation of the large intestine was not present more than a dozen times, if so often ; and then to a moderate degree, which surprised me by its limitation and quasi-incidental character. See the Chapter on Morbid Anatomy.

During 1877-78, true dysenteric symptoms were present in about 5 per cent. of surviving cases, and 10 per cent. of casualties ; this estimate being only approximative, for often it was impracticable to distinguish their milder degrees from the diarrhoeal.

They were rare during a first febrile attack, but not after its critical close ; and they were seen both during and after the pronounced first relapse : amongst pauper patients after discharge dysentery was occasionally heard of as a sequel, and then probably it was not uncommon as a cause of death. I have known it supervene only at the first crisis, and only with the relapse, being absent at other times ; it might be strictly limited to acme, fall or post-critical period, and this well-defined occurrence was sometimes striking. Acuter symptoms might be marked during specific pyrexia ; they were not seen after its cessation when depression comes on, the characters then being those of mild or sub-acute local inflammation. A few patients were admitted with pronounced dysenteric symptoms, combined with high spirillar fever : other local signs, however, were present (as hepatic or pulmonic), and since autopsy was not practicable I am unable to state the probable share each lesion took at the close of illness. Except as a chronic sequence, this complication never seemed alone to cause death: its ordi-

nary duration was a few days, the acuter symptoms being noted for only 24 or 48 hours (as at critical epochs), or lasting 4-5 days, seldom longer than a week.

Commonly there was some degree of attendant pyrexia during the usual post-critical periods, and in severe cases this symptomatic fever (like that of some pneumonias) might be directly continuous with the spirillar, blood-scrutiny alone enabling a distinction of dates to be made; but in ordinary cases, the rebound or secondary fever was deferred for a day or more after critical decline, the reaction not being quite so prompt as with the hepatic and pulmonic complications doubtless oftener beginning at acme. Dysenteric pyrexia has not a fixed character, though tending to remit deeply or even intermit; usually it is brief and mild; and it may be disproportionately low, even with troublesome bowel-complaint.

The pulse has been noted as unusually slow with this complication.

Dysentery often attended the severer febrile attacks in Mussulman weavers especially, also in those ending by lysis, in subjects showing malarious and scorbutic cachexia (there being, however, no rule here); also concurrently with other complications, local or general.

Its relation to consecutive hepatic abscess has been mentioned; and, as well, its presence amongst hospital patients infected in the wards, who were not, however, thereby predisposed to infection. Dysentery previously existing was not exacerbated during spirillar fever.

The acute and sub-acute forms were commonest in young people, the chronic and sequelar in the old: both sexes were implicated.

Diagnosis rested on the frequency, painful character and muco-sanguineous aspect of the evacuations, together with localised abdominal tenderness; and though usually facile, there were many conditions interfering with proof. Sometimes the stools contained little more than a bloody liquid; with no visible signs of hæmorrhoids.

The prognosis depending upon urgency of the symptoms: the typhoid state and extreme debility being most of all unfavourable.

The pathological character of the bowel affection under notice is open to question, on account of the likelihood, always present, of ordinary dysentery preceding or mingling with the specific lesions, if such there be: besides, the forms of true tropical dysentery have not yet been accurately discriminated. The brief duration of spirillum fever, especially in relapses, does not seem to favour destructive bowel-lesion; and the singular brevity of some quasi-dysenteric attacks further points to the absence of such organic changes, as pertain to ordinary tropical forms. Occasionally the symptoms seemed to be of critical character, concurring with specific blood-changes and acme of fever.

Illustrations.—An ordinary form is described in Case No. X. as occurring at the close of invasion; here some pharyngitis also followed; neither symptom recurred with the relapses. CASE LXXXVIII.—Acute dysentery with spirillar infection: M., 13, one of a whole family ill, admitted probably at first relapse, t. 105°·4, p. 120, spirilla many; some jaundice, tongue coated at middle, florid at tip and edges; general abdominal tenderness and some hepatic enlargement: a small quantity of albumen in the urine was noted; the pyrexia daily intermitted, but did not cease with the disappearance of blood-contamination. The typhoid state came on, vomiting, an offensive odour from the person, pallor and anxiety of expression, persistent enlarge-

ment of the liver and death by exhaustion 4 days after apparent crisis : the dysenteric symptoms were much pronounced, the stools being numerous and of characteristic aspect. The lad's chart is reproduced as No. 27, Plate VI. : it shows the paroxysmal character of the fever, which throughout exacerbated with chills and abated with sweats. Autopsy was not allowed.

CASE LXXXIX.—Dysentery without change of temperature. M., 35, two days after fall of first relapse, which was attended with much depression and active delirium ; t. 98.4, p. 100, profuse sweats in the night, aching pains, and a burning sensation in the palms and soles ; the bowels, previously regular, now frequently moved, the stools assuming a dysenteric character ; abdomen somewhat retracted, and tender on pressure along the colon ; this bowel complication lasted 9 days, gradually subsiding without other change ; the temperature remaining at normal level and pulse but slightly quickened.

For a striking instance of dysenteric symptoms at acme of first relapse, concurrent with extreme depression, see CASE XXI., Chapter III. 'Crisis.'

In a lad of 15, sharp dysenteric symptoms came on at the deep fall of relapse, a mild febrile rebound attending (max. t. 100°, p. 90) ; the loss of blood had probably been considerable, and he was so reduced that the evacuations were passed in bed. This state lasted three days, after which convalescence began.

Case XC., below, is an instance of ordinary Dysentery apparently supervening on specific infection ; its cause being possibly other than such infection, and the conjunction therefore incidental.

31. *Hepatic Congestion, Inflammation and Degeneration : Hepatic Abscess.*—The frequency and variety of morbid symptoms and changes connected with the liver, in uncomplicated cases of spirillum fever, have been already mentioned : the particular association of these phenomena not being always possible, in the absence of preciser testing of the blood and excreta than I could practise. It was, however, plain that in this disease the more prominent hepatic signs have a more limited significance than might be anticipated ; and it came within my experience to note, at febrile acme, such enlargement, pain and acute tenderness in the right hypochondrium, as in other fevers would infallibly excite grave apprehension of acute hepatic inflammation with its consequences, yet all would subside after a few hours at critical deferescence, nearly as rapidly as the attendant blood-contamination and pyrexia ; or, at most, some fulness and soreness might persist for a day or two. Judging, also, from the autopsic revelations acute hepatitis, in its ordinary meaning, is not a usual complication of spirillar infection : remarkable gland-changes do, however, occur, and it may be supposed that these, in their degree, entail the acute local disturbances named. Fatty degeneration ensuing has a distinct clinical significance, even if only accounting for the long persistent anæmia of some patients ; as, for example, the case of an adult man sinking exhausted 8 days after the relapse, in whom, besides some opacity of the arachnoid, diffused patches of such degeneration of liver-cells were the most obvious morbid phenomena. Further changes will be alluded to under the heading of 'Sequelæ.'

Another point of interest is the possible connection of Liver changes with Secondary fever.

Notes of 4 selected cases are before me, all of youths 12-19 years of age, who recovered promptly, though displaying high and sustained pyrexia of non-spirillar kind : once this followed the invasion immediately, and three times the first relapse at intervals of 3 days (twice) and 22 days : the hepatic symptoms were invariably mild, and not noted at first onset of the fever, they did not last longer than it, or leave

any trace behind. I was unable to decide if they had not followed the pyrexia or, at least, been due to the same cause as it : yet, in the absence of other likely cause of fever, I acquired the impression of its being due to parenchymatous hepatic lesion.

A priori it is highly probable that the molecular changes undergone by the liver would occasionally result in residual phenomena, short of death ; and such phenomena might be febrile in character. Some such supposition is needed to account for the striking absence of hepatic complications of an ordinary kind, after acute attacks of spirillum fever : the conditions of illness being such, as almost inevitably to entail symptoms of some form.

As evidence of the little tendency there is here to ordinary hepatic complication, I will quote an instance showing that when suppurative inflammation does follow spirillar infection, there is found other sufficient cause for its occurrence.

CASE XC.—F., 25, a weak subject, admitted at the end of invasion ; no spirillum in the blood, red discs shrunk and misshapen, coagulation imperfect. With a sharp rebound following some cough and abdominal uneasiness, there ensued 4 days of quasi-normal temperature and pulse, when the Relapse suddenly set in ; pyrexia now high sustained and of 7 days' duration (spirillum seen on 2nd day), much depression and a tendency to the typhoid state, without indication of localised disease ; crisis moderate, the alvine evacuations few, clay-coloured : then followed a series of pyrexial exacerbations, intermittent in character, max. t. $102^{\circ}4$, p. 124, much debility, diarrhoea with foetid stools not resembling those of dysentery and not becoming more unhealthy towards the end, though purging continued unchecked ; this consecutive pyrexia ceased after 14 days, and 4 days later she sank exhausted. The blood was examined thrice during this period, and as only some peculiar bacterial rods were seen, together with large granule-cells and groups of free granules, besides free protoplasm, it was presumed no second relapse had happened. At autopsy, brain pale, lungs healthy, long, loose, dark coagula in cavities on both sides of heart, spleen, kidney and small intestine healthy ; mucous membrane of large intestine throughout presented many scattered, small, superficial ulcers, some of which were sloughy in aspect, also a few patches of circumscribed redness ; liver weight 2 lbs. 8 ozs., aspect healthy with exception of an abscess the size of an almond, situated near its posterior border, not furnished with a pyogenic lining, contents healthy-looking pus : prosector Mr. B. A. I supposed the small abscess to have been the result of portal pyæmia.

That the spirillar infection may supervene upon hepatic suppuration is shown in the following Chapter.

32. *Jaundice*.—Commonly apparent, this symptom was sometimes striking and at others barely perceptible. The milder degrees of jaundice were simulated by malarious cachexia or anæmia, and yellowness of the native brown skin, or even of the nails, not being always observable, their detection in the eyes might be obscured by increased vascularity of the conjunctivæ ; the aspect of the urine might be fallacious, and I doubt not jaundice was occasionally overlooked, or regarded as only febrile sallowness. Sallowness of the skin once seemed to precede a yellow tinge of the eyes, and a few cases (not all severe) were seen presenting a peculiar fiery aspect, due to superadded injection of the conjunctivæ.

Jaundice was only an occasional symptom, and much less frequent than hepatic or epigastric derangements ; it was most prevalent during pyrexia, and at the primary attack ; and also in fatal cases. Its occurrence being contingent, characteristic examples of spirillum fever, both surviving and fatal, went through their whole course (the latter revealing

typical organic lesions) without exhibiting any perceptible sign of its presence.

A general estimate of surviving fever cases alone, gives the proportion of 79 in 517, or about 15 per cent. of instances of jaundice. During the invasion-attack this symptom varied in frequency from 12 to 22 per cent., being commonest at later periods of the epidemic and amongst Mussulman weavers and low castes; less frequent amongst Hindoos (including most famine-immigrants) at the height of public sickness. During the relapse, jaundice was noted in only 6 to 9 per cent. of all cases; this difference is striking. The data concerning second and third relapses show, at least, equal infrequency of the symptom. Apyretic periods are not included in the above summary, because jaundice rarely seemed to arise at these stages, and seldom persisted longer than a few days after the fall: it is, however, interesting to notice that the symptom occasionally became intensified in the first, second or even third day afterwards; and in 2 or 3 cases, made its first appearance just after the crisis.

The list of fatal cases, embracing 95 instances, showed 52 of jaundice in some degree, or a proportion of 56.5 per cent.: this is the main fact to be noted here, viz. that the symptom was much more frequent in severe than in mild cases, and I may add that it was usually of intenser form.

The grand total of both survivors and the dying, furnished 612 cases with 131 exhibiting jaundice in some degree, or a proportion of 21.4 per cent.

Febrile stages.—Invasion: a yellow tinge may be observed on the third day, seldom earlier and usually at a later date, becoming more frequent towards the end of the attack (*e.g.* in 35 instances 10 times, at the acme or incipient fall): it may, indeed, be noted first with the critical: decline (one-sixth of cases), and commonly augments with its course to the end. Prolonged attacks showed a larger proportion of jaundice-symptoms than those of shorter duration (*e.g.* 2 in 3 of estimated 11 days' duration); and also those terminating by lysis.

Relapses.—Slight jaundice may be detected at the beginning of the attack, but it was usually not until the third or fourth day that this symptom became distinct: these days may correspond to the acme of attack; that there is a tendency to come on now in the relapse, as in the invasion, appears also from the fact that in a series of cases, jaundice supervened 3 times in 10 with the critical fall.

When this symptom had existed in the invasion-attack, it might reappear early in the relapse, or prove altogether deficient; although the recurrent event be fairly developed, and the jaundice of unusually pronounced degree at first attack.

Respecting relapses of briefer duration, these were seldom attended with jaundice; yet in one example (isolated paroxysm) there appeared this symptom now for the first time, the liver during the invasion having been much affected. The contingency of jaundice at these periods, further appears from a case in which its persistence was noted throughout the first apyretic interval and into the relapse, without then undergoing any exacerbation or interruption in its slow abatement.

At all febrile periods, jaundice did not show any certain proportion

to local hepatic signs ; and though these were evident, generally, along with it, yet they might be wanting.

In severe cases there was frequent vomiting of bile, but not always ; and it occasionally seemed to me as if bile were ejected in this way, which otherwise might have entered the circulation.

Non-febrile stages.—First interval : the presence of jaundice was limited to the first few days, in a long list of cases ; the proportion of cases gradually subsiding until the fourth or fifth day : when persisting much longer, this symptom has been noted as intermittent. I have already stated that it first appears or becomes intensified, sometimes, after the critical fall ; and the event is not necessarily of bad augury.

Second interval : Jaundice never now arose afresh, and was seldom seen after the fifth day in uncomplicated cases : about the time of second relapse, it was once seen in mild degree, on arrest of some diarrhoea.

Lesions of the Liver in Jaundice.—In 56 unselected autopsic records, the clinical history showed this symptom 29 times (51·8 per cent.) with enlargement of the liver 21 times, and in most instances the other characteristic hepatic changes : there was no jaundice in 27 cases (48·2 p. c.) with enlargement 16 times, and frequently these other changes. This statement will suffice to indicate the absence of any peculiar organic alterations, in either series ; just as during life there were no special symptoms.

That the autopsic series differed from the main, only by their greater severity, will be apparent on further analysis : thus, in general, jaundice was present at the same periods of disease in both survivors and dying, being commonest at invasion-attack (24 in 56) towards and at its close ; seldomer at relapse (2 in 7) ; and rare in apyretic intervals strictly so-called. A seeming anomaly was the rarity of jaundice in deaths at close of first relapse, but the data are very few ; and I now proceed to show in detail that this symptom was not very strictly related to organic changes. From two considerations it is evident that some such relationship obtained, for presumably organic lesions being most severe in fatal cases, with them jaundice attended in like proportion (56 p. c. as compared with 15 p. c.) ; and, further, the general derangement of the liver (as indicated by its increased volume) was somewhat more marked in the jaundiced than in the non-jaundiced cases (72·4 p. c. to 59·2 p. c.) especially at first attack, which is the most characteristic period of the fever.

At both invasion and relapse, fatal cases were seen in which the liver was greatly affected by acute fatty degeneration, and still without jaundice as a symptom during life ; thus, of 13 instances fatal at invasion, liver larger than normal in 8 ; and of the 5 negative autopsies at relapse-period, liver large, or very large, in 4 : the notes of these cases are before me, but it is not required to quote them ; and all that I had learnt from their study, did not enable me to surmise why jaundice should at one time be present in marked degree, and at another equally conspicuous by its absence. I have elsewhere quoted an example showing that the liver at the end of fourteen days after a febrile attack marked by intense jaundice and subsequent severe relapse, may still not be enlarged or altered in general aspect ; and this striking instance would

indicate that other fatal changes (possibly of the blood) are not necessarily attended with marked derangement of the liver.

In several earlier autopsies, the condition of the gall-bladder and bile-ducts was especially scrutinised with reference to biliary obstruction, and the results were always of negative import: I do not recollect one instance in which there was impediment to the flow of bile; and extremely few in which a deficiency of the secretion was manifest; in perhaps the most striking instance of altered or defective condition of the bile (its appearance was compared to thin scrofulous pus), there was but slight jaundice, and the stools were green-tinted.

The form of jaundice witnessed in these fever cases, therefore, was not that usually attributed to mechanical causes. Bile-acids were always present in the urine when searched for: and though long constipation was a usual initial symptom, yet the stools were hardly ever paler than natural.

Conclusion.—Jaundice though a most striking symptom when fully displayed, was commonly present in moderate forms, which graduated to the quasi-normal aspect on the one hand, and on the other to an intensity of tint hardly to be excelled: these degrees had no absolute relation to severity of illness; thus, in 29 fatal cases the jaundice was estimated as doubtful 2 (once invasion and once first interval), as slight 6 times (all invasion), as decided 13 times (invasion 9, first interval 2, relapse 2), and including 4 instances of so-called typhus form, as strongly marked 8 times (invasion 7, relapse 1). Similar degrees were noted in survivors, but not so large a proportion of the marked cases. Further analysis shows this symptom to be connected with the febrile state, and chiefly its later period or acme, with ensuing crisis, when also the acute liver symptoms and lesions already noted present themselves.

The limited group of cases displaying that association of deep jaundice and complete typhoid state which is understood by the term *Typhus biliosus* is alluded to in Chapter VII., Sect. 2; at present, I observe that the distinction of this group is somewhat arbitrary.

33. *Affections of the Spleen.*—It is noteworthy that the more striking changes known to occur in the spleen during fatal spirillum fever, were rarely indicated by corresponding change or increase of the ordinary symptoms referable to this organ; and I am therefore unable to state whether 'infarcts,' for example, ever occurred amongst the survivors, there being an absence of local signs presumably indicative of their presence, and the attendant general disturbance being possibly due to more than one cause.

The available clinical records of severer cases admitted only to die, were often so brief that little particular information is afforded by them; yet as in the longer seen cases, special fulness or tenderness of the spleen was seldom considerable, or in excess of hepatic changes and other complications, I am led to suppose that practically it may be impossible to learn accurately the degree, and still less the character, of these organ-changes: nor is the common typhoid state itself indicative of more than lesion of blood or tissues generally.

Autopsies showed also an occasional diffused softening of the spleen,

which could not be associated with local signs : limited softening, as of infarcts, may have taken place amongst survivors without being indicated locally ; localised hæmorrhages and even some degrees of inflammation are also possible without other sign than those commonly noted of enlargement, pain and tenderness.

It is striking, too, how rarely recent peripheral splenitis was found, the following being the best marked, yet little pronounced, instance :—

CASE XCI.—M., 20, gave a history of a defined febrile attack, followed by the dysentery complained of on admission ; the remark was then entered that the case seemed one of 'starvation' rather than of disease : after 10 days of gradual amelioration specific fever suddenly set in, which lasted only 3 days, though highly pronounced, and was followed by prompt secondary fever and death. The symptoms were not peculiar, there was much depression throughout, no cough or sputum, purging at the last. Besides showing many infarcts, the spleen was streaked with a little fresh lymph at the borders of a fissure ; there was reddish serum in the left pleura and incipient pneumonia of the left lung, chiefly in upper lobe : right side of chest unaffected : liver large, pale, smooth ; mucous membrane of colon thickened, granular and vascular, superficial erosions in the rectum. The connection here noted of splenic and left pulmonic changes was occasionally indicated amongst survivors, and corresponding pains in the left shoulder have also been observed.

The observations regarding secondary fever submitted under the heading of Liver-changes, will, in part, apply to affections of the spleen : it being, in my opinion, probable that consecutive splenic changes do occur, which are manifested by consecutive fever.

34. **Urinary Organs.**—Retention and suppression of urine : albuminuria : renal dropsy.—Towards the close of pronounced primary attacks and in complicated cases, surviving as well as fatal, it was not very unusual to learn that the urine had been passed during night involuntarily, and the urinary bladder might be found distended ; at the same time, fæces might or might not be evacuated, these symptoms being such as noted in other severe diseases. Retention of urine may supervene also with debility, some days after cessation of fever.

An actual suppression of urine was doubtless rare, even of brief duration ; yet at the critical fall it clearly appeared that the quantity of urine formed was for a time diminished : *vide* Chapter III. of this Section.

Renal complication.—At autopsy it is found that during fever, the kidneys are similarly and as often implicated as the liver and spleen ; and clinical observation showed that after cessation of fever, the renal changes doubtless persisting were as obscurely indicated as the splenic or hepatic. Even repeated attacks of specific fever did not entail lesions detectible at the bed-side. It seems almost anomalous that, whilst at high pyrexia the glandular epithelium becomes swollen and granular, yet the urine remains pale, light and clear ; and briefly, as at present informed, I am unable to name any lasting, serious or peculiar renal lesion which is certainly due to the spirillar infection.

In some severer cases examined with care, during life the urine furnished no evidence of acute nephritis, when after death the cortical uriniferous tubes were found, in parts at least, distended with turgid and clouded epithelium.

Respecting deferred sequælar states, fatty degeneration of the gland-

cells is the only change known to me ; and in its production other influences, such as previous state of patient, malaria and intemperance, may have had a share. Late autopsic experience demonstrated that amongst the poorer classes at Bombay, chronic renal lesion was far less common than in the lower classes of large European towns (*e.g.* London and Berlin) ; thus, out of 74 consecutive necropsies of fever subjects, only 2 revealed the presence of granular kidney.

Albuminuria.—I have elsewhere remarked that at critical stress of spirillum fever, a trace of albumen often appears in the urine : but tubercasts or granular epithelium, were not seen by me at this time ; nor did the presence of albumen appear to be associated with urgency or peculiarity of symptoms.

The following case is worthy of record from its complicated character, the form of pyrexia presented, and the state of the blood :—

CASE XCII.—M., 40, Goanese cook, of cachectic aspect and intemperate habits, was admitted with fever and anasarca reported of 15 days' duration ; there was present also pains in the limbs, basal pulmonic congestion, feeble action of the heart, little perceptible change in liver and spleen : urine scanty, high-coloured, turbid, 1012, acid, albumen $\frac{1}{8}$ vol. : the man was found to have tænia. Subsequently the urine became pale, 1007, albumen very scanty ; the anasarca for a time increased and then diminished, when fever returning his friends took him away, 24 days after admission. During his stay, the pyrexia daily remitted strikingly, or intermitted : it gradually declined to normal from 104°·6 during the first six days ; for three more it remained near normal, and then a second series of 6 paroxysms occurred, also diminishing after the first ; there next followed an apyretic interval of about seven days, when again smart paroxysmal fever occurred, and four days later the patient left in a bad state. Blood-scrutiny was made at the febrile epochs, with negative results except on 2 or 3 occasions, when a small active organism comparable to an immature spirillum was detected : prior to these also some vacuolated protoplasm, and afterwards large nucleated, pale cells. I saw this patient frequently and regarded his case as one of spirillum fever modified in some way, and attended with renal disease probably not recent.

Post-febrile Albuminuria.

CASE XCIII.—M., 25, admitted towards close of invasion, which declined by lysis, and, though uncomplicated, was accompanied with a dry low state ; at the end, purplish spots and œdema of both feet, looseness of bowels, severe pains in limbs ; 4 or 5 days later retention of urine with pain in the bladder, and a small amount of albumen in the pale, clouded urine, sp. gr. 1009, phosphates deposited, the symptoms indicating also slight cystitis: temp. slowly declined and improvement ensued under treatment by strychnia. There was at this time some desquamation of the cuticle of the limbs: the specific infection had been marked, and there were some blood-signs of a latent relapse with the above-named symptoms.

The association of albuminuria with abortion was not enquired into.

The not infrequent brief post-febrile œdema of the feet (dorsum) and sometimes the hands, face or trunk, was not found to be associated with albumen in the urine.

35. **Abortion.**—This event was noted in the first group of fever-patients coming under notice at the Relief camp in April 1877, and subsequently 4 or 5 times ; omitting the unproven instance (though a highly probable one), the ages of 5 women aborting ranged from 16 to 30 years, date of event 4 times during invasion, viz. thrice at about mid-period and once near crisis ; once it occurred 5 days after an

invasion-attack not much pronounced, but terminating by lysis and attended with depression, jaundice and delirium ; no further accident happened, and all patients recovered. The fever, as seen, was not unusually high, but tended to be irregular, and rapidity of pulse was a common feature : relapses occurred 4 times certainly, and once a second recurrence : blood infection was demonstrated always, and in the cases I saw abundantly—once the blood being amazingly filled with clusters of active spirilla. Complications were rarely present, and none unusual followed, as a rule : the husbands of two of the women were known to have fever.

The expelled foetuses were of ages estimated at 2 to 6 months, only the oldest showing brief signs of life : no unusual aspect was noted in these bodies : state of the placenta generally unknown ; but one example I saw appeared quite normal. Examination of the foetal blood did not reveal the presence of the spirillum on two occasions, although the maternal blood might contain several parasites.

From the notes preserved it appears that, in general, the fever was of rather low type : great extremes of temp. not being seen here : the following memoranda refer to the last instance met with.

CASE XCIV.—F., 30, resident in a fever quarter, health fair, 6 months advanced in her fifth pregnancy : admitted on 7th day of first attack, m. t. 104° , p. 120, some hepatic tenderness and splenic fulness, bowels constive, much headache, no sleep, pains in the limbs ; bronchitis, uterine uneasiness and the os a little dilated. Next day ; had been restless at night, fever persisted and the uterine pains increased ; e. t. $104^{\circ} \cdot 2$, p. 128, r. 30, headache severe ; blood loaded with the parasite which also appeared in the sputum ; labour was completed the same night, expulsive efforts being forcible and after-contraction complete ; the foetus is said not to have breathed, but the heart's action persisted for nearly an hour. Next morning I examined the foetal blood going to and returning from the placenta, without perceiving the spirillum ; the liver was pale, its blood containing nucleated cells and liberated nuclei, the spleen was very small, not congested but compact as a piece of flesh, nothing peculiar being noted in its blood : a minute fragment of clot from the maternal side of the placenta contained some quiescent spirilla, though not so many as were seen in the woman's blood. The acme of attack probably occurred the night of abortion, for in the m. t. 105° , p. 140, resp. 38 and dyspnoea considerable, the patient appearing distressed : relief shortly ensued with copious sweating : secondary febrile paroxysms appeared for a time (blood-scrutiny negative) without marked local signs, and the woman rallied in a remarkable manner after cessation of the specific pyrexia.

Extreme derangements of the Menses during illness, did not come under notice.

CHAPTER VI.

ANTECEDENTS AND SEQUELÆ.

Antecedent Sickness.—Amongst 31 instances of hospital patients acquiring spirillum fever in the wards, 4 were healthy infants or children infected from fever-sick parent or family ; of the remaining 27 persons, 11 were admitted for ague or remittent fever, 5 for diarrhœa or dysentery, 4 for organic lesion of nervous system (all women), and 1 under each of the following heads—chronic bronchitis, dropsy, hepatic abscess, lumbago, scurvy, constitutional syphilis, debility after cholera. Total males 20, females 7 (showing a decided preponderance of women proportionately to numbers of sexes in the medical wards) ; general state of males, 12 bad, 8 fair ; of females, all in much impaired health. Mean age of both sexes 30 years : time in hospital prior to seizure, 10 to 77 days.

Of the specific attacks, 11 were known to be relapsing (a second recurrence not seen) : of the remainder, 10 single attacks ended fatally, 10 were not apparently followed by a recurrent-attack, but some of these left hospital too early to render it certain a relapse might not occur. The mortality was rather over 30 per cent., and equal in both sexes : this datum seems to indicate the unfavourable influence of bad health, prior to infection ; but age was also concerned, for the mean age of the dying was either considerably beyond the mean or exceptionally below it. 7 of the relapses were seen in the 11 more malarious subjects, none amongst the 5 dysenteric, 2 amongst 4 palsied women ; no relapsing case died, all the 10 casualties happening at invasion-attack.

There being no absolute uniformity of illness amongst ordinary subjects, it becomes difficult to estimate correctly the predisposing or modifying influence of antecedent sickness ; and on considering the above instances I am unable to perceive a rule determining their comparative frequency and intensity. It might appear that a malarious taint predisposed to spirillar infection, but conditions are here complex, and the cases too few for special inference. Some of these acquired illnesses were striking from their brevity and irregularity of pyrexial manifestation, others were almost typical, and a few unusually pronounced : on the whole, the variety of fever seemed greater than amongst ordinary hospital patients ; yet interesting as is this datum, much stress cannot be laid on it, since hospital admissions may not include slighter attacks of fever staying at home, and did not probably include others speedily ending in death outside.

It happened that the above patients were all free from febrile, or other acute symptoms, at the probable date of their infection ; and

hence there are no means of judging if the spirillar contagium can be implanted in the system, at a moment when other acute disease is present. From many data, it is known that once implanted, its development is not likely to be materially checked.

Sequelæ.—Experience has shown that in time (as well as form and degree), it is impossible to sever abruptly the rarer events from the usual : however, phenomena supervening distinctly after close of the last febrile attack may for convenience be regarded as sequelar. They are commonly chronic in character, but are liable to exacerbation and repetition.

The sphere of observation at Bombay was not favourable to discrimination of the longer deferred effects of spirillar infection, owing to both rarity of cases sufficiently followed out, and the wide prevalence of another cachexia (the malarious) amongst the poorer classes. In a practical sense, amongst the surviving majority complete restoration to health was the rule : some instances were known of patients dying after their discharge from previous organic disease probably accelerated by fever-illness, or from dysentery following the fever ; and a few were re-admitted to die from bowel-complaint, also seemingly colliquative. In such cases it is difficult to apportion the influence of specific infection *per se*, and hardly less so in the milder sequelar affections ; for other deteriorating influences might always be concerned. Bearing in mind the probable pathology of the disease under consideration, the observer will apply general rules according to custom and personal judgment ; pending the receipt of information adapted to strictly accurate decision.

From what was seen at Bombay, it appears that the spirillar sickness may exceptionally inflict both lasting lesion of body-nutrition, which if originating in certain abdominal organs, becomes manifested by general debility, emaciation and anæmia ; and also lesion (essentially nutritive, too) of particular organs, as of the brain (*vide* CASE LVI. Chap. V., and also LV. which connects the two series here indicated).

As examples very significant of mortality amongst the indigent and aged, during public dearth and resulting disease, the following cases will suffice :—

CASE XCV.—M., 25, a sailor, tramp from Madras, 8 days in Bombay and suffering 12 days from fever (which therefore seemed to be acquired before his arrival in town), admitted May, 1878 : it happened I then saw the man and ascertained the presence of abundant blood-contamination ; otherwise, as the febrile symptoms were slight and not peculiar, subsiding next-day (spirillum now absent), the true nature of the case might have remained unknown. A highly cachectic subject, jaundiced, both liver and spleen enlarged. For 34 days there was an almost level temperature, about the normal, pulse variable, and he seemed to be slowly gaining strength, when smart fever came on, deeply remitting, and in 2 days more he died (blood not peculiar in aspect at this time). At autopsy there was found incipient pleuro-pneumonia on right side, liver and spleen of enormous size, the kidneys probably fatty. The man himself allowed his habits to be intemperate, and he had long been exposed to malarious influence.

CASE XCVI.—M., 50, a miserable subject brought by the police in a low, delirious state with moderate fever (? end of invasion), but copious blood-contamination which alone determined the diagnosis : rallying was very slow, some bronchitis followed, general pains and gradual loss of strength ; bed-sores appeared,

and he sank exhausted 53 days after the cessation of specific fever. Through oversight an autopsy was not made.

CASE XCVII.—M., 60, weak and pallid, was admitted at close of invasion, and 10 days afterwards underwent a pronounced relapse (spirilla numerous), there were also indications of a second relapse 13 days later : nearly a month subsequently or two months after admission, he sank exhausted : previously there being no apparent complication of liver and spleen, but a jaundiced aspect, spongy gums, oedema of the feet, some swelling (temporary) of the r. elbow-joint, and finally slight pyrexia at night. At autopsy, there was seen fatty degeneration of heart, liver and kidneys, spleen large, dark and soft, petechial extravasations in the stomach and small intestines and old mucous erosions in the colon and rectum. The unfavourable influence of advanced years was here manifest.

CHAPTER VII.

CLINICAL MODIFICATIONS OF SPIRILLUM FEVER.

ESSENTIAL AND INCIDENTAL : MODIFICATIONS FROM CONJOINED ENTERIC, TYPHUS AND MALARIOUS FEVERS. BILIOUS TYPHUS.

So far as these are yet known, they may be arranged as follows :—

1. Essential modifications. 2. Those due to attendant conditions and complications. 3. Those attributable to supervening diseases. The form known as 'bilious typhus' is considered apart, at the end.

1. That under similar conditions, the fever varies greatly in intensity has been already shown : thus, illness may be mitigated so far as to consist of only a single and not severe febrile attack ; or if relapsing, the recurrence is represented by a mere febricula. In healthy monkeys, I found the sole result of infection to be often such febricula ; and, possibly, no more may occur in man himself. On the other hand, illness may induce death at invasion or relapse.

As regards modifications of character, it appears that under similar conditions, illnesses may differ otherwise than in their intensity : also a particular group of symptoms, indicating a particular modification of contagion, was sometimes seen in small collections of persons infected from a common source, as hospital employés, members of a family, husband and wife. No doubt in some of these instances a common personal predisposition was present, but this is hardly likely in others where race, age and sex were diverse : for details see Chapter on Etiology. In an experiment, I generally found the same material produce similar effects on two or more animals inoculated with it ; and in different experiments, somewhat different effects were noted. Such natural tendency to variation has to be borne in mind, whilst estimating the modifying influence of incidental conditions.

Previous infection.—So far as appears from four or five cases known of relapsing fever being repeated in the same subject, at different dates ; and from similar instances of repeated inoculations of the monkey, a previous illness does not necessarily modify, to a considerable extent, a later attack from another infection. Such later attack may, or may not, resemble the earlier one.

2. Modifications due to race, caste and physique, or to sanitation and season.—Hindoo immigrant agriculturists from the Deccan, seen chiefly in 1877, displayed the fever in pronounced and probably typical form. During 1878 were seen more Mussulman weavers from Hindustan, of feebler physique and oftener with malarious cachexia, who exhibited fever of less sustained form, more depression, more frequently the eruption and head symptoms, with other marks of typhus-like type.

Throughout the epidemic, low-caste classes from various districts, chiefly serving as town-scavengers and dustmen, and of inferior physique, suffered severely, yet not in uniform fashion. The few Eurasians seen at various dates, showed fever of continuous type or complicated with subsidiary pyrexia. All these classes live mostly on, though not exclusively, on vegetable food: there was no reason to suppose that diet operated as a considerable modifying influence, nor could such be attributed directly to mal-hygienic influences, or even to personal destitution and want. Doubtless paupers suffered most severely, yet not from different type of disease.

A particular modifying effect of season upon the character of the fever was not apparent, nor was there noticed any real change from year to year amongst similar classes of men; the group of patients treated in 1879, showing equal diversity of form and degree to those seen in 1877.

The sphere of observation at hospital was too narrow to permit of wide generalisation, but so far as appeared, the spirillum fever preserved all its main characteristics throughout, only in the worst class of subjects tending to assume a low and less regular form, as other acute pyrexias would; yet, I could not help remarking, to less extent than occurs with them. When the number of such subjects is considerable, the character of the 'epidemic' may be said to change accordingly; and when they become fewer, the commoner or average features of the disease again predominate. The modifying influence exerted by pre-existing disease has been alluded to in Chapter VI.; it was not invariably manifest, and greater modification could hardly be anticipated from general influences like those above-named. Instances were noted in which the fever maintained its essential characters amongst subjects just recovering from cholera, or affected with malarious and scorbutic cachexia, or with leprosy, cancer and elephantiasis: and, at most, these attendant complications might lead to depressed or irregular development of specific symptoms, the death-rate being higher without unusual frequency of spirillar accidents.

Modifications due to local disease attended with pyrexia, are described in the Chapter on Complications.

3. Modifications from co-existing or supervening febrile diseases.—There is no valid reason for doubting that more than one specific infection may be present in man, either at the same time or in close succession. First, as to co-existence; supposing that a 'continued' or a sustained 'malarious' fever concur with the spirillar, its presence would become apparent during the intervals of spirillum fever; the combined result being a quasi-continuous pyrexia displaying at certain periods the blood-signs and other symptoms of this fever, and at other times the appropriate indications of the intercurrent disease, as of typhus, enteric or remittent fever; and, *a priori*, there may seem no great difficulty in the diagnosis of such composite cases. Yet apart from the fact that the fevers last named, possess no pathognomonic mark whereby their presence can be invariably established, practical difficulties do arise from certain variable features of the spirillum fever itself; such as its occasional prolonged defervescence by lysis, its liability to local complications like those of other fevers, and especially its not rare adjunct of secondary fever. In more detail, during lysis the pyrexia and general symptoms

may simulate typhus, amongst complications are bowel complaints suggesting enteric, and remittents as well as typhus may be imitated by the consecutive fever. The skin eruption would not have afforded any aid at Bombay.¹

Concurrence of tropical Enteric fever.—Typhoid is known to prevail in Bombay, and several cases passed under my notice in 1877-78 (*vide* Appendix B); hence it was possible that occasionally mixed enteric and relapsing fever might occur. Only a single doubtful instance was, however, seen.²

CASE XCVIII.—M., 18, famine-immigrant, admitted in June 1877, four days in the town and twelve days ill with fever: emaciation and the typhoid state imminent, conjunctivæ much discoloured, no eruption visible, no diarrhoea, gurgling in the r. iliac region. Mental oppression, low delirium, sudamina and continued decline of strength during the four days he remained alive. The blood was examined twice by myself, with negative results; but on the case is an entry that the spirillum was seen at estimated 13th day of disease, m. t. 105°, p. 130; the authority for this entry was unknown to me. At the autopsy, besides pulmonary apoplexy, there was sloughing and ulceration of Peyer's glands at the lower end of the ileum, and also of the solitary glands of the large intestine; kidneys normal, liver unchanged, and spleen unaffected (weight 5½ ozs.) I was not present at the autopsy, but this record is made by a competent hand.

The chart is too brief for analysis; the aspect of the spleen is not that noted in spirillum fever.

¹ Whilst Griesinger on the ground of an abundant eruption of roseola seen in some severe epidemics, would recognise a mixed form of *T. recurrens* and *T. exanthematicus* ('*Infectionskrankheiten*,' 2nd ed. 1864), Wyss and Bock from experience at Breslau, were not disposed to do so ('*Studien ueber Febris recurrens*,' 1869); still later experience at St. Petersburg has led to a fuller development of views; thus, it is stated that clinical observation has shown that 'in the course of enteric and typhus certain peculiar symptoms appear, which indicate the presence of relapsing fever; unusual temperature-changes, sweats, only slightly marked head symptoms, clinically pronounced changes of liver and spleen, the outbreak of primary vermilion-tinted petechiæ quite at the beginning of the attack; these considerations supplemented by the pathological alterations undergone by the spleen, have convinced us of the possibility of concurrence in time of enteric, typhus and relapsing fever' ('*Zur Frage über die Mischformen des Typhus*,' aus der Clinic von Prof. Botkin, mitgetheilt von W. M. Borodulin. *St. Petersburger Medicinische Wochenschrift*, 15 Julie, 1878). Five selected cases are narrated, with charts, and inference is based upon the presence of blood-spirillum and certain symptoms occurring at intervals in the course of illness. In the same journal for 16 May, 1881, is a review of Dr. S. Lebedew's analysis of 216 cases of mixed typhus seen in a temporary hospital at St. Petersburg during the first half of 1880; here the concurrent form was conjoined with typhus 123 times, with enteric 73 times, and in 20 cases it was supposed all three fevers coexisted together: perhaps this last idea (based upon charts) would be regarded sceptically, without necessarily discarding the whole. I am fully of opinion that the ordinary temperature-charts are often an insufficient, and sometimes an unsafe guide in determining the nature of febrile complications.

² Three cases at St. Petersburg (Borodulin, *loc. cit.*) showed a continuous and gradually declining pyrexia of 20-25 days in two recoveries, and a more prolonged and sustained pyrexia of 36 days in a fatal case, where was found ulceration of Peyer's patches in the ileum, double pneumonia and disseminated suppuration in the r. kidney, together with hæmorrhagic splenic infarcts, and seemingly fatty changes of liver and kidneys. Petechiæ and roseola were noted on the third day of disease, the typhoid state soon set in, diarrhoea was present: the blood spirillum was found on two separate occasions, which I take to represent invasion and first relapse; possibly a second relapse occurred. Only the intestinal change seems here remarkable, and in conjunction with the other complications, its significance may be variously estimated. Diagnosis in a fourth case I regard as more doubtful, the chart and symptoms resembling those of secondary fever.

A similar interpretation is applicable to a surviving instance recorded by Dr. Rabagliati at Bradford (*Edin. Med. Jo.*, vol. xix., Dec. 1873). No eruption; no blood examinations at this date: the chart shows recurrence of fever shortly after end of first relapse, being sustained (with a mid-intermission) and prolonging the illness to 36 days.

Concurrence of Typhus.—The identical relations of this disease as regards not only locality and date of prevalence, but also the class of subjects affected, and originating and propagating conditions, would of themselves suggest the probability of typhus being associated with relapsing fever more intimately than enteric; and especially during severe epidemics, is it likely such association would be marked.

At Bombay, it was evident on first detection of relapsing fever that a 'low type' prevailed more frequently than not, and time deepened this impression, for as compared with most European records, there was noted here early depression, cardiac debility, increased prevalence of head-symptoms and an augmented mortality; an ineffaceable eruption was sometimes seen, and whilst the specific pyrexia, seldom high, might not vary much in its course, yet the febrile attacks (especially the first) were often prolonged, or ended by modified crisis, or were followed by secondary fever of equally 'low type,' which proved fatal.

Upon these grounds it was surmised that the local epidemic possessed a distinct 'typhus taint,' which was displayed in serious modification of the ordinary symptoms of relapsing fever; but whether or not veritable typhus was actually co-present in hospital cases, could not be proved from symptoms alone (for these might be changed during combination), and a pathognomonic sign of typhus is yet wanting.

Very early in my enquiries the following instances occurred, which were then regarded as showing the conjunction of idiopathic fever (probably typhus) with the spirillar: the fact of their being both youths is not in favour of that inference, which was based on general symptoms and aspect.

CASE XCIX.—M., 14, resident, works in a spinning-mill, admitted with a history of previous illness and for the last 5 days persistent fever, emaciated and weak, tongue dry and glazed, lips dry, hepatic tenderness, cough with bronchial congestion, e. t. $103^{\circ}4$, p. 124, slight delirium, no stool; for the next five days the temperature remained high and continuous, the lad was drowsy and stupid-looking, delirious at night, skin always dry, cough increased, no eruption seen, urine free from albumen, face puffy and then the legs: probably slight pneumonia was developed. With less continued fever, the tongue became moist, delirium less marked, cough looser, prostration more apparent: for 8 days longer the fever remitted only, not every day, and max. temp. not below 102° ; it then declined more decidedly and gradual improvement began, splenic enlargement and tenderness, temporary diarrhoea, about this time, with pain in r. shoulder (no jaundice): a brief relapse occurred on 31st day of disease. The blood was examined daily at first, and on one occasion showed many spirilla: the medical officer in charge noticed the case as one of 'typhus,' and judging from appearances correctly, although the chart (No. 14, Plate V.) suggests rather a concurring remittent.

CASE C.—M., 12, immigrant, admitted on estimated 8th day of illness with high fever, hepatic complication and cough, some night delirium and drowsiness at day; jaundice appeared and the typhoid state began, some pneumonia being probably developed; with decline of pyrexia improvement ensued, though slowly: spleen enlarged, but no other abdominal implication; febrile paroxysms occurred with exacerbation of these symptoms: the subsequent anæmia was great: no pains: recovery good. Eruption not noted. The blood was many times examined and the spirillum seen on 12th and 13th days, and again on 20th day, when rises of temperature also took place: in the final paroxysm, I failed to perceive blood-change. Chart like that of the last case.

Under ordinary circumstances typhus fever has not been known at Bombay, nor were distinct examples seen during the late public sickness; therefore, if this species of continued fever were ever present, it

must have been almost solely in connection with the new disease ; and of such conjunction the evidence seems defective. The common conditions under which nearest approaches were made to typhus occurred, first, when the spirillum fever was prolonged at the last and slowly subsided by lytic defervescence ; and, secondly, during certain kinds of reactive or consecutive fever. The symptoms supervening at 'lysis' have been already described ; they may usually be termed typhus-like, and since becoming acquainted with the late literature of this subject, I am somewhat surprised to find so little special notice taken in Europe of a modification very manifest at Bombay : perhaps non-use of the microscope in all severe illnesses, may partly account for this deficiency.

Secondary fever simulating typhus has also been alluded to, the interpretation here placed being one forced upon me in the course of enquiry, especially by the fact that infected quadrumana sometimes displayed such fever, and there could be no suspicion of typhus admixture there. See Appendix B, and Charts in Plate VIII.

In brief, it is my opinion that the phenomena sometimes regarded as evidence of the co-existence of typhus with relapsing fever may be explained in a less recondite manner : they were witnessed at Bombay, where typhus is not supposed to exist, and the chief of them also in animals literally wild.¹

Concurrence of Malarious Fevers.—In general, all the occupants of the native town of Bombay are exposed to malaria, the influence of which

¹ Prof. Wunderlich quotes a few references in allusion to the occurrence of lysis in the so-called bilious typhus, and afterwards Wyss and Bock noted that in 'bilious typhoid' defervescence, often without sweats, 'tritt häufiger lytisch ein.' Others go further, e.g. Dr. Rabagliati's account of the epidemic at Bradford is illustrated by charts, of which Nos. 2, 4 and 6 display lytic decline of fever after invasion, and these alone were selected by the author as illustrations of 'typhus' after relapsing fever : in No. 2 the pyrexia was prolonged 21 days and in the absence of details I should infer the concurrence of secondary fever or local complication ; in No. 4 it was prolonged to 13 days ; and in No. 6 to 19 days, doubtless from similar reasons : the author truly remarks 'it is striking how much the course of relapsing fever varies.' Previously, Dr. Claud Muirhead at Edinburgh, had noted the conjunction of 'typhus' with relapsing fever, furnishing a chart which shows secondary fever coming on three days after the second relapse, in a youth of 17 (patient No. 17) : *Edin. Med. Journ.*, vol. XVI., July 1870. Dr. Aufrecht, of Magdeburg, in a later account, describes a case of 'typhus' coming on after *febris recurrens* : it began three days after crisis, lasted thirteen days and ended with cystitis and pyelonephritis ; the whole resembling secondary fever with eventual complication : 'Pathologische Mittheilungen,' 1 Heft. 1881. These examples will suffice for ordinary illustration of views yet requiring definition ; and it is significant of the difficulties to be encountered in establishing these views and furnishing the means of precise diagnosis, that from St. Petersburg, where all typhus-like fevers have long been predominant and whence the recognition of their possible admixture has chiefly come, only scanty evidence has yet been made available. Thus, of the five cases described by M. Borodulin, but one is supposed to represent mixed *typhus recurrens* and *exanthematicus* : patient, a medical student, 24, admitted on 6th day of illness, with symptoms of invasion-attack, spirillum not seen ; sudden fall on 9th day, with an immediate rebound and six days of continuous pyrexia, high yet soon declining ; then a true relapse (spirilla seen), and most probably a second relapse ; eventual recovery. Here the main point concerns the first part of the illness, which is said to have come on gradually : the typhoid state speedily supervened and delirium lasting till 15th day or end of rebound ; there was a 'roseola exanthem.' and no evident blood-poisoning ; these conditions are regarded as adverse to the view of the fever being spirillar ; whilst the crisis and sweats on the 9th day, and subsequent course of sickness are considered as adverse to the view of simple typhus. My studies at Bombay enable me to suggest with confidence that the instance is simply one of secondary fever after invasion of spirillar pyrexia, the absence of visible blood-contamination at first being doubtless accidental, for I have myself in a first examination sometimes overlooked the parasite : there is nothing recorded to contradict this interpretation, which serves to explain the whole case.

is manifested by proneness to intermittent and remittent fever by the paroxysmal character of other fevers (*e.g.* the symptomatic), and by increased liability to visceral congestions and inflammations. It becomes, therefore, of interest to enquire if the late 'new' fever seemed any way modified by malarious influence, or became associated with it.

Careful perusal of the original descriptions of recent European observers, has convinced me that Relapsing Fever has shown precisely the same features in Bombay as in non-malarious countries of the West, where its practically unchangeable character has long been admitted; hence the inference that malaria had not considerably modified the disease, as seen in Western India.

As to association of their specific causes, the similarity of the spirillar pyrexia to that of 'remittents,' the like tendency to periodical recurrence and to splenic implication as in 'agues,' and the preference for malarious districts said to be shown in some European countries by relapsing fever, are the chief points noted in favour of such association: they have little validity in the present connection.

At Bombay the late epidemic fever was not coequal with the malarious, as regards either local distribution or local intensity, being far more restricted in its area, and not severest where common fever worst prevailed: it was frequent where malaria was not abundant, and also in non-malarious seasons of the year.

I do not know that spirillum fever especially predisposed to, or re-excited, the malarious influence.

Conjunction with Remittents.—These even less than typhus possess any pathognomonic characters, and during the pyrexial state it would not be possible to determine whether or not the two infections were concurrent. During the usual apyretic periods, secondary fever arising might be equally undistinguishable in form: nor would the test of anti-periodic treatment suffice here, for the remittents of Bombay are highly stubborn. And, in brief, clear evidence of the simultaneous prevalence of the two fevers never presented itself; but when it becomes possible to test malarious fever as infallibly as the spirillar, such evidence may be found. What is known, is that relapsing fever will pursue a regular course in a thoroughly malarious subject; and also when succeeding closely to a typical malarious attack.

Intermittents: Agues.—I have previously stated that single and repeated paroxysms of fever were not uncommon as third, or second, and occasionally as first relapses; they were also seen as sequelæ of spirillum fever, and in rare instances as rebounds of temperature after invasion and first relapse, or even preceding these attacks. As there are no means of distinguishing such last-named transient events, except by their association with the better known, discussion of their true character would be futile: the onus of proof that they are really aguish, would be on those who make this affirmation; at present, I regard the phenomena as incidental attendants on the spirillar infection.

It is right to add that a difference of opinion has obtained concerning the relationship of relapsing fever and intermittents, even amongst physicians residing in the same locality; and until the means of more precise diagnosis become available, such difference seems likely to last.

The conditions under which ague-like paroxysms occur in the body are very numerous, and include many non-malarious influences.¹

Icteric Fever, commonly Spirillar. Synonyms.—Yellow, icteric or bilious Relapsing fever; Yellow fever (*typhus icterodes*); Bilious typhoid (*biliöses typhoid*); *Typhus biliosus*; possibly some bilious Remittents of the East.

An unusually fatal form of fever distinguished by deep jaundice, irregular pyrexia, early prostration and a tendency to localised inflammations, was noted early in the Bombay epidemic and continued to its close. So far as I know, it had not been seen of late years previously to 1877, and hence bore a distinct relation to conditions then arising. Most of the cases I saw were referable to the spirillar disease, constituting a variety which merged by many gradations into the average forms of relapsing fever: there remained, however, a residuum of examples not displaying specific blood-contamination, and yet not to be separated, in a clinical sense, from the majority; and these cases gradually passed towards the class of remittents, forming a kind of intermediate group of themselves.

It is, I presume, because experience elsewhere (in Eastern or tropical areas, at least) was of similar character, that doubts have been expressed respecting the nature of 'biliöses typhoid' (Greisinger); nor can I, also relying upon observation in a malajia-district, free myself wholly from such doubt. Taking the Bombay data as they stand, I have decided to include under the present heading some of the non-spirillar cases seen; they are commented on at the end, and it is to be remembered that all selection of cases for analysis has been made at personal discretion.

The occurrence in Western India of genuine icteric relapsing fever forms a notable link in the chain of continuous data, establishing the identity of the late epidemic there with European epidemics.

Though not absolutely the severest kind of fever seen, this icteric form was in aspect the most striking, and as such was certain to attract particular attention. Characteristic instances occurred in about 5 per cent. of all admissions; amongst casualties the proportion would be 15–20 p. c., according to individual judgment.

Examples were noted at commencement of the epidemic, before microscopical aid to diagnosis was employed; of 20 selected later ones, 13 dated in 1877 (oftenest when hospital and city mortality were highest), 5 in 1878 (also concurrent with most casualties), and 2 so late as 1880.

Intense jaundice being invariable, it was generally accompanied by an eruption of red spots or petechiæ; appearing early at invasion-attack, beyond which most cases did not pass. At the relapse, however, jaundice might be most marked amongst casualties, or amongst survivors no longer present: it had no special relation of its own, nor were the severe symptoms conjoined with it peculiar to such combination. There was nothing to distinguish the form or duration of pyrexia during invasion; only, as in bad cases generally, the history often pointed to persistence of fever longer than 7–8 days, and the mode of defervescence tended to

¹ The somewhat discordant views of Niemeyer, Lebert and Litten on the connection of Ague (chiefly sequellar) with relapsing fever, need not be detailed here; since for its adjustment the whole subject has yet to be scrutinised by exact methods. Bombay was not a suitable locality for such investigation.

the lytic, a marked crisis not being seen in the worst examples : secondary fever and local complications were as frequent as in other severe cases, tending, as usual, to efface the average features of their temperature-charts. Rarely were the higher thermometric readings seen, viz. thrice only ; the max. t. being 106° F. at end of invasion ; commonly the pyrexia very moderately pronounced, and readings somewhat below the mean of all cases at same stage.

The 20 patients were males, at early adult age : I do not recollect seeing any infant or very young, and rarely an aged person or female, deeply jaundiced. Most subjects were in an emaciated or cachectic condition ; yet not all, amongst recoveries especially. The typhoid state was present or imminent 14 times, and in nearly all the casualties: great depression of the system was invariable.

Localised attendants were an eruption of vermilion-tinted spots, or of petechiæ, common yet not in proportion to severity when other complications existed ; parotitis, hiccup, hepatitis, enteritis (2), dysentery ; pneumonia (4) pericarditis, epistaxis, cerebral hæmorrhages (2) ; collapse. Minuter tissue-changes were also found, and in general death seemed referable to some manifest lesion, yet not invariably so.

Diagnosis was made at a glance ; respecting assumed resemblance to yellow fever, I may remark that in the only two fatal cases known of copious gastric hæmorrhage, jaundice was absent. Prognosis—the recoveries were 6 or possibly 7, and convalescence promised to be tardy : it is, however, certain that intense jaundice attended with eruption was consistent with comparatively mild specific pyrexia ; such favourable result being due to the absence of severe complication or accident. That 13 or 14 deaths were known in 20 cases is significant, especially in connection with ascertained lesions. 14 of the 20 displayed the blood-spirillum, and of these 5 survived. The following notes pertain to CASE XXXII., summarily described in Chapter IV. as an instance of intercalated relapse.

M., 35, admitted with his young son from a weaving factory, on estimated 8th day e. t. $102^{\circ}\cdot 2$, of invasion, p. 128 ; the usual symptoms of spirillum fever with much debility, haggard look, deep jaundice, great hepatic tenderness and bruising pains in the r. shoulder, tongue dry and shrunk, numerous purplish spots in the left infra-clavicular region, large, level, ineffaceable, of irregular form and clustered, others elsewhere and red patches on the back : the blood was full of spirilla. Next afternoon the *perturbatio critica*, t. 104° , p. 120, crisis with sweats, and on 10th day m. t. $98^{\circ}\cdot 2$, p. 96, much depression, jaundice more marked, has sore throat (much vascularity visible), and pain on deglutition, the eruption more abundant and manifest in the now pallid skin, and it merges into true petechiæ : a rebound shortly followed, e. t. 101° , p. 110, much depression, fresh spots now partly papular, hiccup (? similar spots in the stomach). 11th day—has rallied a little, more skin spots and apparently others in the fauces and on tonsils, e. t. 100° , p. 112 : next day, still rallying, no new eruption and the former spots are fading, e. t. 98° , p. 86. 13th day—improves, yet still pain on deglutition and aching of joints, the eruption fades quickly, some older spots having disappeared and the later become papular as if from œdema ; axillary glands not affected, a slight e. rise of temperature without chills and increased aching of the loins. 14th day—most of the spots have subsided, a few smaller ones are new, jaundice nearly gone ; aching pains severe and a smart febrile paroxysm (e. t. 104° , p. 130, and spirilla in the blood), slight chills preceded and copious sweats ensued : this was an intercalated paroxysm, *vide* Chart No. 10, Plate V. Next m. t. 98° , p. 88. 16th day—still some soreness of throat and a slight tenderness of r. parotid region, no redness of fauces ; he is pale and giddy, but eats well ; some doubtful fresh spots. Next day, improves, but sleep disturbed by his having to wait on his sick son. 18th day—m. t. $98^{\circ}\cdot 8$, p. 88, slightly feverish in the night and feels weak ;

there are spirilla in the blood (specific incubation-period), and next day, the relapse suddenly came on, e. t. 104°·6, p. 120; renewed debility, no fresh eruption or jaundice, but severe pains and a haggard look; smart fever lasted 3 days and ended by crisis, some minute hæmorrhagic spots then appearing on each side of the sternum: gastric irritability and splenic tenderness, weakness, yet soon appetite and as quick rallying as in his son (who was not jaundiced and showed only slight traces of an eruption). Previously I had not known such favourable ending of so unpromising a case, but here no complication had persisted and the constitution seemed fair. In another instance—M., 30, the first aspect was also unfavourable, jaundice and pallor being marked, petechiæ present and the debility great, there was epistaxis, too, at the fall; yet in the absence of local lesion, rallying was prompt after the brief relapse which ensued.

The 9 fatal cases formed a striking series: chart of 1 is copied in Pl. VI., No. 25, being that of a man, 30, dying of pneumonia three days after apparent first crisis ('bilious pneumonia'), fever of remittent type, pulse moderately quick, jaundice deep, two petechial spots, the typhoid state and delirium, great enlargement of the liver with thin black stools, *prolapsus ani*; even here slight rallying after cessation of specific fever was indicated, but the exhaustion proved insuperable, pneumonic symptoms being latent. After death the l-ft lung was found inflamed, the spleen and liver very large, gall-bladder empty, kidneys (like liver) pale, ? fatty: the brain firm. A second case is No. LXXVIII., the man dying of pericarditis and hæmorrhages at close of first relapse; and another instance of decease at this date, was seen in a man, 30, admitted in a state of great debility at close of invasion, with rebound; yet rallying and promising convalescence when the relapse set in, this proved to be prolonged and pronounced, and death occurred without fall of temperature; both spleen and liver were found greatly enlarged. In a later case the patient showed but slight fever on admission (t. 99°·2), and overlooking the rapid pulse (140) and quick breathing (48), together with the peculiar dyspnoea and abdominal distension, I deferred close scrutiny till next day, when death ensued: the blood was full of spirilla and proved to be so infective that two young men engaged at the autopsy caught spirillum fever, one dying.¹ In this example with other characteristic changes, there was found enteritis (diphtheritic): *vide* CASE LXXXIV. for details: jaundice was also present in another instance of this kind, though not so deep as here and hence I have not included the example in the present list. The clinical charts belonging to this series, offer no peculiarity for notice.

Six of the cases did not show the spirillum, and as a sample of local experience I subjoin a brief analysis of them.

1. One not admitted till the 12th day, or after probable subsidence of specific fever; the typhoid state present, and speedy death from pneumonia (max. t. 102°): the brother, ill for 6 days only, admitted at same time from same house (others were ill there), showed the spirillum and recovered; he, too, was deeply jaundiced and had abscesses after crisis (1877).

2. One admitted on 15th day, in typhoid condition, also had pneumonia and was removed by friends in a moribund state: max. t. 103°: judging from the whole circumstances of the case, this, too, was an instance originally of specific infection.

3. Admitted on reputed 5th day of illness, with all the aspect of a patient at the critical fall of spirillum fever: depression great, rallying slow, and a kind of long, low relapse followed; then fever of more intermittent type, increasing cachexia and fatal exhaustion; no autopsy. As the fresh blood upon several occasions failed to show visible contamination (improved method of scrutiny then unknown), the true nature of the case remains unproven; but a neighbour was admitted with spirillum fever about the same time, and there was no moral doubt as to the specificity of illness.

4. Admitted on reputed 5th day, t. 103°, subsequent pyrexia resembling that of remittent fever, subsidence gradual to normal after 10 days; during this time parotitis and epistaxis, which at this epoch were practically unknown in Bombay, except

¹ These accidents at Bombay afford proof of the specific character of 'bilious typhoid,' parallel to the deliberate inoculation successfully practised in Russia, which is usually quoted by continental authors as evidence of the 'recurrent' nature of this form of spirillum fever. In neither of my hospital assistants was jaundice a marked symptom: for details see Chapter on Contagion.

in connection with the new fever: the man was unconscious on admission, abdomen distended and tender in both hypochondria, liver and spleen found to be enlarged: patient an immigrant from the famine-districts; the fresh blood was repeatedly examined with negative results, this happening in all forms of secondary fever.

5. Admitted without a history, in state of collapse, was said to have come from the Deccan a few days previously: the symptoms included intense debility, restlessness, not real stupor, eyes bright though injected; an eruption of ineffaceable pink spots; pupils contracted; the typhoid state imminent: rallying slight, the liver was implicated, soreness of the skin, much irritability of the muscular tissue, no stool for three days. In a few days fatal exhaustion, and at the autopsy double pneumonia; the liver contained a small abscess, seemingly old; spleen reported shrunken and dry, its weight 3 ozs., kidneys probably not diseased, sub-mucous hæmorrhagic spots in stomach and at end of ileum, with congestive patches elsewhere. Blood-poisoning was indicated, its source untraced. I have known similar results attend spirillar infection.

6. Admission on reputed 6th day of illness, t. $102^{\circ}4$, p. 96, fever continued for seven days, being moderately sustained; blood examined twice with negative results: patient low and delirious, urine retained, increase of jaundice, liver-dullness lessened (?), the splenic increased, abdomen tympanitic, respirations slow, pupils contracted; the breath ammoniacal; coma supervened. At autopsy, the liver weighed 2 lbs. only, yet seemed healthy, the hepatic cells being reported unchanged: spleen enlarged, kidneys congested, cerebral congestion with effusion of serum: patient a police constable exposed to malaria, and other infection.

Regarding the above, such cases had not been seen of late in Bombay, and their symptoms and history, when not suggesting a prior relationship to spirillar infection, were seldom inconsistent with such relation: instances like the two last of pyæmia (?) and cholæmia (?) may have had a different connection, but too little of the illness was seen to warrant a decided opinion as to their original character. I have termed this group of cases 'Icteric Fever,' because not in all was the blood-spirillum seen, and hence proof positive of their specific nature is wanting; but my opinion is that 4 of the 6 last non-spirillar cases really belonged to the same specific disease as the earlier ones, and that the remaining 2 of the 6 are examples of lesion consecutive to fever. There is nothing here to gainsay the extreme probability that *typhus biliosus* was the same disease in Bombay, as it is in Europe.

CHAPTER VIII.

MORTALITY OF SPIRILLUM FEVER.

GENERAL FEATURES—1. DEATH-RATE. 2. DATE OF DEATH. 3. APPARENT CAUSE. 4. MODE. DETAILED FEATURES—5. INFLUENCE OF SEX. 6. AGE. 7. SEASON. 8. PERIOD OF EPIDEMIC. 9. SOCIAL STATION. 10. BIRTHPLACE. 11. RACE. 12. HABITS. 13. PREVIOUS DISEASE. 14. MENTAL DEPRESSION. 15. BODILY EXHAUSTION.

1. *Death-rate*.—Of 616 demonstrated cases 111 died, the rate of decease amounting to 18·02 per cent. Amongst ordinary hospital patients the greatest mortality was noted at the J. J. Hospital, where there were 82 deaths in 453 admissions (equal to 18·1 p. c.) ; whilst at the G. T. Hospital, the deaths were 11 in 94 admissions (or 11·7 p. c.). Amongst the known instances of disease acquired by contagion at both hospitals, the deaths were 18 in 69 cases, or at the rate of 26·1 p. c. The proportion of deaths from famine-fever occurring at the Camp of Refuge, 1877, was not ascertained.

When compared with the statistics of most hospitals in European countries (*e.g.* 4·03 per cent. in British hospitals, 4·3 to 7·2 at Breslau, though as high as 14·97 p. c. at St. Petersburg in 1865), the above data will serve to show how severe was the late epidemic at Bombay ; nor is this feature annulled by the circumstance that an unusually large proportion of ordinary admissions happened at advanced stages of illness, or by the fact of acquired disease amongst those already sick being uncommonly severe : for the first is a common condition, and the second may not be peculiar to Bombay : see the Memorandum below. Details are the following.

TABLE XII.—DEATH-RATES AT BOMBAY.

Hospital	Subjects	Survived	Died	Total	Death-rate per cent.
J. J. H.	Admissions . . .	371	82	453	18·1
	Contagions all :—	39	17	56	30·3
	{ Patients . . .	20	10	30	33·3
	{ Servants . . .	9	6	15	40·
G. T. H.	{ Pupils . . .	10	1	11	9·
	Admissions . . .	83	11	94	11·7
	Contagions all :—	12	1	13	7·6
	{ Patients . . .	1	—	1	0·
	{ Servants . . .	6	—	6	0·
	{ Pupils . . .	5	1	6	16·6

2. *Date of Death*.—Analysis of the 99 deaths occurring at the larger hospital, after excluding 7 examples of uncertain date (though most probably near termination of first attack), furnished the following results.

a. *Invasion-attack*.—48 deaths (or rather more than one-half) took place at this stage; viz., on estimated fourth day 3, fifth day 4, sixth day 3, at or about apparent acme of fever 27, and at stage of defervescence 11. Respecting deaths at the earlier of these periods, it is possible some belonged rather to the acme of short attacks, for events so much abbreviated were distinctly noted; and hence it might be assumed that $\frac{2}{3}$ of all casualties at this stage took place at or near the culminating point of illness, when the patient's strength is often severely tested, and when always the blood seems to undergo a rapid change of visible condition. Deaths at the decline of fever chiefly belonged to the rarer form of gradual, or lysis-like defervescence.

b. *First Interval, or Period after specific pyrexia*.—24 deaths, or about $\frac{1}{4}$ of the whole series: thus, there were three casualties on the first day after crisis, 1 on the second, 4 on the third, and 2 on each of the succeeding three days; the numbers subsequently being scattered over dates ranging 1–2 months, and pertaining rather to sequelar phenomena. It is, therefore, either immediately after the first crisis, or within about a week later, that one-half the deaths at this epoch may be expected to occur,

c. *First Relapse*.—6 deaths, or about $\frac{1}{15}$ of the whole: possibly this diminished proportion of casualties at first relapse is excessively small, from some patients overlooking the earlier attack; yet there is other valid evidence of a common mitigation of symptoms, upon fresh or auto-inoculation of the system. Deaths occurred after the third day, and as then the acme of attack is always contingent, it may be said that they tend to happen (as during the Invasion) in connection with this culminating epoch.

d. *Second Interval*.—11 deaths, which occurred chiefly from 2 to 8 days after crisis; the remainder being distributed over periods reaching to the 18th day.

e. *Second Relapse*.—There was only 1 instance referable to the third day of illness; and, as in most late casualties, local inflammation was present.

f. *Sequelar periods*.—2 patients died in hospital, at 30 and 42 days after cessation of specific pyrexia: whether or not others after leaving hospital, failed to recover, is unknown; but occasionally the state of the sick impatiently insisting upon their discharge, was unfavourable to life.

Analysis of the 12 casualties occurring at the G. T. Hospital, furnishes results which may be estimated as follows:—4 deaths during first attack (all with temperatures little raised); 5 during first interval, 2 happening immediately after first defervescence; 2 deaths at or near close of first relapse; 1 very shortly after the second crisis.

The hour of the day when death was recorded varied indefinitely, but most frequently it was from midnight to 6 A.M., and next oftenest from noon to 6 P.M., which are the usual periods of decline and rise of body-heat respectively: further inference was not practicable here.

3. *Apparent Cause of Death.*—In by far the majority of cases, life seemed to be destroyed by febrile distress or consequent exhaustion, such being the conclusion arrived at in 63 of 99 casualties: cerebral hæmorrhage was ascertained 7 times and may have been rather more frequent: 2 deaths arose from copious gastric hæmorrhage and 1 from femoral thrombus: 17 were attributable to pneumonitis, and milder degrees of this complication may have been overlooked: there was acute dysentery 8 times, and once hepatic abscess. The remarkable state here termed 'febrile stress,' consisted in exaggeration or acute modification of the severer symptoms elsewhere described as occurring at the close of attack during Acnie, at initiation of Crisis and in course of Lysis defervescence: the circulatory and respiratory organs being most obviously affected, as well as the viscera contained in the upper abdominal zone. In characteristic instances this distress came on more or less suddenly, and was of brief duration; when not proving fatal, it was followed by prompt and complete recovery, and from personal experience I know how great is the suffering at culmination of the febrile periods, before the advent of delirium and unconsciousness. Hence, upon wide grounds I gained the impression that were critical epochs prolonged, even slightly, the mortality from spirillum fever would be much greater than it actually is.

Under the head of exhaustion, should be included the state of collapse in which febrile crisis may terminate. Possibly my observations were defective, but it happens that death in the condition of extreme depression was most rarely indicated; and however pronounced, this state did not appear in itself to be one of peril, patients (contrary to first anticipations) rallying from it surprisingly well. There is also a fatal form of debility, sequelar in its advent.

The symptoms attendant upon ordinary local lesions, need no further allusion here. In general, inferences formed during the later hours of life were confirmed at the autopsies, which were practised in upwards of three-fourths of all casualties. Whether or not superadded blood-contamination (*e.g.* pyæmic or other auto-genetic kind) might be more frequent than appeared, I will afterwards enquire. Sect. III. Chap. III.

4. *Mode of Death.*—Whilst the blood-poisoning usually terminated in insensibility, often, prior to the end, there were signs of stupor or coma; and at close of the febrile attacks, especially, cerebral hæmorrhage in varying degree, might be regarded as a likely contingency. How far, in addition, the sympathetic ganglionic centres in the abdomen became implicated, and by their derangement contributed to the fatal suffering, can as yet be but indefinitely surmised.

It was sometimes apparent that death began at the lungs with active congestion or inflammation, as primary phenomena: at other times, derangement of the breathing was but part of more general disturbance.

Death of cardiac origin was not rarely indicated by the frequent and feeble action of the heart, without signs of other lesion than concurrent derangement of the breathing, and marked fulness and tenderness of the abdomen.

5. *Influence of Sex.*—Of the total deaths 71·77 per cent. were males, and 28·83 p. c. were females: as amongst total survivors the proportion of females was only 15·32 p. c., it would appear that this sex suffered

more severely than males from famine-fever, and I may observe that the only 2 ward nurses attacked in the J. J. Hospital died at invasion, and as well 3 out of 8 female sick seized in the same hospital, of all ages. The excess of female deaths was noted under the age of 15 (particularly from 6-10), and again after 36 years; whilst during the intermediate periods, and especially at the epoch of greatest total mortality—viz. 26-30, the proportion of females dying was less than that of males.

6. *Age*.—The influence of age was apparent, in the greater comparative mortality at both extremes of the scale of years: thus the general mean death-rate being about 18 p. c., the rate was 27 p. c. up to the age of 10 years, and then in the two succeeding decennia declining to 11 p. c. (11-20 years) and 16 p. c. (21-30 years), it rose with advancing age above the mean to 24.5 p. c. (31-40 years), 29.4 p. c. (41-50 years) and 37.5 p. c. (51-60 years). The material for these calculations is contained in the appended Table XIII.

TABLE XIII.—AGE AND SEX IN SPIRILLUM FEVER.

Age in years	Survivors			Dying			Grand Totals		
	M.	F.	Tot.	M.	F.	Tot.	M.	F.	Tot.
To 1	—	1	1	2	—	2	2	1	3
2-5	2	6	8	—	1	1	2	7	9
6-10	7	13	20	2	6	8	9	19	28
11-15	25	4	29	2	1	3	27	5	32
16-20	65	4	69	6	3	9	71	7	78
21-25	105	10	115	14	5	19	119	15	134
26-30	116	14	130	24	4	28	140	18	158
31-35	35	5	40	12	3	15	47	8	55
36-40	28	9	37	6	4	10	34	13	47
41-45	7	2	9	—	1	1	7	3	10
46-50	12	3	15	6	3	9	18	6	24
51-55	3	—	3	1	1	2	4	1	5
56-60	4	3	7	4	—	4	8	3	11
Totals . .	409	74	483	79	32	111	488	106	594

As the late epidemic at Bombay was mainly due to an influx of persons either sick or most readily falling ill, useful comparison of the above data with Age and Sex of normal population, or ordinary mortality in the town, cannot be made: nor could the total immigration data be accurately estimated. Respecting comparison with experience of famine-fever in Europe, I note that besides a high general death-rate, the considerable mortality in early life at Bombay seems peculiar; whilst afterwards there is an accordance in the progressive augmentation of mortality according to age.

7. *Season of the Year*.—There was no distinct evidence that during the heavy rains at Bombay, or in the succeeding more malarious months, *cæteris paribus*, the death-rate was considerably or uniformly altered; and from local knowledge I should infer that a seasonal influence, if present, must have been quite subordinate.

During the two chief years of sickness the larger hospital mortality was 20 per cent. of admissions in both hot and wet seasons, and about 14 p. c. in the cold season (data then fewer and imperfect) ; during the year 1879, the 8 deaths noted at my smaller hospital mostly occurred in the rains and cold season. Temperature ranges on Bombay island are probably too narrow alone to influence the course of a contagious malady, and owing (as would seem) to purely contingent circumstances of race and general condition, the death-rate of famine-fever during the rainy months varied from 18 p. c. in 1877 to 25 p. c. in 1878.

8. *Period of Epidemic.*—Here, too, the disturbing influences were too considerable to permit of adequate estimate of mortality at beginning, middle and end of the epidemic ; but so far as appears, the mere intensity of infection (epidemic influence) may not have greatly varied throughout. During 1877 the death-rate of the J. J. Hospital was 18 p. c., and in the following year it was 21·2 p. c. although the public sickness had become much less : in 1879 at the smaller hospital it was only 15 p. c. of admissions, yet I know that the infective virulence of the disease was still great, at this late date.

9. *Station in Life.*—With rare exceptions, the deaths occurred amongst the poorest classes of patients admitted ; town residents supplied a few petty traders to the list of casualties, but the well-to-do sections of the native community were conspicuous by their absence here. I heard, however, of isolated fatal cases amongst merchants outside hospital ; and the fact of several well-nourished hospital servants, and a few well-fed and well-lodged medical subordinates dying of the infection, sufficiently shows that, in detail, social status may have little essential influence on mortality.

10. *Birthplace and Residence.*—In the chapters on History of the Epidemic and on Contagion it is stated that the town-residents of Bombay, most of whom were born in the Mofussil, displayed nearly as high a mortality-rate as the immigrants themselves ; and this circumstance indicates the comparatively little effect on death-rate of slighter differences in personal states, when the conditions of infection and insantiation generally were favourable to flourishing of disease.

11. *Race.*—I have elsewhere pointed out some differences in the death-rate amongst the two principal races of Hindoos and Mussulmans, the indications being that the less robust weaver class from N. India suffered rather the most. The out-caste sections of the community, also, were excessively implicated. So far as race and caste represent differences of diet (and such differences are not considerable in Western India), virulence of the new fever did not, *per se*, appear to be essentially influenced thereby.

12. *Injurious Habits.*—The constant use of intoxicating drugs was too rare amongst the poorer classes to attract attention, and only in a few particular instances did it seem that prior intemperance led to an unfavourable issue of illness.

13. *Previous Disease.*—So far as appeared to me, the attacks of famine-fever were not necessarily rendered more fatal by the circumstance of pre-existing malarious cachexia ; such impairment of health, in its several degrees, was doubtless widespread throughout the community, and might be difficult of detection in fever patients seen for the first

time, yet I was not impressed with the evidence of its particularly hurtful influence, except in some extremest cases. The large mortality amongst patients in the J. J. Hospital who became infected by contagion, would at first sight indicate the baneful influence of previous disease, yet the death-rate amongst healthy hospital servants at the same institution, was nearly as large; and if (as is not improbable) the constant or frequent breathing of impure or tainted air had a bad effect, still I note that of 9 lads acting as clinical clerks in the medical wards, who became infected, not one died or came near to death during the worst year.

14. *Mental Apathy and Depression.*—This state was common amongst hospital patients, and may have aggravated the enfeebling effects of infection; in a few instances its effects were marked.

15. *Bodily Want and Exhaustion.*—Unquestionably the injurious consequences of fatigue and privation, were not less marked at Bombay than is recorded of European cities; yet whilst the whole history of the epidemic under notice, tends to show that spirillum fever prevailed most (if not first) and most severely, amongst the famine-immigrants flocking townwards, yet it was also apparent that not all such immigrants were either starving or exhausted (for many travelled by rail and had some means, or could work); and, as matter of fact, only a certain proportion of the dying in hospital, whether new-comers or residents, were in a state of actual emaciation upon admission. It is possible that debility of the frame was often concerned with the marked tendency seen to death from exhaustion, at acme of the febrile attacks; still exceptions to this view were not very rare, and at last I came to refer casualties to either unusual degrees or qualities of infection, or else varying personal predispositions: both of these influences, however, being presumptive only, and neither of them open to close investigation.

MEMORANDUM ON THE MORTALITY OF RELAPSING FEVER IN EUROPE OF LATE YEARS.—From a brief review of some recent writings, I find European experience to have differed somewhat from that at Bombay.

Death-rate.—In my estimates, no distinction was made between the ordinary form and the severer type of relapsing fever known as *bilious typhus*, because it did not seem to me to be required; but in the St. Petersburg epidemic of 1865, such distinction was marked by Dr. H. Zorn ('Petersb. Zeitschrift,' vol. IX. 1865), who states that the death-rate of the common form was 9.69 per cent., whilst that of the bilious form was 46.34 p.c. Admitting this discrimination (which may be serviceable in India), it becomes obvious that the death-rate of an epidemic will be high or low, according to the proportion met with of the severer type; and in the course of the same epidemic, it will also vary accordingly. The bilious type has not been common in Great Britain, nor in recent German epidemics; and casualties attended with deep jaundice were not numerous at Bombay, but it is quite possible in other parts of India experience has been different.

Date of Death.—In no European records met with is the proportion

of deaths at invasion and first interval, nearly so large as that above stated for Bombay. Thus, of 129 casualties enumerated by Drs. Murchison (England), Zorn (St. Petersburg), and Litten (Breslau), only 13 or about 10 per cent. occurred at the invasion-attack of fever; but about 30 p. c. at first relapse, and 17 p. c. at late or sequelar periods of illness. Deaths during the first and second intervals were 16·2 p. c. and 21·7 p. c. respectively. These ratios greatly differ from those I have named above, and it is not easy to account for this difference. It is, however, to be noted that Dr. Zorn distinguishing instances of bilious typhus, found that in this form the mortality was not only higher but more prompt, than in ordinary cases of relapsing fever; thus of 36 casualties 30·5 p. c. took place at invasion, nearly 20 p. c. at the first interval, and 30·5 p. c. at first relapse. All the figures are as follows:—

TABLE XIV.—DATES OF DEATH IN RELAPSING FEVER.

Dates	Inv.	1 Int.	1 Rel.	2 Int.	2 Rel.	Later	Total
Common form, Europe	13	21	37	28	8	22	129
Bilious typhus, Europe	11	7	11	2	—	5	36
Bombay experience	52	29	8	12	1	2	104

This datum is a very significant one for the sanitary history of India. I have not access to documents permitting of comparison of the late Bombay results, with those met with in casualties from the contagious fevers of N. Indian districts and large gaols during recent years; but such collation must prove instructive, and it may be Indian experience has its own special features.

Causes of Death.—So far as ascertained, there is here a close similarity of results between St. Petersburg and Bombay; for in both cities upwards of 60 per cent. of all casualties have been attributed, in general terms, to febrile distress or exhaustion, whilst at Breslau (1872–3) only about 15 p. c. On the other hand, deaths from pneumonia or other lung implication were only 9 p. c. at St. Petersburg, but about 18 p. c. at Bombay, and about 80 p. c. at Breslau. This datum shows that in severe epidemics like the first two named, death tends to occur from the fever itself; in the milder outbreaks from complications, chiefly of the lung. A preponderating cause at Bombay, was cerebral hæmorrhage since Dr. Zorn does not allude to it; but another contemporary observer (Dr. Kremiansky) expressly names hæmorrhagic meningitis as a frequent cause of death in St. Petersburg, and Dr. Lebert has adopted this opinion. The summary of these analyses is that in India relapsing fever was uncommonly severe, early causing death and that mainly from the fever itself; without there being seen, at all frequently, the deep jaundiced type known as ‘bilious typhoid.’ It is necessary to add that my examples were all of demonstrated spirillum fever, whilst those of Drs. Zorn and Kremiansky were not diagnosed by blood-scrutiny.

CHAPTER IX.

ANATOMICAL LESIONS.

INTRODUCTORY OBSERVATIONS. SUMMARY. DESCRIPTION OF LESIONS.
LESIONS ACCORDING TO STAGE OF FEVER: FINAL REMARKS.
MEMORANDUM ON EUROPEAN OBSERVATIONS.

MATERIALS were derived from the two Native General Hospitals during the years 1877-8-9. A few records have been mislaid, but I know they were not peculiar: no selection of cases was made. Here only instances are employed, which during life were demonstrated by examination of the blood, to belong to the spirillum-fever series. Several other data were available of cases more or less presumptively of the same nature, which may be said in great measure to correspond to the data quoted in works of authority published prior to 1873, but such are not included and seldom referred to below; no peculiar lesion was found amongst them. Of the J. J. Hospital records 64, of the G. T. Hospital 10, in all 74 examples are now analysed: they pertain to classes of patients representing in proportion of sexes, mean age, ratios of race, caste and occupation, residence and home, the bulk of famine-fever patients. Autopsies were most numerous at times when sickness was greatest: 48 of them were made at an interval of under 6 hours after death, and only 7 after a period of 12 hours: twice only was there any sign of incipient decomposition of the bodies.

Medical officers in charge of the sick entered the details, stating in customary terms the aspects, and commonly the weight, of each organ; all notes are preserved: at the great majority of autopsies I was present. Special scrutiny of the fresh tissues was made when practicable, and some parts are preserved.

I have added a memorandum on the post-mortem appearances of inoculated monkeys, dying or killed at various stages of specific fever.

Below, the morbid lesions are first summarised, then described in anatomical order, and finally considered with reference to the stage of fever at which they were found. Histological memoranda are appended; from my last specific illness ending with ophthalmitis, they are but few. Some repetition of the data was inevitable in the course of these analyses; to supplement deficiencies, some brief notes copied from published European sources are subjoined, and these will serve the additional purpose of aiding in the identification of spirillum fever as seen in India and Europe. Minute description of the Blood is relegated to the Section on Pathology, Chapter I.

I. SUMMARY.

Emaciation was rarely extreme. Vibices and spots on the skin were never prominent. The aspect of the Blood was in no respect peculiar, nor was it uniform. Unusual deliquescence, with staining of the endocardium and inner coat of blood-vessels, was very rarely noted.

Rigor mortis.—At invasion-stage rigidity had not set in so early as 1 hour after death, and seldom under 2 hours; it was most marked between 6 and 8 hours, and sometimes then commencing to disappear; at 10 hours, it had generally subsided, or much earlier in emaciated subjects, but exceptionally lasted till 12 or even 16 hours; it was present when the body still retained warmth and the blood was fluid; and had sometimes passed off while fluidity remained: it has been noted when a certain amount of decomposition had begun, 16 hours after death as the critical fall of fever. It began rather later (*i.e.* after 2 hours) and lasted longer (*e.g.* 13 hours), in subjects dying during the first apyretic interval. In death during first relapse, it had come on so soon as 1 hour after death (body well nourished). At the second apyretic interval its advent was somewhat longer delayed. There did not appear, however, any absolutely fixed relation between the onset of post-mortem rigidity, and the febrile or general state of the subject during life. The presence of even deep jaundice, seemed not to influence this phenomenon.

The aspect of the voluntary muscles, so far as exposed to view, rarely struck me as being peculiar; nor was their consistence markedly abnormal. Subcutaneous and deeper-seated extravasations of blood, or collections of serum, and changes in adipose or fibrous tissues were extremely rare. Some degree of bilious staining of the tissues was not unusual, and in the more jaundiced cases pronounced.

Main aspect of organs; one or more changes may co-exist.

Brain: 57 inspections. A quasi-normal aspect in 3, congestion 8, inflammation of meninges 3, hæmorrhage chiefly meningeal 10, subarachnoid effusion of serum 23, pallor 9, an old clot once.

Lungs: 74 inspections. Quasi-normal 10, pale and collapsed 21, congested 13, consolidation chiefly pneumonic 21, subserous petechiæ 4, bronchitis old and recent 2, pleurisy old and recent 3.

Heart: 74 inspections. No apparent change 7, clots in right cavities 47, substance decidedly pale 10, pericarditis 1, subserous petechiæ 5, valvular disease (old) 3, fluid blood 1.

Liver: 74 inspections. Quasi-normal 4, enlarged (congested or pale) 36, congestion 12, pallor 11, mottling 6, softening 2, abscess (old) 2, cirrhosis 1.

Spleen: 74 inspections. Quasi-normal 6, enlargement 46, infarcts 14, softening 8.

Kidnies: 74 inspections. Quasi-normal 10, enlarged 8, congested 24, pallid 27, subcapsular petechiæ 3, granular (old) 2.

Intestinal canal.—Stomach: 17 inspections; pallor of mucous membrane 5, congestion, hæmorrhagic spots or ulcer 12. Duodenum: 34 inspections; pallor or no change 21, congestion or petechiæ 13. Jejunum: 37 inspections; pallor or no change 21, congestion or spots 16. Ileum: 42 inspections; pallor or no change 21, congestion,

inflammations or petechiæ 21. Cæcum : 32 inspections ; no change 17, spots or ulcers 15. Colon : 36 inspections ; pallor or no change 21, congestion, spots or ulcers 15. Rectum : 31 inspections ; no change 23, congestion, spots or ulcers 8.

From this general view, it appears that there is no invariable lesion of the coarser kind to be found after death from spirillum fever : the term quasi-normal does not here exclude minuter change, and most probably the number of limited congestions or hæmorrhages is understated.

As contrasted with a nearly equal number of contemporary autopsies made after death from remittent fever (so called), the special features of relapsing fever may be said to be cerebral hæmorrhage, collapse of the lungs (pneumonia being about as frequent, viz. in near 33 per cent. of all necropsies), enlargement and pallor of the liver, enlargement or firmness and infarcts of the spleen, enlargement and pallor of the kidneys, congestion and extravasations in the walls of the intestinal canal.

Coarse changes noted in monkeys dying of specific fever after inoculation with spirillar blood, were the following—liver congested and once enlarged ; spleen large and congested ; kidneys healthy-looking : mucous membrane of stomach once unchanged, twice inflamed about the middle. In two animals dying on third day, the small intestines were inflamed throughout, beginning abruptly at the pylorus and ending at the ileo-cæcal valve ; there being hæmorrhagic spots also. Petechiæ were seen twice on the lungs and on the heart ; the brain pallid only. Splenic infarcts were never noted, or pneumonia ; and I acquired the impression that the many morbid lesions of spirillum fever in man, might be due partly to inherent or acquired weakness of the tissues.

2. DESCRIPTION OF THE LESIONS.

The Nervous System.—In the absence of symptoms referable to the spinal cord, this large centre was but rarely examined ; it then seemed to be healthy. The sympathetic system, too, was only casually inspected.

1. *The Brain.*—The normal mean weight of the brain in Natives of Western India, has not been ascertained ; probably it is similar to that of indigenous inhabitants elsewhere, viz. somewhat over 40 ozs. in males, and somewhat under in females. Pathological weights would, I think, fall within normal ranges.

External parts of the head.—The scalp was usually congested, especially at the occiput ; the cranial vault and dura-mater with its serous lining, were very rarely altered in aspect ; the venous sinuses were commonly filled and sometimes engorged. The outer membranes and adjoining surface of the cerebrum, were almost exclusively the seat of the morbid changes ; thus, in the arachnoid cavity and subarachnoid space more or less copious hæmorrhages, commonest over the upper convexity of the hemispheres, and in the pia-mater here congestion and serous effusion. Rarely had the deeper-seated substance of the brain, either grey or white, undergone visible changes : the ventricles were not distended and their vascular appendages were either pale or but moderately congested. Inflammation of the brain or membranes, is rare in spirillum fever.

The 3 instances in which the brain offered no apparent change, show that this organ may escape altogether, and they join on to the next series of simple vascular repletions : 2 deaths occurred in a febrile stage, 1 in the sequelar.

The conditions of the cerebral circulation, and the facility with which serous transudation takes place from the distended and bare veins of the pia-mater and subarachnoid spaces, will account for the commoner appearances here seen ; and I should add that at least 64 p. c. of the deaths occurred during a febrile state of the system. Decided surface congestion was, however, sometimes present in autopsies of those dying during an apyretic interval, *e.g.* when pneumonia co-existed. Occasionally the blood was fluid enough to flow out of the vessels when first the chest was opened, and hence the degree of their impletion may have been underestimated. The significance of vascular turgescence, with or without attendant serous exudation, is not always alike. Cerebral congestion was either limited to, or most prominent at, the vertex : where, too, alone inflammation was seen, and hæmorrhage was most frequent : it seemed almost always of passive (venous) character and was seldom pronounced or proportionate in the substance of the brain (*puncta* few and tardy) : usually serous effusion co-existed, and repletion commonly persisted after hæmorrhages. The degree of turgescence varied much, and when considerable the accompanying serosity had a more or less reddish tint.

There were two or three instances of active congestion implicating the smaller vessels of pia-mater and brain, and two of decided inflammation ; the transitions are gradual.

Occasionally the cerebral arteries contained blood, their walls being flaccid ; and I thought this might be significant of impeded circulation. In a lad of 14 dying at end of invasion, the veins at vertex of brain were congested ; at the base the arteries contained fluid blood, and on tracing the middle cerebrals into the Sylvian fissure, signs of extravasation were seen around them, but no distinct coagula outside their walls ; and, as in some other examples, it was difficult to say if slight hæmorrhage had occurred or only transudation from some smaller vessels : the influence of gravity was not concerned. Punctate hæmorrhage in the convolutions and pinkish subarachnoid fluid, have been noted, with this state of the arteries.

Serous effusion was practically limited to the loose subarachnoid tissue at the vertex and sides of the brain, gravitating where plentiful towards the base : little was found in the arachnoidal sac : vascular congestion was present in 15 out of 20 instances and 8 times pronounced, when a sodden and semi-opaque condition of the arachnoid might co-exist. The quantity of transudation varied, and might be considerable : its aspect was usually yellowish and clear, sometimes reddish and very rarely turbid. The brain-substance was almost always wet and of diminished consistence (especially in infants and the very young), the pia-mater somewhat soaked or loosened, and convolutions sometimes compressed. Marked serous effusion was most commonly seen at the close of pyrexia (at which time decease is frequent) and very seldom in an apyretic interval : its significance is probably not more considerable here than in other febrile disease.

Pallor as the prominent appearance was seen chiefly at the end of critical fall and during the later sequelar stages of the fever, or after copious hæmorrhages : serous transudation and a wet condition of the brain usually co-existed.

In a considerable proportion of the autopsies I witnessed, there was a pallid, wet, and shrunken state of the cerebral convolutions, combined with slight opacity of the arachnoid and pia-mater loose, pale or congested and wet. This aspect reminded me forcibly of the brain-changes noticed after typhus fever, in England. At two autopsies of complicated cases described by other observers, the brain was termed firm or very firm ; once with meningeal congestion, and once with serous effusion.

Inflammation of the Brain (Meningitis).—This was distinctly manifested in 3 out of 57 autopsies : it was indicated about as often.

An elderly woman with specific fever of uncertain duration, presented symptoms of pneumonia after what was probably the acme of attack : at autopsy a spot of localised inflammation and suppuration of the membranes and adjoining surface of the convolutions, was seen at the vertex of the left hemisphere ; there was some congestion and serous effusion around, and no other change of brain structure : here the cause of the meningitis remains unknown, in the following instance it was doubtless cerebral hæmorrhage.

An old man, famine-immigrant, died on 5th day of the first apyretic interval, two days after the low fever and head-symptoms had rather quickly come on.—*Vide CASE XXIII.*, page 84. At autopsy, dura-mater unchanged. Arachnoid opaque only at seat of inflammation ; subarachnoid space not all over-distended : right side, dark extravasations along median fissure and over middle lobe at vertex, also to a less extent over frontal lobe, but behind none ; between these dark spots, purulent foci and infiltration, with softening around ; l. side, similar but less wide extravasations in middle lobe and similar but more limited purulent infiltration : adhesions along middle line only, and no hæmorrhage in cavity of arachnoid. Base of brain, membranes unchanged, but some sanious fluid in interpeduncular space ; hæmorrhagic spots on under surface of frontal lobe, on contiguous surfaces of longitudinal fissure and in Sylvian fissure of right side. Substance of brain and ventricles : substance soft, wet ; *puncta vasculosa* on section ; r. lateral ventricle distended and its upper cerebral parietes ploughed up by a very large clot, in centre of which two firm rounded masses of fibrine : floor of ventricle unaltered, septum torn yet little blood in the l. cavity. Cerebellum : hæmorrhagic spots and some opacity on upper surface and posterior border of both lobes ; substance unaltered, and 4th ventricle unaffected. Arteries of brain seemingly everywhere pervious ; no atheroma : veins filled. Medulla oblongata not changed : spinal cord and membranes, normal. No valvular disease : lungs inflated and pale ; apex, l. lung the seat of small inflammatory nodules, with pus in them ; no tubercles or adhesions : no splenic or renal infarcts : there were punctate hæmorrhages in stomach and intestines.

There was no history or sign of previous paralysis : the patient's daughter was admitted with him, and died after the relapse with thrombus of the left femoral vein : this coincidence of vascular phenomena is noteworthy.

A woman, 50, admitted in a delirious state with specific fever of unknown duration ; the day before death there was some dilatation of the left pupil and signs of left hemiplegia. After death, pneumonia was found and localised inflammation and suppuration beneath the arachnoid on the right side, at vertex of the hemisphere. These changes were quite isolated.

Head symptoms commonly associated with chronic inflammation of the brain or membranes, may or may not be thus connected in this fever :—

A woman of 35, suffering from chronic mania and in poor health, was seized in hospital with specific fever and died on the 4th day : nothing more than serous effusion, with some congestion, was noticed in the brain.

A man of 30, famine-immigrant, admitted just after invasion-attack, with debility

and delirium passive, and resembling mania, underwent a relapse which was attended with exacerbation of the head-symptoms: 9 days afterwards he sank in a reduced state. The brain was somewhat congested on upper surface (arterial) with some evident opacity of arachnoid; sanious serum one ounce: substance of brain normal; clear serum, one drachm, in lateral ventricles, velum interpositum very pale.

Cerebral Hæmorrhage.—This serious lesion I thought best to describe in connection with its symptoms, amongst the 'Complications' of spirillum fever; and would, therefore, refer to Chapter V. of this Section for the details, page 186.

Pre-existing brain-disease.—Two instances were seen, which illustrate the little influence arterial degeneration and old lesion may have, in the production of febrile meningeal hæmorrhage. In one (F., 55) there was an old clot in the *corpus striatum* and the cerebral arteries were atheromatous: some congestion was present, but no fresh hæmorrhage: death early during invasion, and abundant blood-contamination. In the other (M., 60) also dying at invasion, the arteries were found atheromatous, yet not obstructed; brain-substance soft and wet. Both these subjects were patients infected in hospital. Once a cavity in the left hemisphere was found in the body of a famine-immigrant (M., 30), dying in hospital 6 days subsequent to defervescence: blood-spirillum not seen, the patient probably coming in too late. The cavity may have been remains of a clot, as there was reported a history of fits, and no sign of recent cerebral inflammation or fresh effusion.

I have no minute memorandum on the state of the *Spinal cord*.

Histological Notes.—The brain-tissues were examined whilst fresh, and with no re-agent besides acetic acid. The state of the blood-vessels leading directly to copious cerebral hæmorrhage, was not ascertained; but from the invariable absence of coarse arterial disease, it may be presumed the lesions described below were present. These refer to commoner phenomena amongst the casualties; namely, first, to a state of coma or syncope (the two being connected), concurrent with irritation and accompanied by fatty degeneration of the capillaries and smaller arteries; thrombus also sometimes being present: ex. 1 and 2. Next, a frequent state of the brain, in which head-symptoms are subsidiary, yet local lesion not absent: see examples 3 and 4. Lastly, a condition nearer to pure exhaustion; ex. 5 and 6. The instances here quoted—six in number—were not selected, and are by no means exhaustive.

1. M., 30, Mussulman weaver, admitted on 7th day of illness, t. 103°, p. 114, many spirilla in the blood; the symptoms those of low fever, no delirium. Next day a pseudo-crisis, with copious sweats and much depression, no headache, conjunctivæ injected and yellow: on the following day pyrexia increased, headache and local symptoms generally slight. I marked the case as typical of specific pyrexia with debility; the blood swarmed with spirilla in clusters; a few hours before death, the man became unconscious and restless, skin supple, pupils of normal size and sluggish, breathing 40 and shallow, no stertor, the state syncopic, pulse thready. At the autopsy, lungs pale and collapsed, characteristic lesion of heart, liver, kidney and spleen. There was congestion and serous effusion of the brain, exteriorly, slight opacity of arachnoid; interior of brain pallid and rather soft, suspected extravasation in some convolutions of the Sylvian fissure. Microscopic examination—no hæmorrhage found and two specimens from the suspected spots showed nothing abnormal, a third, however, displayed much fatty degeneration of the walls of vessels and also of tissues immediately around them; in another place were seen accumulations of bright particles in the coats of small vessels, no obstruction of the lumen or impacted contents

visible. Curiously, in the first preparation a living *Filaria* was present, apparently not derived from outside; it was shorter and thicker than the *Fil. sanguinis hominis* of Lewis, dimensions $\frac{1}{1200}$ in. by $\frac{1}{180}$ in., tail acute pointed. The blood of the corpse still showed many clusters of spirilla.

2. M., 24, also a weaver and admitted on 8th day of invasion, a similar low type of fever of the icteroid form; there was a copious eruption of red spots, hæmorrhagic and mixed, conjunctivæ injected and ecchymosed, pupils contracted and equal. He soon became semi-conscious and moaned, respirations 40, shallow and chiefly abdominal, and the nostrils worked. Next day a brief remission and high rise of temp. and death: a few hours previously I noted the skin becoming moist, he was quite unconscious, yet restless, the left arm and leg being less moved than the right, their tone remaining, and left side of face a little distorted; eyes less suffused, pupils contracted. The blood was brownish, thin and imperfectly setting; red discs not much altered, several pale granule-cells and some with oil in them; a few spirilla seen, languid, and a few free granules. This was doubtless the commencement of crisis. At autopsy, the anatomical lesions were numerous and characteristic, including marked diphtheritic enteritis. There was much superficial cerebral congestion with extensive subarachnoid hæmorrhage, and a spot of suspected extravasation in the superior peduncles of the cerebellum. Microscopic examination—at a blood-stained spot on superficies of cerebral convolution: nerve-tubules and ganglion-cell fibrils not visible, their place being taken by a quantity of granular matter; blood-vessels in great part unchanged, but some are dilated and others contain bright granules of large size, resembling fatty particles; besides, there are appearances of fatty degeneration of the vessels and tissue around; the fatty matter has a yellowish tint, but is not to be confounded with debris of red-blood discs. Case referred to as No. LXXXV. Chap. V., and below.

From the above data I infer that the cerebral hæmorrhage in spirillum fever, may be attended with fatty degeneration of the smaller vessels. In the two following examples there was an absence of hæmorrhage, and during life only subordinate head-symptoms; yet, appearances indicate that the brain suffers like, if not equally, as other organs of the body during this infection; and the datum is important. The last examples reveal less advanced change, such as doubtless is common in severe cases of fever; prominent local lesion (probably of similar nature) being found in other organs.

3. M., 22; death on 7th day, with high temp. See CASE XVI., page 75. Autopsy after 3½ hours. Congestion of membranes, no hæmorrhage or opacity, dark blood in cerebral arteries, brain-substance rather soft and wet, convolutions shrunken and pia-mater loose; floor of 4th ventricle streaked with large veins. In blood at death no spirilla found, but some large granule-cells, not fatty, Micr. ex. of floor of ventricle towards upper end: there are streaks or stains not seen in other parts, extending below the surface, and still deeper small vascular spots are visible, especially on the l. side at upper end, near a dark nucleus of origin of a cranial nerve (? *par vagum*); no alteration of consistence. The prominent change here is the accumulation of white cells in the perivascular spaces, which is sometimes so large as to resemble extravasation and distinctly encroach on the nerve-tissue; the cells are uniform and clear: none of the large granule-corpuscles of the blood are here visible. In some places there seems to be thickening of the walls of the larger vessels, with smaller accumulations of round cells between their layers: whether this be a proliferation of connective-tissue corpuscles, or (more likely) a gathering of wandered cells from the blood, may be questionable or small apparently unchanged. There is no fatty degeneration of the capillaries or small arteries: the white-cell exudation seems to be connected with venous radicles.

My figures show a state of the brain-tissue, which might be termed leucocytic inflammation, extravasation or thrombus-formation; and the site of lesion may have been connected with the depressed circulatory and respiratory symptoms, noted before death.

4. M., 25. d. 6th day? with low temp. See CASE LXXXIV., Chap. V. Autopsy after $1\frac{1}{2}$ hour. Congestion of membranes, sub-serous effusion, shrinking of convolutions, brain-substance unaltered; two veins seen on floor of 4th ventricle, hardly abnormal in aspect. Mic. ex. of this spot—no morbid change around the clot-filled veins; $\frac{1}{4}$ below medium furrow was a larger vessel containing a clot, and around this spot the brain-substance was studded with many *corpora amylacea* of different sizes, some being in clusters; blood-vessels seemingly unchanged, and no white-cell infiltration; nerve-tissue probably unchanged. Corp. amyl. were not seen beneath the lining membrane of ventricle. As the patient had been ill only 6 days and showed no head-symptoms at the last, it is probable these amyloid bodies had no connection with his illness: the thrombi were doubtless associated with the contaminated state of the blood.

5. M., 34. d. on 15th day. CASE LXXXIII. Gastric hæmorrhage. Autopsy after 4 hours. Some congestion and turbid effusion; brain-substance rather wet and soft, no increased vascularity; floor of 4th ventricle streaked and stained a little. Mic. ex. of the part; there are several distinct hæmorrhagic spots in the floor of the ventricle, and a few are found at $\frac{1}{8}$ to $\frac{1}{4}$ in. beneath the surface; here, too, some vessels are distended with blood without being diseased; amongst their contents is a large amount of clumpy protoplasmic matter which resists the action of acetic acid and is like that elsewhere found in the blood; white cells here few.

6. M., 23. d. on 9th day, temp. not high, moribund on admission; many spirilla in the blood. At autopsy intense pulmonary congestion with apoplexy, splenic infarct, vascular patches in the intestines; much meningeal congestion, brain-substance slightly softened, pallid within; suspected extravasation in *crus cerebri*. Minute examination of this spot, just above the locus niger; there is some softening and staining of the tissue, vessels at various depths distended and of bright red hue; actual extravasation not seen. It is likely that emboli were present: spirilla not visible within the vessels: they are always most difficult to see in unstained specimens.

2. **The Lungs.**—On account of their exceeding vascularity and elasticity, combined with scantiness of parenchyma, the lungs at autopsy are apt to vary extremely in volume, aspect and weight, even when disease is not suspected; and as in spirillum fever no specific pulmonary changes have been recognisable, the usual record of post-mortem appearances necessarily seems somewhat vague. The larynx was rarely examined, there hardly ever being symptoms referable to this part.

The mean normal weight of either lung, has been estimated at 7, 14 and 21 ozs.; as such a wide range interferes with much particular inference in disease, it would be worth while studying the conditions leading to variation of post-mortem lung-weights.

In 74 autopsies the lungs were regarded as being in a quasi-normal state 8 or 10 times, and this oftenest in deaths during the pyrexial stages.

The entire lung on either side was pale and inflated, or pale and collapsed, 21 times, of which 17 deaths in febrile stages; this is a noteworthy circumstance, even if it be assumed that the conditions immediately preceding decease are chiefly concerned: of the patients' symptoms at the agony there are seldom any records, but two examples are given below. A certain degree of congestion of the lung-tissue (behind) and of the bronchial mucous membrane, with a varying amount of serous exudation (usually scanty) was commonly present, and sometimes lobular solidification, especially in deaths during pyrexia; but the general bloodless aspect of the lungs most impressed me, as being significant of impeded pulmonary circulation from altered quality of the blood.

The expanded or collapsed state seen upon opening the chest is explicable in the usual manner; persistent lung-inflation, independent

of adhesions (very rare here), indicates impaired elasticity and to all appearance may be rapidly induced : it was seen oftenest and late (viz. chiefly in deaths towards close of invasion-attack) ; and in still later deaths (viz. at critical fall), was succeeded by the congested condition.

Partial paleness, with collapse or inflation, was found in many of the remaining inspections, when other more manifest changes were present : lobular collapse, especially, is apt to be overlooked or misinterpreted.

M., 22 (student), dying in the height of fever at 12.45 noon ; at 11 A.M. the chief symptom was cardiac weakness, the pulse being hardly perceptible and uncountably frequent : no cough or dyspnoea, but respirations 70 per minute and very shallow (chiefly abdominal) ; the belly full, tense and very tender in the hypochondria ; at 12.30, it is reported 'became suddenly insensible, respiration greatly embarrassed and gasping ; he moans loudly.' 3½ hours after death the lungs were found to be pale, not collapsing, everywhere inflated and crepitant ; no hypostatic congestion, bronchial mucous membrane reddish only : no adhesions ; there were pale clots in the heart's cavities ; weight of both lungs in this lad, 20 ozs. CASE XVI., Chap. III.

M., 25, dying at reputed 5th day of fever, presented 6 hours before death a peculiar state of depression : moans, yet is quite rational, breathing 50 very shallow, pulse 140, very soft ; he is recumbent but restless and turns on side, with knees drawn up : dyspnoea or urgent breathing, with full and tender belly, are the most prominent signs. 1½ hours after death, the lungs were pale and collapsed, and greatly compressed by the enormous liver which projected to level of 3rd costal cartilage on r. side ; diaphragm pushed up by distended stomach and enlarged spleen on l. side : lungs nearly void of air and blood, pallid and as if emphysematous in general, with collapsed areas of purple hue, depressed, solidified and sinking in water, most extensive at posterior borders ; several sub-pleural hæmorrhagic spots : lungs weigh R. 12 and L. 11 ozs. ; both alike in aspect. Death 15 hours after admission, when the blood was crammed with spirilla ; both the young men engaged in this autopsy became infected, and one died. CASE LXXXIV. Chap. V.

A similar condition of the lungs is frequent at death supervening upon that form of cerebral hæmorrhage, which occurs in spirillum fever ; thus, in the instances narrated above, after excluding one of localised pneumonia, the lungs were either healthy-looking, or pale and inflated or collapsed ; and in the two cases where vascular engorgement was co-present, it is described as very marked and defined lobular congestion at the back part or as disseminated pulmonary apoplexy in small patches : in all these cases the weight of the lungs was light, the means being 8-9 ozs. each.

Congestion.—As the prominent condition was noted 13 times, chiefly limited to the febrile periods when the pneumonia begins, which is so often developed in the succeeding apyretic intervals. The lungs may be but moderately congested after death in high fever with insensibility : commonly it is the bases and back part which are most implicated, and the form and degree of vascular turgescence are as variable here as in the brain and other viscera. Instead of being diffused, it may be limited to a collection of lobules and so resemble lobular collapse, inflammation (early stage) or apoplexy, the state of congestion preceding or supervening upon each of these conditions. True hypostatic congestion was comparatively unusual in the brief spirillum fever, being chiefly noted in the typhus-like cases occurring amongst Mussulman weavers and the more destitute : in such instances, the anterior part of the lungs might be in a state of collapse ; and on the other hand the hypostatic congestion be

attended with pulmonary apoplexy, which would account for the slight hæmoptysis sometimes noted during life.

M., æt. 23, a miserable-looking subject, admitted in a moribund state at the end of invasion-attack: many aggregated spirilla in the rather thick blood. Hypostatic congestion of both lungs and hæmorrhagic patches in both, at posterior part; bronchial mucous membrane intensely injected and tubes filled with frothy, red serum. Pyrexia not high; dyspnœa the most prominent symptom. There were infarcts in the spleen and minute hæmorrhages in the brain and intestinal canal: jaundice was present.

Pneumonia: Pleuro-pneumonia and Pleurisy.—These topics have been considered in connection with their symptoms under the heading of 'Complications' (Chapter V. of this Section, page 201).

As instances of some ultimate lung-changes, I add here memoranda of two cases not verified microscopically, but almost certainly belonging to the spirillar series.

Gangrene of the lung. M., 30, a recent famine-immigrant, admitted with a history of relapsing fever and dying 10 days afterwards. There was double basic pneumonia at the stage of suppuration, and a large gangrenous cavity on the right side: other viscera little changed in aspect.

M., 27, admitted at height of the epidemic in a low state and jaundiced, underwent a relapse during which an anal abscess formed; diarrhœa and symptoms comparable to phthisis ensued, and he died a month later. Both lungs were adherent, the left congested; the right, in its upper lobe contained a large cavity filled with pus and having sloughy walls: liver large and fatty, the other organs little changed.

The following cases illustrate a state of the lung best regarded, I should think, as acute œdema.

M., æt. 46, in good condition, died the day after cessation of a well-marked relapse; the temp. subsiding to $100^{\circ}5$ in the interim, but promptly rising to 103° a few hours before decease: there was then low delirium, shallow, troubled respiration of 50 per minute, universal moist sounds and impaired resonance. Lungs inflated but pale, corrugated at apices but free of deposit; pallid on section but peculiarly frothy, there being a universal diffusion of serum and minute air bubbles. Weights $14\frac{1}{2}$ (right) and 13 ozs. Heart contracted, left side quite empty, valves and substance healthy looking: cerebral congestion, splenic infarcts and a large flabby liver: jaundice.

M., æt. 40, died in 36 hours at the crisis of first attack (last t. noted 95°), respiration gasping as in asthma but not asthmatic, and compared in my notes to that of cardiac embolism: spirilla in the blood at first swarming, then few with some large fatty and endothelial cells. Lungs collapsed and substance generally dry, but base of left lung infiltrated with frothy liquid, as in capillary bronchitis, yet without increased vascularity; the tissue floats deep in water; weights 18 ozs. (right) and 15 ozs. Heart (9 ozs.), right side contains a large, firm, decolorised coagulum extending into the pulmonary arteries to their end; left side contains a small black clot only: kidneys showed early granulation.

Hæmorrhage.—Occurring towards or at the close of primary fever, pulmonary hæmorrhage assumes one or both of two forms, at least; and in either, general congestion of the lung may be present or absent. In pulmonary apoplexy the lungs have been found pale and inflated, whilst both were studded throughout with patches (apoplectic) in size from a pea to a peach; no inflammation: co-existing petechiæ in the anterior mediastinum, and larger effusions elsewhere. In 2 other cases, there was congestion of the lower and back part of the lungs accompanied by apoplectic consolidation.

In the petechioid or sub-pleural form, similar hæmorrhages were always found elsewhere in the body, so that it may be said the lungs (highly vascular as they are) are not peculiarly predisposed to sanguineous effusion; the common site was the surface of the lower lobes; congestion if present, was hypostatic. An instance may be appended of the four in my list:—

M., 24, suffering from the typhus-like form of spirillum fever, died during the first *perturbatio critica* (last t. 105°·8). R. lung intensely gorged with black blood (hypostatic); pieces float deeply and crepitate somewhat, the tint brightens on exposure, there are sub-pleural hæmorrhagic spots at the back of the lower lobe, weight 20 ozs.; left lung, in a similar condition, weight 18 ozs.; in front the lungs are collapsed; no pulmonary apoplexy: there were hæmorrhages in the brain and elsewhere.

It is a striking circumstance that 'tubercle' in any form, state or site, was never noted in the whole series of 74 autopsies.

Histological Note.—The following observation refers to that localised collapse of lung-tissue, which has been above mentioned: it is instructive.

M., 25, died soon after admission, with symptoms of urgent dyspnœa: many spirilla in the blood. Lungs pale and collapsed, and so greatly compressed by the projecting abdominal viscera, as to be nearly void of air and blood: emphysematous in general, they show depressed and purple-hued areas of solidified and heavier tissue, most extensive at middle of posterior border. Also sub-pleural petechiæ. Micr. ex. The consolidated tissue is dry and solidified in an extent not defined as lobules, but interspersed with aerated patches in which the air-vesicles seem only small in size: no extravasation of blood, but walls of vesicles deeply congested and in parts encroaching on the cavity: even here there is little less blood, and the idea imparted is that of thrombus in the pulmonary capillaries. White cells not increased; there are some rounded collections of oil granules (besides some free), which correspond to the large fatty cells seen in the blood; spirilla not visible, and cause of embolism or thrombus not clearly made out. The chief points here, are the intense congestion of the solidified areas and also of the surrounding lung-tissue, with no extravasated blood and no cell-production of inflammatory aspect.

The Heart.—A marked change in aspect of the heart whether transitory or structural was by no means invariably noted, and the brief descriptions of this organ contained in my records chiefly refer to size, appearance and arrangement of the blood-clots found in auricles and right ventricle: it is probable that alterations in the heart-muscle were overlooked, yet they seem not so frequent as might be anticipated. Recent endocardiac or valvular disease, in direct connection with the fever was not detected: the sub-serous petechiæ were but examples of a widely-distributed change, and here, as elsewhere, inflammation was seen in connection with the hæmorrhage as perhaps the most characteristic event of this disease. Besides occasional pallor, a friable consistence of the thicker muscular walls might be noted; and the organ commonly appeared to be large from distension of its flabby parietes. It even proved to be heavier than usual, especially at advanced stages of the fever; its weight being often entered as 9–11 ozs., and that not in elderly subjects.

Upon a few occasions (7), the heart appeared so little altered that the term 'healthy' was applied; they all related to deaths during febrile periods, chiefly towards the close.

Blood-clots within the organ commonly attracted attention; very seldom was the heart empty, and then it might have been accidentally deprived of its contents, especially when it happened that these were in a diffuent condition. Clots of varied aspect are the chief entries 48 times (65 p. c. of all autopsies): in at least one-quarter of these cases, the clot was nearly or wholly limited to the right side, or in near two-thirds if the critical periods and apyretic intervals of sickness be alone considered: when present on both sides of the heart, those in the right cavities were almost invariably larger, paler and more adherent: clots limited altogether to the left auricle or ventricle were never seen. These data would be useful were the conditions known under which blood collects in the heart or is detained, coagulates and becomes partly decolorised; I have not attempted to elicit these conditions, and only observe that the hour after death when the autopsy was made did not seem to influence the aspect and disposition of blood-clots. Thus, the mean interval was 7 hours for both series of large, pale clots and no clots, and even a more or less fluid state of the blood might be present after an interval twice as long as this; hence neither coagulation nor decolorisation is an invariable post-mortem phenomenon. The more striking isolation and retention of blood-fibrin did not appear to follow any one mode of death, or vigorous or feeble action of the heart (as indicated by aspect of the heart-muscle), or visible state of the blood as related or not to stage of fever alone, anæmia or the reverse, malarious cachexia, results of hæmorrhage, differences of sex and age, or finally to the conjoined state of the pulmonary organs, as for example pneumonia, in 14 cases of which with clots more or less blanched on the right side, coagula were nearly or wholly absent on the left 7 times.

The various aspects of the contents of auricle or ventricle may be arranged as follows:—1. Blood wholly fluid (usually dark and most abundant on r. side) or mingled with small clots, black or pallid and non-adherent.

2. Coagula either uniformly dark and loose, or pale in interior and loose, or with pallid exterior surface and adherent.

3. Decolorised masses of varied hue, consistence and volume, more or less adherent in ventricle or auricular appendage, and nearly always limited to the r. side.

Illustrations are as follows:—

F., 35, caught fever in hospital and died on 4th day, last temp. 104° : a little clear serum in pericardium; right cavities filled with a large, pale and adherent clot, which extended into the pulmonary artery; left auricle was also filled with a pallid, less adherent clot, the left ventricle contained a small, dark, loose coagulum limited to its cavity: heart-muscle and valves healthy looking: weight of organ 7 ozs.: lungs of normal aspect and volume, somewhat congested posteriorly. The patient was emaciated and probably died of exhaustion due to blood-contamination and pyrexia: the above appearances are almost typical.

M., 25, died on reputed 5th day of invasion-attack: clear serum in pericardium, r. side of heart flaccid (now), l. side firm; dark clots on both sides, loosely formed and non-adherent (blood still partly fluid): heart substance firm, red. Some sub-serous petechiæ: the man had jaundice. Lungs pale, collapsed and greatly compressed by thrusting up of the diaphragm: autopsy $1\frac{1}{2}$ hrs. after death.

M., 60, died in stage of semi-collapse at close of invasion-attack: many spirilla still in the blood: clear serum 1 oz. in pericardium, a large, pale, adherent clot in r. cavities passing into pulmonary arteries; a similar but larger and longer clot in l.

cavities, also adherent and extending a considerable distance within the aorta: substance and valves healthy, weight 12 ozs. Lungs congested, but otherwise healthy, weights 24 and 18 ozs. In another man of 60 dying during pyrexia, a large, pale and adherent clot, extending into the aorta, was found in the left ventricle; here was atheroma of the aorta and ossification of the coronary arteries: lungs healthy looking.

Small quantities of serum, clear, seldom turbid, yellowish even when no jaundice apparent, were frequently found in the pericardial sac.

Cardiac Hæmorrhages.—These were limited to the sub-serous tissue, and almost exclusively to the sub-pericardial: they were petechial in aspect and hardly ever attended with signs of irritation: they were found after death occurring at the close of first attack, and almost always contemporaneously with petechiæ elsewhere. Their usual site was near the base of the heart, front or back, but they were also seen beneath the parietal layer of the pericardium.

Illustrations are the following:—

F., 30, hospital-nurse, characteristically affected with fever of typhus-type. Pericardium nearly empty: a few hæmorrhagic spots beneath the parietal layer of the sac, and others beneath the visceral layer over the base of the heart at the back, where some are of considerable size: no sub-endocardial hæmorrhage, but spots in the brain, lungs, kidneys, intestinal canal and skin. There were blood clots on the r. side of the heart (pale on their outer surface): lung: inflated, congested behind.

M., 32, died immediately after the critical fall (first attack), seemingly from exhaustion, after prolonged death agony: large black clots were found in both right cavities and in the left auricle, l. ventricle contracted: numerous bright-red, hæmorrhagic spots over anterior surface of the heart, small and superficial, no pericardial effusion or opacity. Lungs everywhere crepitant, inflated: some bronchitis and behind congestion: no other petechiæ noticed, but at the *perturbatio critica* there had occurred copious and repeated epistaxis.

Hæmorrhagic Pericarditis.—This event was rare in Bombay, for only one autopsy was available of this fatal complication. See 'Complications': Case LXXVII, page 208.

M., 30, washerman, admitted near close of invasion, very ill, being highly jaundiced and delirious: blood-spirilla numerous, large, and with rather a sluggish movement; the critical fall gradual, seeming convalescence established on the 10th day of apyrexia, when a well-defined relapse took place, lasting seven days and ending with sudden fall of t. and death (spirilla numerous just before): the pulse failed 40 hours prior to decease. Autopsy 2½ hours afterwards. Body emaciated, rigor mortis present, tissues stained yellow, blood semi-fluid and dark-coloured. Head—much dark, grumous blood in arachnoidal sac, chiefly on l. side, no trace of inflammation or arterial obstruction, except a small, pale nodule felt in a communicating artery, but not obstructing its channel: brain congested, substance firm, healthy; weight 34½ ozs. Chest—Heart: 2 ozs. of bloody fluid in pericardial sac, both serous layers highly inflamed, covered with villous lymph (readily scraped off) and marked with patches of hæmorrhage; heart contracted, muscular substance pale; a small partly decolorised clot in r. side, l. ventricle more firmly closed, but containing a scanty pale clot: weight of organ 7¼ ozs. Lungs: collapsed: structure healthy; weights 8½ (r.) and 7 ozs. Abdomen—no fluid in peritoneum. Liver: normal size, surface smooth, aspect dark, structure congested yet of normal appearance, weight 2 lbs. 14½ ozs. Spleen: enlarged, capsule thickened in one or two places, surface mottled and beneath the paler parts were infarcted masses in the interior: pulp generally somewhat indurated and having diffused throughout a large number of pale red masses; the more superficial of these had a deep yellow colour and were well-defined: those exceeding 1 inch in diameter were softening in the centre: there were also hæmorrhagic patches throughout the spleen pulp: weight 14 ozs. or nearly three times the normal. Kidneys: size normal, capsule easily separated, surface mottled yellow and

ered from hæmorrhagic stains; on section, structure firm and as if infiltrated with some yellowish deposit: weights $3\frac{3}{4}$ (r.) and $3\frac{1}{2}$ ozs. Intestinal canal—small intestine: congestive and hæmorrhagic spots in the mucous membrane which had concentrated around Peyer's patches, especially at one spot about a foot above the ileo-cæcal valve; no ulceration; higher up the *valvule conniventes* were of deep hæmorrhagic colour, but this hue gradually subsided as the *jejunum* was approached. Large intestines: mucous membrane of deep leaden hue, especially in the cæcum. Mesenteric glands nowhere particularly enlarged. (From notes by Mr. Sukharam Arjun). It seemed to me that the hæmorrhage into the arachnoid was of some days' standing, or at least gradually effused; that into the pericardium appeared to be more recent: there was no defined deposit in the kidneys: the condition of Peyer's patches was this—raised, granular in aspect, defined and of deep blood hue; the mucous membrane around for some inches being highly congested and blood-stained. Total duration of illness 21 days (possibly 3 or 4 days longer); time between convalescence or onset of relapse and death 7 days; highest temp. in relapse 103° , quickest pulse 126, no special symptoms noted till vomiting and collapse at the end. The temperature and pulse chart is not peculiarly different from many of ordinary relapsing fever.

Organic disease of the heart was seldom found in this series of autopsies. There is one instance of an adult man dying towards the end of a first attack of icterus type, when some thin fine bands of old adhesion and a milk-white patch were noticed in the pericardium: numerous petechiæ in lungs and intestines: kidneys enlarged and mottled, probably fatty.

M., 30, died at the first critical fall, of double pneumonia; on admission 4 days previously a loud systolic murmur was heard, most distinct at mid-sternum and ensiform cartilage; there was also dyspnœa and palpitation, these symptoms, with pain, being estimated as of 15 years' duration. After death the mitral valve was found thickened, opaque and narrowing its orifice; there were pale, adherent clots in the cavities and large vessels on each side of the heart: both lungs were hepatised in their lower lobes.

Histological Notes.—There is only a general accordance between the results of naked-eye and minute scrutiny of the heart-muscle; and I find its pale, friable condition is not necessarily associated with such fatty degeneration, as was not unnaturally assumed to be present at some of the autopsies. 14 examinations were made, of which 9 in deaths during invasion, 1 at first interval, 2 at the relapse, 1 at second interval and 1 at sequelar stage.

Of the first 9, the organ was apparently and really unimpaired in structure in 2; whence it is evident that death (and even prompt death) at this time, may take place without evident lesion of the heart. In 7 there was some change, but seldom considerable and never extreme. This change might be regarded as a granular degeneration of the muscular fibres extending from the position of their central nuclei, in the longitudinal direction; and being often attended with pigmentary accumulations. Neither in this stage of fever nor any succeeding one (whether febrile or non-febrile) did I meet with genuine fatty degeneration of the heart-muscle; the sole exception referring to the case of an old man (æt. 60) dying at the sequel, and whose other organs were fatty. In another man of 60 (the remaining patients were young adults or children), the coronary arteries were atheromatous and the left cavities contained large pale and adherent clots, so that the probability of a feeble heart seemed evident; yet central pigmentary degeneration of some

muscular fibres, was the chief lesion found. This patient was admitted for chronic rheumatism, acquired infection and died in hospital, on 5th day of attack: there were very scanty signs of fat in the renal cells. Other instances are the following, the same method of examination being adopted: portions of the left ventricle the selected parts, and acids, alkali and ether being alone used as re-agents.

F., 30, d. near end of invasion. There were many petechial skin-spots, and some beneath both pericardial layers; muscular substance of heart pale and soft (autopsy after 6 hours). Mic. ex. the fibres look coarse, yet they clear up on the addition of acetic acid, excepting some rows of yellow granules of different dimensions and form, which are situated chiefly between the fibres, but also in their interior: in the former case they seem to belong to the capillaries, and in the latter to be connected with the nuclei of the fibres, though sometimes found at their exterior. These granules do not resemble fat, and do not run together: they are placed at both ends of the long axis of the nucleus, gradually dwindling in amount as they recede in a longitudinal direction. This appearance was the ordinary one, and at first was mistaken for a fatty change; it soon however became evident that that view was not correct. See Plate II, Fig. R.

M., 22, d. about the same time as the above, and probably from the same infection. Autopsy 3½ hours after death. Muscular substance of heart red and firm: examined after being in spirit for a few hours, the fibres seemed friable, had almost lost their transverse striæ and readily split up; they looked as if converted into fibrous tissue, or affected by a waxy change; there was very little fat and few granules, colour rather paler than usual. On the addition of acetic acid all cleared up, the fibrils still retaining some longitudinal markings: granules and fat very scanty. Here the change was regarded as one of fibroid degeneration, and it contrasted strongly with that described above; being neither granular nor fatty. This patient seemed to die of cardiac debility, there was no suspicion of syphilis: the fibres did not show a tendency to separate transversely. Weight of heart 7 ozs.

M., 25, died on reputed 6th day of invasion: (*Typhus biliosus*), autopsy after 1½ hours. Sub-serous petechiæ outside the heart; substance firm, red; weight of organ 8 ozs. After action of spirit for three days; fibres rather firm and friable, splitting longitudinally and fibrillæ readily separating in this direction; sarcolemma indistinct: transverse division of the fibres not prominent; striæ very clear, though coarse-looking; no fat, fibrous tissues distinct. On the addition of acetic acid all clears up except some central rows of granules which are placed at either end of some of the nuclei, running in the length of the fibre: these granules vary in amount in different fibres, they do not resemble fat and may possibly sometimes arise by proliferation of the nuclei, but not always; for some rows of particles lie at a distance from the nuclei. The change is like that first named above.

In a man dying during first apyretic Interval, the heart-muscle being described as reddish and soft, the fibres were found very clear and so the striæ; yet there was a wide prevalence of central pigmentary degeneration. In a woman dying at the close of the first relapse, the heart seemed healthy, and the fibres though coarse-looking, displayed the striæ very clearly in most cases: and in another case, where the heart-muscle was pallid, granular degeneration of the fibrils was alone found. In a young man dying after the relapse, the muscular fibres of the heart appeared to be hardly changed; some yellow granules were seen around their nuclei.

Aortic erosion.—Instances like the following may have been overlooked. M., 24, d. on 9th day (? acme) of invasion-attack, symptoms those of *typhus biliosus*. The blood at the last contained a few spirilla and several pale granular cells, some with fat in them. Limited cerebral hæmorrhage, kidneys albuminoid and fatty, spleen small, liver large and pale; there was a distinct granular (diphtheritic) exudation in the mucous membrane of the ileum (lower end) and cæcum—no ulceration: the small cerebral vessels at site of hæmorrhage contained fatty granules, and were irregularly distended, but no embolus or thrombus was made out. The aorta showed just above the semilunar valves a few bright-red spots, which with a pocket lens were found to be excoriations of the inner membrane covered with a thin, granular layer; and all down the main aortic trunk there was found a delicate interrupted layer of

pale, granular material. Atheroma not present in this young subject. Mic. ex. the aortic erosion was depressed on the surface; section of the vessel (hardened in chromic acid) showed the whitish, softened appearance to extend through the middle coat as far as the outer: nothing more than abundance of fat was seen in this spot, but elsewhere the deposit was found to inclose also flattened endothelial scales; and from the abdominal aorta a specimen was obtained displaying such scales loaded with oil globules, and readily to be detached from the lining membrane. Hence the idea that sometimes the fatty endothelium seen in living blood during and just after pyrexia, may be derived from the vascular endothelia proliferating and degenerating under specific blood-irritation. The diphtheritic diathesis in this case was, however, peculiar; and I did not notice similar appearances again.

The Abdominal Cavity.—The cavity of the peritoneal seldom contained an excessive quantity of liquid, serosity not being much augmented, even when the liver and spleen were greatly implicated, or the sub-serous vessels in parts distended to rupture. When the blood is unusually diffuent (as occurs in anæmic and deeply jaundiced subjects), with other evidence of impeded circulation in the form of petechiæ, there may be an increase of yellow-tinted serum.

Opacity, adhesions old or recent, the presence of lymph and hyperæmia were extremely unusual, and always limited and traceable to incidental cause and connection. A sodden aspect was rare, and tubercle never seen.

Sub-serous hæmorrhages were not uncommon over the viscera, in omenta and on the parietes; and when abundant, may extend to the subjacent fibrous muscular and glandular tissues. Extensive extravasation was seen only in the diaphragm and its crura, and in the right *rectus abdominis* muscle above.

In general, the peritoneum seems to be less liable to lesion in spirillum fever, than any of the other great serous membranes.

The muscular walls of abdomen were rarely implicated to a marked degree; softening of the *recti* near the pubes was not recorded.

The Liver.—Like the lungs, the highly vascular liver is capable of assuming much variety of aspect within quasi-normal limits, and to this also conduces its superadded portal and biliary systems of vessels; hence in a paroxysmal fever such as the spirillar, its appearance differs at different dates, and from varying special implication, sometimes also in cases of similar duration and intensity. In this infection the blood-circulation is especially implicated; and as by vessels the liver is closely associated with the spleen and stomach, it will be useful to consider the chief abdominal changes together, after dealing with each group separately. The mean normal weight of the liver in adult Natives of India may be estimated at 38 to 45 ozs. avoirdupois; the organ is probably larger, proportionately to body-weight, than in Europeans: changes due to stimulating diet and intemperance, are much less frequent, but, on the other hand, malarious influence is a constant disturbing item unknown in Europe. The following data should be considered along with those furnished in Chapter III. *sub voce*.

In 4 of 74 autopsies (5·4 per cent.) the liver seemed normal; of these two dated as sequelæ, a third also several days after fever, and the fourth very early in first febrile attack. From this summary it is likely that some morbid change was either imminent or subsiding, and that

the term 'normal' is not strictly applicable here : see also below. Enlargement was the most prominent condition in one-half the inspections, but very seldom noted as the sole change. When the enlargement was pronounced, the liver exceeded 3 lbs. (48 ozs.) in weight ; it was then associated most often with a pale or mottled aspect (24 in 46 inspections) or with congestion of the organ (16 in 46), and commonly with a somewhat diminished consistence. Where the enlargement was slight the liver weighed under 3 lbs. (in adults) congestion was here the ordinary accompaniment (11 in 20 inspections) or sometimes a pale or mottled aspect (6 in 20). The maximum weight measured was 6 lbs. 8 ozs. in a man of 25 dying on 32nd day after an invasion-attack with jaundice and pneumonia, the liver was in the state of nutmeg-congestion, and there was a spleen of 5 lbs. (ague-cake) ; here the excessive enlargement was incidental to a connection with prior malarious influence and intemperance ; the minimum weight measured was 8 ozs. in an infant of 7 months, dying towards end of first febrile attack (spleen $2\frac{1}{2}$ ozs.) Increase of volume was nearly twice as frequent in autopsies of fevered patients, as in those dying during non-febrile periods (60 and 33 p. c. respectively) ; and enlargement conjoined with other change, was even more preponderating.

In cases at Bombay resembling the so-called *typhus biliosus*, the size of the liver was not different to the average ; indication of acute atrophy was so rare, that only one instance occurred of the liver being below the mean weight during fever (2 lbs. 4 ozs., death in the first relapse), patient a man of 50. There does not appear to be any anatomical difference between this form of spirillum fever, and the ordinary severe kind, so far as regards the state of the liver.

Pallor and mottling.—A general or partial paleness of the liver, was one of the most striking features at autopsy ; 17 times it was the most prominent character, and much oftener was witnessed a minor degree. There are shades of transition in all these morbid appearances, which need not be separately enumerated. Commonly the liver was enlarged as well as pale, seldom much congested and sometimes even bloodless in aspect : the hue was yellowish-grey, rarely brown, and though the changes here indicated pertain mainly to fatty degeneration, there was not seen that translucency and perfect smoothness belonging to the fatty liver in chronic disease. The entire organ may be thus affected, or one lobe more than another, or parts and sections of any : in the latter case, there ensues a mottling of the natural or artificial surface, and the pale patches sometimes seem tumefied. Fatty changes are probably the rule, though not perceptible to the untrained eye when partial, slight and diffused : they early attend parenchymatous inflammation. The substance of the liver is less firm than normal, easily torn, and not displaying with clearness the swollen lobules. The aspect of lardaceous disease was practically never noted ; and in no instance that I remember was there any induration, general or partial, of the liver-substance.

In a comparatively small group of cases, the liver whilst enlarged and palish was of deeper yellow tint, flabby consistence, somewhat nodulated on the surface ; and, on close inspection, the interlobular tissue seemed lax and dark, whilst the lobules became more defined and prominent, and the whole aspect resembled that of incipient cirrhosis.

All the patients were male Mussulmans, generally from N. India, malarious subjects, and certainly the majority not drunkards.

The connection of the two series here distinguished, is not obvious : probably their differences depend chiefly upon vascular changes, since no uniformity of tissue-lesion was apparent in the quasi-cirrhotic group (see below).

The signs of chronic interstitial hepatitis amongst the entire number of autopsies, at Bombay, were never met with in even mild degree ; and this feature strongly contrasts with experience in Europe, where true cirrhosis is comparatively frequent.

Congestion.—This was entered as the prominent condition in some instances not minutely described ; being sometimes specialised as portal or hepatic, but commonly repletion of both sets of vessels was understood. I have occasionally remarked a highly turgid state of the portal vein and its branches ; as, for example, in a man of 30, dying on fifth day of first relapse with cerebral hæmorrhage, the whole organ being of enormous size, firm and of uniform aspect. The spleen was also very large, and free from mottling. Congestion is noted oftenest immediately after cessation of fever, or a little later. Minor degrees were common, being of the passive form and probably connected with impeded circulation through the lungs and heart ; but in no uncomplicated casualty was there any approach to the nutmeg-aspect. Considering the extent to which the liver is implicated during spirillum infection, it is remarkable how seldom there could, at any time, be said to be a state of active congestion : possibly this precedes the parenchymatous inflammation, and if so its duration is brief, or its persistence incompatible with swelling of the hepatic cells.

Diffused hepatitis as an attendant on spirillum fever must be very rare, notwithstanding the seeming urgency of local symptoms during the height of pyrexia ; and the following is the only instance met with, to which this term would be applicable. The clinical history was necessarily defective, and no microscopic examination of the tissues was made.

CASE CI.—M., 14, admitted on reputed 5th day of specific fever (blood highly recharged with spirilla), died seven hours afterwards in a very low state : no jaundice or petechiæ. Liver : very large for a lad (55 ozs.) actively congested, yellow-red in colour and mottled with large, pale patches found on section to extend deeply, and the like to be disseminated throughout, all being of irregular size and form, ill-defined and often surrounded by a vascular zone ; these patches were not apparently due to deposit, but rather to acute fatty degeneration of large portions of the liver gland ; gall bladder contained dark, viscid bile, no obstruction to ducts. Spleen 11 ozs., dark, firm and containing pale patches (so-called infarcts) similar to those found in the liver : kidneys congested. There was yellow fluid in the peritoneal sac ; no general peritonitis but the largely exposed upper surface of the liver (both right and left lobes) was smeared over with a little soft lymph ; this was valid evidence of serous inflammation, attending the parenchymatous, and hence of a general hepatitis.

Acute abscess of the liver, whether single, multiple or metastatic, was never traced directly to the spirillar infection ; and, in this respect, experience at Bombay seems to have been peculiar. Hepatic abscess concurrent with the fever, was alluded to in Chapter V. : see CASE XC.

The only form of hæmorrhage seen in connection with the liver, was in the form of sub-serous petechiæ, of which a large collection was once

noted upon the left lobe ; similar extravasations being found elsewhere in the peritoneum.

The state of the Gall-bladder and aspect of the bile appeared not to maintain any constant relation to liver-change ; departures from normal range being most rarely observed in either.

Histological Notes.—The minute structural changes which I have seen, consist chiefly of cloudy swelling, and pigmentary or fatty transformation of the gland-cells ; the result being enlargement of the individual lobules of the liver, and hence of the entire organ. A much less frequent change, conducing also to the same end, is the production of leucocytes in the connective tissue connecting the lobules. The blood-vessels rarely were changed in aspect, and alterations of the bile-ducts were not noted. Free crystalline bodies were very rare. The tissues were examined in fresh state, and with simplest re-agents.

It is probable none of these appearances, in the abstract, is peculiar to the spirillum fever : here, as in other fevers, early enlargement of the hepatic cells is attended with increase of their nuclei, often two and sometimes three, becoming visible on the addition of acetic acid. The degree of change was often remarkable, and some varieties are noted below.

So far as I know, the extremely localised cell-degeneration sometimes evident, may be peculiar to this infection ; and it would indicate stasis of circulation in the finer branches of the portal vein.

As to course of morbid alterations, there is, earliest, enlargement of the liver with uniform or disseminated pallor, and friable consistence ; all of which quickly supervene on the pyrexia, and are most pronounced in the first attack. At this time there is granular turgescence of the gland-cells, probably general, and the interlobular connective tissue is only seldom implicated (viz. 3 times in 16 special scrutinies) ; fatty transformation of the cell-substance is yet scarce. With cessation of pyrexia the turgid condition subsides, or it passes into the state of fatty metamorphosis, which I have found to be commonly present in casualties at post-invasion periods, whether or not accompanied by cloudy swelling of the cells. Recurrence of pyrexia entails repetition of enlargement (though to less amount), and gland-cell alterations. Finally the organ tends to resume its normal dimensions, but traces of cell-lesion may long persist.

In 21 examples the relation of changes to stage of disease, was as follows—Invasion-period, great alterations, granular or hyperplastic 3 ; less pronounced, granular and fatty 6, hardly perceptible 2, total instances 11. First interval, some degree of pigmentary, granular or fatty change 2, little or none 1, total 3. First relapse, fatty degeneration 2. Second interval some fatty change 1, or none 1. Sequelar stage, much fatty degeneration 1, pallor of cells 1. Pyrexia of uncertain date, much fatty change 1. As a rule, the least apparent alteration occurred either before fever has lasted 3 or 4 days, or 10 days or more after it has ceased ; but variations arise from both severity and complication of the spirilla pyrexia, and uncertainty of patients' reckoning.

Although my enquiries have shown that the liver suffers in Remittent fever somewhat similarly, and that there may be nothing absolutely

peculiar here to the spirillum disease ; yet for future reference, particulars are subjoined.

Invasion attack.—An infant of 7 months was infected by its mother in hospital and died on 7th day. The liver weighed 8 ozs., was of deep maroon colour and firm in consistence : the hepatic cells contained coarse granules, and very little oil.

A destitute woman æt. 29, died on reputed 4th day (cerebral hæmorrhage). The liver was large, but seemingly normal ; hepatic cells very distinct in all parts, and fat not abundant.

A lad æt. 22, died at the acme, jaundice slight. Liver 52 ozs., congested, firm, of uniform palish tint, surface smooth, sectional aspect unchanged, bile in gall-bladder of pale yellow hue : the hepatic cells seemed small and angular, they were filled with dark granular matter, and showed bright yellow particles (bile), no free oil ; some cells were shrunken and some empty ; on addition of acetic acid, there was a general clearing up, all the cells displayed a few minute oil globules, their nuclei distinct ; hyperplasia of connective tissue not noticed. These appearances are unusual in a liver so much enlarged ; possibly some concomitant change was overlooked.

CASE XVI.

F., 30, died at the acme, jaundice marked. Liver 62½ ozs., rounded, mottled, firm, pallid on section, granular when torn : hepatic cells affected with granular and slight fatty changes, no free leucocytes. The gall-bladder and ducts contained dark-green bile, no obstruction. CASE XIII.

F., 35, was seized in hospital and died on 4th day. Liver 44 ozs., and reported as simply congested : on closer examination I found pale patches disseminated throughout, and in these alone the signs of incipient fatty transformation. This example is instructive. The partial and seemingly irregular distribution of fatty changes, of considerable degree, was well shown in the case of another woman ; some of the cells contained also clumps of biliary matter ; the organ was large and of softish consistence.

Hyperplasia of connective and no cirrhotic aspect.—M., 25., *typhus biliosus*, death on reputed 6th day. Liver 73 ozs., pallid, with paler patches on the surface barely visible, edges not thickened, surface smooth and shining ; sectional surface uniform, lobules indistinct, some hepatic congestion ; a little viscid dark-green bile in the gall-bladder, no occlusion of ducts. Hepatic cells filled with albuminous granules, of brown tint, little oil or pigment, nuclei unchanged ; the slight fatty change not limited to exterior of lobules but disseminated in patches. Connective tissue in some portal canals decidedly overgrown, and this change doubtless contributed to the great enlargement of the organ : blood-vessels and ducts not materially altered.

Cirrhotic aspect and no hyperplasia.—M., 30, death at close from pyrexia and exhaustion. Liver enormous (74 ozs.) congested, of deep orange-brown tint, nodulated on the surface uniformly ; everywhere the lobules are collected in masses, varying in size from a pea to a walnut, deep-tinted and the sub-lobular (hepatic) veins repleted : the interlobular connective tissue appears scanty, loose, and of dark-grey colour ; peritoneal coat not opaque ; general consistence of liver soft and flabby. Autopsy 8 hours after death. The connective tissue has a coarse and indistinctly striated aspect ; on the addition of acetic acid very few cell-nuclei are seen, either oval or round, and there is no fat ; no cell-accumulation is visible in the coats of the vessels, or any gelatinous thickening ; the connective tissue may be attenuated, and the great enlargement appears due to vascular turgescence and distension of the gland-cells themselves. These are filled with fine granules and bright pigment particles, nuclei distinct ; oil globules detected in a few only of the cells.

Pseudo-cirrhosis and no hyperplasia.—M., 34, died at close of pyrexia, probably the first attack, of gastric hæmorrhage. Liver (after the hæmorrhage) hardly enlarged, 41 ozs., pale-yellow, very firm, tough, slightly nodular on surface ; on section a granular aspect, as of isolated groups of lobules, uniform ; left lobe very small, no opacity of serous coat, or adhesions. At first sight, this looks like an early cirrhotic change, but the impression is not confirmed, for the interlobular connective tissue is loose and not increased in amount. Much dark-brown bile in the gall-bladder. The lobules seem compressed from distension ; there is very much fat ; hepatic cells are of bright yellow tint, highly granular, and their nuclei invisible, they are closely compacted and their outlines are indistinct. On the addition of acetic acid the

amount of oil globules is seen to be large, the cell-nuclei appear; there is no sign of cell proliferation, and the walls of the vessels are seemingly unchanged.

These three last instances, with the following one, suffice to prove that a true cirrhotic change was not witnessed at Bombay as the result of the spirillar infection; the appearances are certainly peculiar, and, I think, much elucidated by the last example above quoted.

Of 3 deaths during first Interval, one or perhaps two are connected with the series just discussed.

M., 40, died on second day after crisis with pneumonia. Liver 52 ozs., surface pale yellow, mottled with red, and somewhat nodulated at the thin edges: on section, an appearance of yellowish distension of the lobules, with limiting vascularity and softness of their investing connective tissue: consistence flabby; autopsy 4 hours after death. The hepatic cells are filled with fine albuminous and pigment granules, the nuclei clear and very little oil visible; connective tissue seemingly unchanged, and no free leucocytes.

M., 30, died on 7th day with jaundice and diarrhoea. Liver 57 ozs., pale, soft, rounded edges: gall-bladder empty. The hepatic cells seemed small, many were converted into bags of oil, and in others was an accumulation of dark pigment: there were also free cells resembling leucocytes, with fibrous appendages, the appearance being that of connective-tissue overgrowth around.

M., 10., death on 10th day with pneumonia. Liver 23 ozs., noted as healthy-looking; but on close inspection, faint pale patches were detected throughout the organ, being disposed in clumps and streaks, made visible with a lens and seemingly encroaching on the exterior of the lobules themselves. In these pale spots the gland-cells had undergone an extreme degree of fatty transformation: small-cell infiltration was not seen. This instance illustrates a late liver-change.

In 2 deaths during first relapse, both of women, the liver once weighed 47 ozs., and was rather soft, there being diffused fatty change; moderate in degree. Once the weight was 54 ozs., aspect pallid and consistence firm: amidst the gland-cells of normal appearance, there were scattered many others which had undergone an extreme fatty change. The notes of another case, M., 46, show the liver to have been very large and flabby, bile-ducts gorged, lobules indistinct, congestion portal: there was an appearance of glandular atrophy and no sign of interstitial growth.

In 2 instances of death at second Interval (4th and 9th days respectively) the evidence of fatty transformation was less than above. I add another example—

M., 30, dying, exhausted on 9th day. Liver rather large, mottled with pale patches which extend into the substance of the organ; otherwise normal in aspect. To my surprise the cells in these pale patches had an unchanged aspect, clearing on the addition of acetic acid and showing no fat globules. This instance proves the converse of others, namely, that the appearance of fatty degeneration may be illusory, at late stages of fever; here the gland-cells seemed to be recovering (not reproduced), wanting only some of their coloured bile-constituents. Such recovery may not ensue, as a longer deferred instance shows.

M., 60, enfeebled, gradually sank two months after relapse. Liver 46 ozs., pale, firm, dry; there was hepatic congestion. Extreme fatty degeneration of the cells throughout; a condition not indicated to the unaided eye.

The connection of liver-changes with varying aspect of the blood as regards both spirillum and new cell-forms, did not strike me as being apparent; and, so far as I could judge, it was not peculiar. Morbid hepatic lesions in this disease, seem attributable chiefly to pyrogenetic

influences similar to those existing in some other specific fevers ; and if a special part be allotted to the visible blood-additions, it should, I think, be limited to explaining the remarkably defined extent of frequent degenerative lesions ; *e.g.* the disseminated pale patches of fatty changes, which in character are comparable to so-called 'infarctions' of the spleen. The liver-infarcts may be secondary to the splenic.

The Spleen.—That owing to malarious influence, this organ is habitually enlarged in a large proportion of the grown-up population of India, has been generally admitted ; yet within the range of my experience in W. India, such a state of permanent hypertrophy is by no means universal, or even common. Hence I am disposed to place considerable reliance in the data furnished below, as evidence of spleen-changes during the spirillum disease ; and, in fact, these data seem to correspond fairly with those elicited in Europe, where malarious influence does not much prevail.

Taking weight as a gauge of volume, the range in healthy adult Natives of W. India (Bombay and the Deccan in particular), was estimated at 5–12 ozs., or from $\frac{1}{250}$ to $\frac{1}{350}$ of body-weight ; the lower figure being so much nearer the average, that it may be said the prevailing normal weight of the spleen is about 5–7 ozs., or near the mean for European residents, whose bulk, however, is $\frac{1}{4}$ or $\frac{1}{3}$ greater, and in whom the larger weights are practically wanting. Variations according to age are doubtless similar everywhere.¹

Spleens of quasi-normal aspect.—Four of the 6 pertain to apyretic intervals, and are simply illustrations of the rapidity with which the spleen may resume much of its ordinary state after fever ; the other 2 cases were the following :—

M., 50, weak, while in hospital with chronic bronchitis caught fever and died in 5 days ; fever not high or sustained, spirilla in the blood to the end. Spleen 6 ozs. and comparable in aspect to a healthy kidney, its capsule smooth, pulp rather softened (report of officer in charge) : liver 42 ozs., pale and soft : autopsy made 12 hours after death. This case seems to show that until midway, at least, of a first febrile attack, the spleen may not be very sensibly enlarged.

M., 22, dying at the critical fall. Spleen reported as unusually small (2½ ozs.) : liver not enlarged (42 ozs.) ; autopsy 5 hours after death. As it stands, this example may indicate the immediate subsidence of turgescence on cessation of pyrexia.

Another instance of little enlargement occurred in a lad of 7, dying of high fever on 7th day. Spleen 2½ ozs., or $\frac{1}{4}$ of liver-weight and not more than weight of a kidney in same subject. As a contrast, an infant of 7 months dying at a similar epoch, had a spleen of 2½ ozs., or nearly $\frac{1}{3}$ of liver-weight and more than both kidneys together.

Enlargement.—Was noted alone in 64 per cent. of the autopsies, or combined with other changes, in 76 p. c. Absolute increase varies with stage of fever, being greatest during fever and tending to subside after crisis : see the next Sub-section. Alterations concurring in a total of

¹ Useful general data for Bengal were supplied by Dr. S. C. Mackenzie, in the 'Indian Medical Gazette,' July 1878 : and some valuable statistics of body-changes in the famine-stricken by Dr. A. Porter in a Report on the Sanitary and Medical Aspects of the Famine in Madras 1876–7, issued under the superintendence of the Sanitary Commissioner, Mr. W. R. Cornish, C.I.E. At the Bombay hospitals it was seldom that the diminishing influence of starvation had to be allowed for, but the point was borne in mind ; and it is not unlikely the small dimensions of some infarcted spleens, were due to mal-nutrition of the patient.

55 examples were congestion 6 (chiefly during first attack) infarcts 7, softening 20; commonly the organ was turgid and dark-coloured, its edges rounded, capsule thin, tense and smooth, not easily torn off, and free from external adhesions. Incidental to conjoined old malarious disease were limited opacity and thickening of the capsule, uniform firm consistence, and generally prominence of the Malpighian bodies. Post-mortem softening being very likely to occur early in a tropical climate, not much stress can be laid upon this conjunction; yet a decided diminution of consistence was noted in 7 of 12 autopsies made under two hours after death. Enlarged spleens sometimes displayed pale specks and streaks scattered throughout the pulp; the cut surface was often dry, and generally the dark blood-tint quickly became bright on exposure to air.

Softening.—This change in some degree is probably constant, when considerable it was usually recorded without comment: the mean weight of 8 specimens was 9 ozs., and the mean time after death of necropsy 6 hours. It seems unquestionable that the consistence of the spleen is sometimes greatly reduced during life, at least during the last period prior to decease. The softened organ is easily torn and its pulp may be almost pultaceous; there is usually much congestion, possibly some extravasation; the capsule was thinned. Though oftenest seen at close of invasion-attack, yet extreme softening was proportionately commoner at the succeeding interval. Even when not due to incipient decomposition, it may not always be of the same character.

F., 44, died in a drowsy state at reputed 6th day of first attack; blood loaded with spirilla. Spleen 8 ozs., its pulp said to be seemingly disorganised: liver 40 ozs., congested; there was acute fatty degeneration of the kidneys. Body much emaciated: autopsy 4 hours after death.

Rupture of the spleen was never detected at Bombay.

Splenitis and abscess were not seen as simple results of fever. In connection with infarcts, limited and scant perisplenitis was once noted (see below); and there are notes of a small softened infarct occurring in a child suffering from a recurrence of fever—nature unknown of first attack, with abscess in the shoulder: spirillar infection may have preceded, but the great rareness of these acuter sequelar phenomena at Bombay, is in contrast with recorded experience at St. Petersburg and some other European cities. It occurred to me that possibly malarious induration of the spleen might render the organ indisposed to such changes. Metastatic abscesses and thrombosis of the splenic vein were not witnessed here.

Infarcts.—These I find to be altered portions of the spleen-pulp, and as such they are usually comparable to the pale patches seen in the liver. Minor and rarer changes of the Malpighian bodies of the spleen are illustrated further on.

The 14 instances alluded to in the Summary (equal to 20 p. c. of all autopsies), were of the more pronounced degree; and if obscurer or incipient examples be included, the number of infarcted spleens would be nearly doubled.

As contrasted with the parenchyma generally, these altered portions are, at first, somewhat swollen, of lighter reddish or yellowish semi-translucent hue and firmer in consistence; their dimensions vary much,

and so their number and form. In position they may be superficially placed, or deep-seated. At a later stage, their tint becomes lighter and more opaque, the margins more defined and consistence softer, especially in the centre. Appearances differ according to site; thus, when deep-seated the spots are apt to be less defined, of irregular shape and redder hue; whilst the superficially placed assume a pyramidal form (base outward), and are paler and sharply limited. I have found infarcts at the periphery alone, whence it is likely they are earliest formed there; they were commonest at the edges of the organ.

The detection of this pulp-change sometimes requires care, and upon several occasions no particular alteration in the cut surface of the spleen was noticed at once; but after a few minutes' exposure to the air, a manifest difference appeared, and shortly the outlines of the changed parts became clearly visible. This is a point of some practical importance, and when desirable, I have had bodies re-opened for closer search, finding the spleen thus changed although apparently not so at first examination: hence a rule always to examine this organ a second time, if doubt existed as to the presence of infarcts. Slight dessication and the action of atmospheric oxygen, renders distinction of normal and changed pulp more apparent; the altered portion becoming lighter and shrinking less: this difference may be seen even through the capsule of the spleen.

Enlarged Malpighian bodies and delicate white streaks in the pulp may co-exist with infarcts; and occasionally there were seen small patches intermediate in character. Apparent hæmorrhagic spots may co-exist, and when the changed area is well-defined, it may be surrounded by a zone of dark vascular shade; this is oftenest seen around superficially placed infarcts. In them, too, the splenic capsule may be slightly thickened, and the serous coat at this spot dulled; there being a distinct raising of the surface. At a later stage, there may be slight depression of the changed area.

Infarcted spleens were commonly enlarged, yet not quite invariably so in equal proportion; they were usually firm, but sometimes of diminished consistence, and this independently of post-mortem softening.

In 53 deaths occurring during febrile periods, manifest splenic degeneration of this kind was noted 11 times; and amongst 21 deaths at apyretic intervals 3 times: when appears its connection with both specific pyrexia and decease. The mean age of these 14 patients was 26 years, extremes 18 and 46.

Other particulars are furnished in the following summaries of cases.

M., 18, feverish and delirious, died a few hours after admission at reputed 5th day of invasion: the chief lesions were found in the blood (which was dark and coagulated slowly, contained many large white corpuscles, protoplasmic masses and active spirilla) and in the spleen. This organ was large (9½ ozs.) dark-hued, firm; capsule smooth, thin, translucent and inadherent. On the outer convex surface an ovoid, pale-red patch, an inch across, surrounded by a darker purple zone and, on section, traceable for an inch and a quarter into the pulp, where it assumed a conical form, the base corresponding to the ovoid outline on the surface. Spleen-substance—the whole pulp was infiltrated with smaller pale-reddish masses of softish consistence, the lesser of which might be compared with enlarged Malpighian bodies, whilst others were like coarse granules or nodules. Speedily on exposure to air, other infarcts became visible along the edges and fissures of the organ being distinguished

by their opaque reddish tint: the capsule over these places was somewhat thickened. The liver was large and marked with pale patches on its convex surface.

M., 46, admitted with his brother at close of invasion, underwent a typical first relapse and died just after its termination, pneumonia being apparently imminent. Of all organs the spleen was most changed in aspect, weight 20 ozs., colour dark, consistence soft and lacerable, hue on section purplish: there were several pale, raised patches on the convex surface and borders, which were due to soft, pale-red infarcts, others being present in the interior of the organ: diameters half an inch to an inch, form cuboid or elongated. Liver large, yellow, flabby.

M., 35, admitted in a dying state, possibly at end of relapse; the blood charged with spirilla. After death the signs of copious gastric hæmorrhage. Spleen 24 ozs., livid, firm, edges rounded, capsular nodules (old), no adhesions; at first no light patches visible on the surface, but upon partial dessication they make their appearance and it becomes evident, on section, that the spleen pulp is not of uniform structure, there being large, palish-red areas disseminated throughout, oftenest, perhaps, near the centre or hilus; these paler spots contrast slightly with the darker bluish pulp around, yet cannot be always traced to the surface, where also red patches are indicated; no real discrepancy obtains here for the outlines are too indistinct to map out fully. These appearances would be explained by diffuse capillary hæmorrhage (giving the dark hue to pulp), or by wide fatty degeneration (causing the paler aspect); since the more prominent surfaces on dessication correspond to the darker tinted pulp (the paler collapsing somewhat), there has been probably some interstitial hæmorrhage yet no loose clots, of even small dimensions, can be picked out, and this idea therefore remains unverified. Liver pale, firm, not enlarged. Autopsy after 4 hours.

With reference to parenchymatous extravasations, there are some other notes, one of which belongs to a case of cerebral and pericardial hæmorrhage, the statement reading that together with a large number of pale red infarcts (the more superficial having a deep yellow colour and being well defined) there were hæmorrhagic patches distributed throughout the splenic pulp. I also remarked that the larger pale spots in the spleen ($1-1\frac{1}{2}$ in. deep) were softening in the centre. Patient a male of 30, dying at close of first relapse. CASE LXXVII.

Concurrence and likely connection between enlargement of the splenic Malpighian bodies and incipient infarction, was indicated in the following cases; see also below.

F., 35, died towards close of first relapse. Spleen, double the usual size, livid, edges rounded, some pale patches on the surface; pulp firm, deep purple, and studded with small white dots—Malpighian bodies; these are as large as a pin's head and very prominent; besides, corresponding to the pale patches seen under the capsule, is a similar pallid infiltration of the pulp reaching to the depth of $1-1\frac{1}{2}$ inches. The patient was lately parturient, and infected in hospital.

M., 14, died at invasion. Spleen 11 ozs., firm, dark, friable; capsule thin and showing through it a dotted aspect due to subjacent spots, probably large Malpighian bodies: there was also slight indications of pale patches, comparable to those existing in the liver. Spirilla in the blood of the corpse.

After death from malarious fever these bodies are often distinct; and upon direct comparison, I once noted the malarious spleen (much enlarged) to differ from the spirillar, in being of firmer consistence, of greyer tint, uniform throughout, Malpighian bodies very clear and the fibrous capsule more readily torn off.

The following is the only instance seen of perisplenitis.

M., 20, died on 4th day of second Interval. Spleen $19\frac{1}{2}$ ozs., there is recent lymph on the dark coloured surface, at the edges of a fissure; there are visible also light patches which correspond to pale reddish masses occupying the pulp beneath

and rather firmer than it: the splenic parenchyma is of less dark hue than the surface, and rather soft. The infarcts are most distinct at the anterior border of the spleen, where probably the circulation is most languid. Liver large, smooth, pale: pneumonia was present.

Morbid histology of the Spleen.—My notes refer to the cell-constituents of the pulp; fibrous structures and the blood-vessels when examined, furnishing little sign of departure from a quasi-normal state. 14 unselected specimens passed under notice, 8 of which pertained to the Invasion-attack, 3 to the first Interval, 2 to the first Relapse and 1 to an uncertain febrile date.

The spleen of an aborted foetus was small, not congested and like a bit of kidney; nothing peculiar was detected about it; spirillum not found in the blood.

Invasion-period.—In a male infant and a lad of 7, the spleen weighed equally $2\frac{1}{2}$ ozs., appeared healthy and showed no abnormal elements on minute scrutiny: in a man of 24 the spleen was actually and relatively smaller than usual (9 ozs.; liver 61 ozs.), and the pulp was filled with cells which seemed to be simply multiplied. These 3 cases are evidence that the spleen may seem unchanged as regards its structural elements, in fatal cases of spirillum fever; and the last one, at least, shows that this may be so when other organs are characteristically altered. The remaining 5 cases furnish testimony of a change in the spleen-pulp, comparable to that simultaneously occurring in parenchyma of liver and kidney: this demonstration was a novel one to me.

F., 35, died on 4th day (contagion in hospital). Spleen 7 ozs., dark, soft and friable: interspersed amongst the normal cell-elements were some oil globules, and + acetic acid a large number of fat globules appears, varying in size from the smallest imaginable to drops as large as a red disc; they arise in the spleen-cells; Malpighian bodies not affected. Date September 1877, and specimen marked "Acute fatty degeneration of spleen:" the same change was present in the liver and kidneys, which also were little changed in general aspect.

M., 35, d. 8 day. Spleen 16 ozs., friable; amongst the trabeculae and pulp are parts which have undergone as much fatty change as seen in the kidneys, possibly on site of extravasated blood, yet the cells (nuclei and substance) are also implicated; there are parts of the pulp less changed, the blood-vessels and trabeculae being unaltered.

F., 30, d. same date. Spleen, 18 ozs., soft; acute fatty degeneration of pulp-cells is very distinct in places, their nuclei are invisible.

M., 20, d. near end. Spleen $38\frac{1}{2}$ ozs. dark, firm but friable, and having many small infarcts dispersed throughout the pulp: there are here numerous cells loaded with fat and some very large masses of fat granules, besides fine, free oily matter. Some large granule-cells were seen in the right cavities of the heart; they may evidently come from the spleen. The blood was very thin and of pale brown hue; it contained many spirilla.

M., 25, d. near end. Spleen 36 ozs., turgid, rather soft ($1\frac{1}{2}$ hrs. after death) and containing many faint pale-red patches. Micr. ex.—Normal darker part of pulp. It is tougher and more flaccid; fibrous structures distinct, pulp cells less crowded and their fatty aspect less pronounced; some fatty cells are still seen, + acetic, and large endothelial cells; but their number is comparatively small, and degeneration slight. Pale patch in interior—many large cells with nuclei like those found in the blood; ordinary leucocytes not so abundant, and there are transitions from these to the larger granule-cells, which hence may be hypertrophied and degenerated pulp-cells. + Acetic acid, the field mostly clears, the small cells swelling into a compact mass; but there are places where extreme fatty degeneration is indicated (nuclei still visible); also fatty endothelium and occasionally large oval masses of oil are seen amidst unaffected tissue. The evidence of a fatty change, whether or not preceded by cloudy swelling,

in this infarcted area is decided ; the material here is paler, less translucent, turgid and friable, and the histological resemblance to pale patches in the liver is sufficiently close: infarcts, therefore, are areas of degeneration.

Of the 3 instances dying in first interval, 1 detailed below shows a condition essentially belonging to the preceding pyrexial period ; 1 is the case of a man dying of liver-abscess just after the apparent termination of invasion-attack (contagion in hospital), the spleen weighed 16 ozs., and my notes state that large granular, nucleated cells were seen in the pulp, such as are often found in the blood at critical fall. The last case is that of a girl dying of dysentery, eight days after a specific attack of uncertain date ; spleen-weight $1\frac{1}{2}$ ozs., and no peculiar appearances detected.

M., 35, caught the fever in hospital and died in a sharp rebound ensuing immediately on the crisis. Spleen 19 ozs., turgid, dark and firm, no infarcts on first section, but afterwards indicated after exposure to air. Mic. ex. of a pale part preserved in alcohol: dense collections of small cells, most seem normal, others are larger and of granular aspect ; + acetic ; most clear up, but the large granule-cells remain for a time, finally clearing and not showing nuclei ; decided fatty degeneration was not apparent, and it would therefore seem that the first stage of infarction resembles the cloudy swelling of hepatic and renal cells, to which fatty transformation succeeds in the spleen, as elsewhere.

The specimens from death in first Relapse (2) and at undetermined date (cerebral hæmorrhage) (1), seemed to belong respectively to the series of no minute change of spleen-elements, of cloudy swelling or granular degeneration, and of incipient fatty degeneration of pulp-cells: in the first case numerous chains of vibrating particles were seen, resembling some visible in the blood taken before death.

Upon review of this detailed series, I find that affected spleens are commonly of large size, and that the liver at the same time is also large ; both organs in 6 examples being simultaneously implicated : in 2 others, however, the liver alone was chiefly affected, the spleen in one being rather large but not diseased, and in the other still less enlarged yet decidedly diseased. This last case proves that in death from spirillum fever so early as the fourth day, the spleen (as other organs) may undergo prompt fatty degeneration with very little other visible change. Simple febrile enlargement of the spleen is due mainly to multiplication of the pulp-cells, vascular turgescence also contributing part. It is remarkable how rarely dark pigmentary degeneration of the spleen-cells, was noted at Bombay.

The Kidnies.—Lesions of the renal secreting structure may be non-apparent, and in part obscured or simulated by incidental changes ; hence ordinary data have but a limited value. Another drawback arises from the undetermined amount of pre-existing kidney disease, likely to be found in the less temperate Native races ; this source of fallacy, however infrequent by comparison with European experience, has still to be borne in mind.

The normal mean weight of each kidney in adults, is estimated at $3\frac{1}{2}$ to 4 ozs.

Quasi-normal aspect.—Under this indefinite term, some examples of minor organic change were doubtless included ; yet here is evidence

of but little visible alteration at stages, both febrile and non-febrile, during which these 10 instances equally occurred.

Congestion: 26 examples.—This as a prominent character, was seen in deaths during pyrexia; occasionally the organs were also enlarged, and sometimes in parts pallid; attendant hæmorrhage was very rare. The degree of persistent congestion seldom amounted to active hyperæmia, such as is supposed to precede acute nephritis; though a certain degree of vascular turgescence no doubt goes before and attends, the glandular degeneration described below. The weight of the kidneys was only slightly above mean, and considering the intensity of spirillar pyrexia, it is noteworthy how little hyperæmic these organs have appeared; specific fever is, however, of brief duration, and epithelial alterations may be quickly induced.

Congestion is not said to be limited to either cortical or medullary substance: sometimes it was mainly hypostatic.

Interstitial hæmorrhage was very rare.

Acute parenchymatous nephritis was never met with, nor the diffused or disseminated suppuration (metastatic and local) mentioned as attending severer cases of recurrent typhus in Europe.

Pale and enlarged kidney: 33 examples inclusive (nearly 45 p. c. of all autopsies).—In frequency this change answers to the concurrent alterations in liver and spleen: it was found almost exclusively in deaths at the febrile acme and fall, and early ensuing apyrexia, being rare before fever and congestion had existed for a certain time. The maximum weight of a kidney (the right being somewhat oftener the largest) was $6\frac{1}{2}$ ozs., seen at both acme and fall; the lightest specimen weighed 4 ozs. From my tables it appears that the increase of volume after invasion-attack is so marked, that the kidneys weigh more during the first apyretic interval than they do during the relapse, or even during the earlier days of invasion-attack: this fact is worthy of recollection.

Consistence seldom so firm as natural, friable and flabby.

The colour is described as pallid or pale yellow, distributed either uniformly or in patches over the surface of the kidneys; stellate vascularity increasing the mottled aspect was not unusual. Upon section, the cortex and medulla are not distinct; the secreting portion is enlarged, and sometimes of coarse aspect from the presence of vascular streaks and dots; the pyramids compressed, striated and congested at their bases. The aspect of partially localised pale infiltration was not uncommon, and sometimes marked; but distinct renal infarcts were never seen.

The mucous membrane of the pelvis is congested, and occasionally the site of small hæmorrhages.

Capsule.—Thin and translucent, as a rule, commonly non-adherent, and sometimes even readily separated: at other times without thickening, vascularity, or much opacity, the capsule was adherent, wholly or in part, and could not be removed without laceration of the glandular superficies. If not pertaining to pre-existing disease (which was never proven), this last condition would indicate irritation attendant upon degenerative changes, answering to more occasional thickenings of the serous envelope of liver and spleen. The cellular coat of the kidney is

the seat of small extravasations, corresponding to the commoner sub-serous petechiæ.

Illustrations accompany the histological memoranda.

Instances of pre-existing kidney change are the following :—

M., 40, died at the (first?) critical fall. Kidnies 6 and $5\frac{1}{2}$ ozs., much congested, flabby, capsules very thin and when separated bringing away renal tissue : substance coarse, studded with cysts. The left kidney was most affected, and showed signs of interstitial fibrosis, unquestionably of older date than this febrile attack ; a similar change was beginning on the right side.

In a girl of 8, dying of femoral thrombosis just after the first relapse (her old father died about the same time of cerebral hæmorrhage), the kidneys were pale and firm, capsule rather adherent ; some stellate mottling of the surface, but no disease apparent. There was a double ureter on right side, in connection with a narrow elongated organ. Urine not albuminous.

Histological Notes.—I examined 26 specimens taken at different stages of the fever. The only lesion usually found is strictly comparable to that commonest in liver and spleen, consisting of a granular tumefaction of the parenchymatous or secreting cells, which is apt to end in fatty transformation of their contents and probable disintegration of structure. Not only do the renal cells exhibit this cloudy swelling with or without ensuing fatty degeneration, but they are also at first multiplied ; their nuclei becoming more numerous, like those of the hepatic cells. These changes sufficiently account for the pallor and enlargement of the kidneys above described ; in some degree and in some part of the cortical substance, they are probably invariable. According to my experience, the fatty transformation is somewhat less frequent in renal than in hepatic and splenic lesions ; cloudy swelling is at least as marked, being also more readily recognised in the broad uriniferous tubes. As regards the liver, quasi-physiological conditions may simulate incipient morbid changes ; the spleen is liable to prior malarious hypertrophy ; and so the kidneys may have been damaged by previous nephritic disease, likely to entail lesions similar to the above. Hence the need of careful discrimination, when estimating the character of post-mortem appearances.

A distinguishing feature of the fever-lesion at Bombay, was the general absence of concurrent interstitial cell-infiltration or fibroid thickening ; very few instances were noted of any change of the inter-tubular tissue and vessels, and this fact shows the rarity of comprehensive nephritic lesion in fatal spirillum fever at Bombay. Corresponding in such negative feature, was the seldom-changed aspect of the urine : see Chap. III. In Russian and German towns, renal implication is said to be frequent and pronounced ; hence a difference open to varied interpretation.

Lesion according to Stages of Fever.—Invasion-attack : 15 specimens. Granular turgescence of the renal cells, was in some amount always present ; and in $\frac{2}{3}$ of cases, fatty changes were also more or less clearly indicated.

Examples.—M., 30, died of cerebral hæmorrhage with specific infection. Kidnies large, congested, substance flabby and yellowish, capsule thin and somewhat adherent. The renal epithelium was coarse-looking, distending the tubes in many places, but the amount of oil present very scanty : Malpighian tufts rather large and wax-like, no fatty degeneration here. F., 35, died on 4th day, exhausted ; kidneys, like the other

organs, comparatively unchanged in aspect. Some congestion of inter-tubular veins, Malpighian tufts not distended, epithelium of tubes very coarse-looking and often much accumulated, no free oil: + acetic acid, the renal oils were wholly free from fat; many small, elongated collections of minute oil-globules were seen between the tubes, in their walls or in the stroma itself; Malpighian tufts not diseased. This appearance was not again seen outside the uriniferous tubes.

F., 30, death from fever. Kidnies large, capsule not adherent but congested and containing some petechiæ; substance congested and flabby, surface mottled with yellowish patches: on section, the cortical portion broad, of deep yellow tint, dotted and coarse-looking, petechiæ in the pelvis. The uriniferous tubes were filled with granular cells, in which only traces of oil were present, the capillaries of Malpighian tufts unchanged, inter-tubular tissue unaltered: an occasional red disc was seen within the tubes. No fatty degeneration of other organs. A similar instance occurred about the same date, in which a distinctly pallid, friable and tumid part of the kidney proved to be wholly free from fatty changes: these examples showed how little dependence can be placed on mere appearances.

Kidney at crisis.—M., 32, famine-immigrant. Organs large, pale, mottled, firm, capsule not adherent; the most striking change is pallor, some intertubular congestion is noticeable in places. Renal epithelium very coarse and partly detached (autopsy 16½ hrs. after death), its granular aspect uniform and not as if due to oily particles, Malpighian tufts unchanged, blood-vessels in places distended: on addition of acetic acid, sometimes not a trace of oil, the cells clearing wholly, nuclei distinct and granular; sometimes there are distinct granules and globules of oil to be seen in the tubes, but this is not common, and only a few of the tubes displayed incipient fatty transformation: the greater number being rendered opaque and much distended with dark-brown, granular epithelium or masses of granules: a few oil globules were to be seen in the capillaries of some Malpighian tufts.

Analysis of four examples belonging to the severest form of spirillum fever (*typhus biliosus*, death at acme of first attack), showed the kidneys to be larger than usual, pallid or mottled with blood-vessels, and flabby; the cortical part pale, soft and seemingly fatty: the fibrous capsule thin, adherent twice over limited areas, and non-adherent twice; petechiæ in both groups. The histological changes in the cortex were as follows:

Uriniferous tubes distended in parts, with hypertrophied epithelium of finely-granular, thickly clouded and brownish aspect, nucleus not visible; twice the cells were so swollen and compressed as to have lost their contour, and become blended into an opaque granular mass, apt to become detached from the walls of the tubes. The appearances might be confined not only to certain *tubuli uriniferi* but even to different lengths of the same tube; and this feature is noteworthy, when taken in conjunction with a similar localised distribution of liver and spleen cell-changes. By such lateral expansion (not, so far as I saw, limited to flexuosities of the tubes) the adjoining blood-vessels become compressed, and hence (as in the liver) the comparatively bloodless aspect which conjoins with the tint of the cloudy swelling to impart a general pallor to affected parts. On the addition of acetic acid, the clouded aspect disappeared wholly, cell-nuclei becoming apparent, except when the epithelium was converted into a uniform granular mass, in which alone globules of fat abounded. The Malpighian tufts were, at most, congested; and the inter-tubular tissue not altered, except when the fibrous capsule of the organ was adherent. The last of the cases showed this. M., 25, dying soon after admission: kidneys large, pale (uniformly), flabby, capsule thin and adherent at parts, a few petechiæ in its substance. Mic. ex.—Cloudiness of the tubular epithelium, sometimes very marked, sometimes contents of the tubes have become detached: + acetic all clears up, and there is no fatty degeneration: Malp. tufts clear and entire; in some parts there is slight cell-proliferation of the inter-tubular connective, but no thickening of the matrix: the tubes vary in diameter.

Two other instances of more pronounced kidney-disease merit

special notice ; both patients were admitted in a moribund condition, and the urine was not examined.

F., 44, autopsy after 4 hrs. Kidnies 5 ozs., firm, slightly granular and mottled, capsule thickened and in part adherent ; cortex engorged, dotted with blood and encroaching on the medulla. Blood loaded with quiescent spirilla. There is universal fatty degeneration of the renal epithelium, nuclei in walls of Malp. capsules loaded with fat ; hæmorrhagic spots amidst the tubes, with much congestion (cortex). + *acetic*, degree of fatty change extreme : the congestion is chiefly venous, and there is effusion of blood in the tubes themselves ; vessels of Malp. tufts partly covered with fat globules : probably there is also a new formation of fibrous tissue, for some free nuclei are seen interspersed amidst the glandular tissue. Heart normal ; pleural adhesions, but peritoneum healthy ; no dropsy : duration of fever reported at 5 days only.

M., 30, d. 8th day. Kidnies 6½–7 ozs., congested, softened ; int. surface uniformly yellow, medullary pyramid indistinct, capsule firmly adherent. Renal epithelium coarse, dark and commonly in a state of fatty degeneration ; some of the tubes are of normal aspect, others much enlarged. Malp. tufts show fat globules in the walls of their capillaries ; + *acetic* this becomes more evident, and large drops of fat are seen within the cells and tubes, in certain parts. The liver and spleen furnished evidence of fatty change ; spirilla were common in the blood ; and my impression is that in this case, if not in the preceding one, the morbid changes found may have been due solely to specific infection.

First interval.—The dates of death ranged from 1, to 7, 9 and 32 days after the critical fall : pneumonia was always present : state of kidneys as follows :—Dimensions somewhat less, renal epithelium always granular and twice showing scanty, partial fatty changes. In the death just after the fall, the liver cells were granular and rather less fatty than the renal ; in the death on 7th day, the liver cells were very fatty in parts, and the renal less changed in this direction : in the two later cases the appearances were as if granular or albuminous degeneration were slowly subsiding (coarser granules, with less intumescence, being present) by absorption of the diffused intra-cellular material ; but the presence of pneumonic fever may be here concerned, since I find none of the renal changes under notice are peculiar to spirillum fever.

First relapse.—Both the deaths were of females (ages 20 and 35), one taking place on the 4th day (fall commencing and cerebral hæmorrhage) and the other on the 5th day, specific fever still being high (splenic infarcts). It is remarkable that in both cases, the kidneys seemed only congested ; and on minute examination I found the renal epithelium in the first hardly altered, and in the other there were but scanty signs of granular and fatty change (the latter being limited to the Malp. tufts) : hence it might be inferred that the kidneys do not suffer much, in even well-marked first relapses.

Second interval.—In a man of 20 dying with incipient pneumonia on 4th day (splenic infarcts), the kidneys weighed about 3½ ozs. and were pale : most of the renal tubes were distended with highly granular epithelium, fat very scanty ; small collections of broken-down red discs in some capillaries. In a man of 30 dying exhausted on 10th day, the kidneys were equally small, but not pallid : the renal epithelium looked coarse, but did not contain fat.

Sequel.—A man of 60 sank exhausted a fortnight after recurring attacks : kidneys 4–5 ozs., smooth, pale, tinged with bile, capsule slightly adherent in parts : the renal epithelium had undergone extreme fatty degeneration and the capillaries of the Malp. tufts were sometimes affected : crystals of leucine and tyrosine amidst the tubes. The liver and heart also were fatty.

Uncertain date.—A girl of 12 dying 10 days after cessation of fever (dysentery), had renal organs congested but otherwise normal : some of the uriniferous tubes were doubled in diameter by accumulation of granular epithelium ; the nuclei seemed multiplied : no fatty matter. A man of 35 died at crisis of fever (cerebral hæmorrhage) : kidneys 4 ozs. each, pallid, effusion of blood around apices of pyramids, capsule non-adherent : the renal epithelium had undergone an extreme degree of fatty change, and some arterial capillaries were affected ; there was venous inter-tubular hæmorrhage. In other cases of cerebral hæmorrhage neither kidneys nor heart were fatty, and subjects were young. Adhesion of the renal capsule in some degree, was noted only 6 times in these 26 autopsies.

Uterine System.—Only this portion of the generative apparatus was scrutinised.

F., 30, unmarried, death during invasion, with characteristic symptoms. Uterus healthy, mucous membrane pallid and no signs of hæmorrhage (there were petechiæ elsewhere).

F., 35, was delivered of a full-grown child about 10 days before fever came on ; she died towards the close of the relapse : no symptoms referrible to the womb. Uterus 4 ozs., a little large from defective involution ; the cavity seemed also large, but was empty and the lining membrane healthy : so the ovaries and Fallopian tubes. (Mr. Bh. A.)

In a woman of 20 who died during the relapse, the uterus was found to be healthy in aspect.

THE INTESTINAL CANAL.—No anatomical details are available respecting the mouth, fauces or pharynx ; nor are any histological notes yet prepared.

Stomach.—16 autopsies. Though not numerous, these were fairly representative, and they include two examples of black vomit. In the absence of lesion elsewhere, the mucous membrane alone of the stomach is alluded to. Palpable change of consistence was never seen (not even of the post-mortem variety) ; congestions were common, and seldom, I believe, wholly accidental ; hæmorrhages certainly were peculiar : inflammation and ulceration of the mucous membrane were rare and never pronounced. There being no sign of chronic or minuter lesion, the microscope was not applied to fresh specimens, and the preserved are as yet unexamined ; probably chronic stomachal lesions are rare in the comparatively abstemious Hindoo races. Briefly, in the vascular character of most post-mortem lesions there is an accordance here and in the remainder of the intestinal canal, with other organs of the body : the state of the blood itself being identical.

It is proper to add that stimulant drugs and alcohol had been administered prior to decease, in most of the cases here analysed.

Congestion : 10 instances.—Common in deaths during specific fever (8 in 11) ; but found in later febrile complications, and as a sequel. When associated with blood-contamination it was present about the mid-region and towards the pyloric end of the stomach, and had not an accidental aspect ; sometimes it might be termed active ; hæmorrhage was usual, and small erosions have been noticed in the same spots.

A connection with epigastric symptoms during life seemed always possible.

Hæmorrhage : 8 instances.—Also closely connected with the febrile state (7 in 11), and not noticed as a sequel. The extravasations were

always small (petechioid), often in clusters and when limited, situated along the lesser curvature or towards the pylorus: their site was apparently the sub-mucous connective; minute erosions were sometimes perceptible over them, and petechiæ were always present in other parts of the body. The instances of black vomit are not here included.

Pallor: 5 instances.—Seen during both fever and intermission, and this fact sufficiently shows the contingent character of vascular changes here and in other parts of the body, as complications of spirillar infection. However, during actual pyrexia it is likely the mucous membrane is injected; with the crisis it may become pallid, and it is probable that copious gastric hæmorrhage then takes place: see below.

Illustrative memoranda:—

M., 22, died on 7th day of invasion. Stomach distended, its mucous membrane congested to a pronounced pink hue: little intestinal congestion, liver and spleen congested. Autopsy after 3½ hrs.

F., 30, death at 8th day of invasion. The stomach distended; sub-serous petechiæ; mucous membrane highly congested, and numerous sub-mucous extravasations of small size along the lesser curvature; some viscid mucus streaked black with blood, coated the surface, and had been forced into the œsophagus; hence had this patient lived, a certain amount of 'black vomit' might have appeared as a symptom during life (other patients have exhibited such a sign). Similar black mucus in the intestines. Autopsy after 6 hours.

M., 34, died at *perturbatio critica* of invasion. There were localised congestions and small hæmorrhages in the stomach and intestines. A case of 'bilious typhus.'

M., 35, died immediately after the critical fall, a sharp rebound intervening. Mucous membrane of stomach rugous and villous, of uniform deep pink colour; no extravasation of blood here, a few petechiæ in the adjoining small intestine. Liver and spleen enlarged. (Mr. S. A.) Autopsy after 2 hours.

M., 55, died during first interval, of cerebral hæmorrhage and inflammation. Stomach distended with a green liquid, intensely congested and marked with bands of vivid punctate hæmorrhage along the small curvature and towards the pylorus. Congestion and petechiæ at lower end of small intestine. Liver large and pale; spleen small and dry.

M., 22, died in the first relapse, with pneumonia. Great vascularity of mid-region of the stomach, with hæmorrhagic spots. There had been much epigastric pain and vomiting, at the last.

M., 30, died at close of first relapse, with cerebral hæmorrhage. Stomach distended, several narrow streaks of hæmorrhagic patches running lengthwise; apparent erosion over some of the spots. Autopsy after 2 hours.

M., 60, cachectic, died 42 days after last attack of fever. Along with fatty degeneration of heart, kidney and liver, and a malarious spleen, the stomachal mucous membrane was pallid, with red patches and longitudinal streaks, over which were numerous shallow erosions. Sequelæ stage of fever.

Black vomit.¹—M., 34, died at first critical fall (t. 95°), autopsy 1 hour after

¹ There are notes of two other fatal cases of fever, one having a history of a relapse and the other a stranger; in both deep jaundice and the typhoid state: no vomiting in either. As the blood-spirillum was not seen, these instances are omitted from the text; yet their symptoms and general character undoubtedly indicate their spirillar nature. Gastric hæmorrhage in the remittents of Bombay, I never saw. M., 20, died during pyrexia (? secondary to relapse), the stomach (and intestines) contained a black, grumous liquid; and there were small, soft coagula adhering to the pale mucous membrane. M., 40, blackened mucus in the stomach, a group of dark spots on the posterior surface below the lesser curvature, at the pyloric end a large group of bright pink spots, and at the termination of the œsophagus longitudinal streaks (½—1½ in. long) of deep black extravasations, which at their upper end merged into bright red spots (no change in the pharynx), over these the mucous membrane seemed about to slough: the intervening tissue was healthy-looking; the lymphatic glands in connection large, dark and soft. No morbid

death. Stomach distended with more than 2 pints of a thin pitchy-black fluid; on its evacuation, muc. membr. seen to be thrown into large rugæ, surface pale all over, large veins not distended, and neither diffused congestion nor hæmorrhagic spots were more than indicated. Close scrutiny showed the entire absence of unusual vascularity or of any erosions of the surface; but rather decided pallor, and the rugous state resembled that of most stomachs under contraction. Liver large and softish; gall-bladder contained $2\frac{1}{2}$ ozs. of glairy, yellow-tinted bile, quite diverse to the pitchy fluid. Small intestines also filled with a similar dark liquid, and no vascularity here. There had been copious hæmatemesis a few hours before death. No infarcts in the spleen. See CASE LXXXII., page 213.

M., 34, died at decline of fever of uncertain duration: no vomiting before death. Stomach contained air and a pint of black, grumous fluid; glairy discoloured mucus lines the interior surface; muc. membr. seemingly intact, and nowhere congested: all coats of stomach pale, no thickening, and no dullness of serous covering. On scrutiny with a lens, I found the muc. membr. universally pale; yet after gently scraping the surface very minute red puncta might almost everywhere be seen, most numerous at larger end and beginning of lesser curvature; these had a stellated or blotchy aspect, and appeared to be placed beneath the unchanged epithelium. There were a few larger pink streaks, deep-seated. Upon pinching with forceps around these small dots, the specks became larger and deeper-tinted, and sometimes a minute quantity of blood exuded, slowly trickling away on the free surface, together with mucus also thus squeezed out of the stomach-follicles; the source of the gastric hæmorrhage during life being in this manner rendered obvious. Muc. membr. of small intestine was unchanged in aspect; contents of the gut some blackish mucous and fæces: liver not enlarged, very pale; much dark-brown bile in gall-bladder: spleen very large and infarcted. CASE LXXXIII.

The **Pancreas** was examined on a few occasions and found to be unaltered, excepting that it appeared to be enlarged and firmer than usual, and more nodulated.

Small Intestines.—Owing to circumstances, the autopsies are here fewer and briefly recorded. Notes concerning the ileum are the most numerous, because this part was more closely scrutinised and certainly oftener found diseased: when it alone is mentioned, it may be understood that the preceding portions of the small gut presented no obvious change, for the ileum was rarely examined alone. No selection of cases was made, and all stages of the fever are represented in this series. Remarks apply to the mucous membrane and its glands; other tissues being named when required. Autopsies 58 in the total of 74.

Duodenum.—Total inspections 33, pallor or no change in 21, congestion or petechiæ in 13. Staining with bile is not here included. The duodenal mucous membrane may, or may not, present the same general aspect as that of the adjoining stomach: congestion is the commonest local change, it may be limited to a few of the *valvule conniventes*; hæmorrhagic spots may co-exist, they are seldom found here alone: the lower end of the gut is most affected.

I may here mention the condition of the muc. membr. throughout in the two above-named instances of gastric hæmorrhage. In the case of hæmatemesis intestines contracted all but their middle part, and at intervals occupied by a pitchy fluid like that found in the stomach, but now thicker in consistence: it shows through the thinner ileac coats. Duodenum—some thickening, surface rugose and mammil-

appearance at the cardia, and no suspicion of irritant poisoning: no scurvy. Much altered blood in the small intestines; petechiæ on the brain: liver large and pale; spleen very large and soft, not infarcted. These cases concur with the last-named in the text, as evidence of gastric hæmorrhage without vomiting of blood.

lated ; there seems to be either a deposit on the surface or a turgescence of Brunner's glands ; congestion slight. Jejunum—nothing particular seen, a few lumbrici. Ileum—nothing peculiar, Peyer's patches healthy. In the second case, the three segments of gut are described as contracted and pallid, their contents a blackish mucus, bile-tinted below, scanty ; no hæmorrhage, or even congestion, of the surface anywhere ; all parts alike.

Jejunum.—Total inspections 37, pallor or no change in 21, congestion or petechiæ in 16. As contrasted with neighbouring portions, there is no peculiar change here. For convenience, I may mention the inclusive details of two interesting cases :—

F., 20, died suddenly of cerebral hæmorrhage at end of relapse : several ounces of reddish serum in the peritoneum ; small intestines—walls of normal thickness, muc. membr. generally pale, but throughout occasional hæmorrhagic and congestive patches (about 12 in number) extending over 3 or 5 inches, isolated, visible from the outside ; commonest in jejunum, and at one, a short invagination easily reduced ; lumbrici all along. Peyer's patches barely visible in the ileum, which in general was normal in aspect ; mesenteric and lumbar glands not altered. Stomach healthy, and so large intestines throughout : liver large, pale : spleen large, infarcts (?)

M., 60, dying 42nd day after a febrile attack, exhausted. Small intestine—on the peritoneal surface old hæmorrhagic spots of dark purple hue. At the upper part of the jejunum, sub-mucous extravasation extending over a space $1\frac{1}{2}$ inch wide, and involving the *valvule conniventes*, the mucous membrane here on the surface tinged red (? local inflammation) ; minute, recent hæmorrhagic spots in the ileum. These changes seemed to belong to the sequelæ of specific fever. See Case XCVII., Chapter VI.

Ileum.—Particular interest attaches to changes here, not only from their comparative frequency, but because the same part is specially affected in enteric fever ; and hence a question might arise if there be any connection between spirillum fever and typhoid. In Bombay remittents, too, the lower end of the ileum is sometimes inflamed, with apparently little other morbid change ; but all fevers I have seen differ from typhoid in their implicating the ileum only occasionally, and then in different degrees and in wholly diverse manner. The fact, however, remains ; that the termination of the small gut and beginning of the large, are particularly prone to hyperæmia in several acute infections, or 'idiopathic fevers,' which thereby acquire a certain bond of relationship.

The statement that in $\frac{1}{2}$ of all autopsies, some amount of congestion, hæmorrhage or inflammation of the ileum is present, seems to be valid, and the proportion may be larger. Different opinions would doubtless be held respecting the import of the simpler changes found in spirillum fever, and I express only my own when laying some stress upon the details given below.

The 21 instances when the intestine is reported as healthy-looking, include 15 of deaths during specific pyrexia (both first and second attack) ; and it must therefore be held that the ileum may be quite unaffected, in even typical severe cases of spirillum fever. 7 such records noted by myself are before me, and I am confident that any marked change would have been mentioned. This does not imply that minuter lesions were absent ; I speak of the ordinary aspect, and include pallor amongst the quasi-normal conditions of the mucous membrane.

As to the exact site of vascular changes in the ileum, very seldom

was it the upper end or that next the jejunum alone, but most frequently (viz. 13 in 21) the lower end or that next the cæcum is named, usually alone. The terms applied here were as follows :—

Deaths during invasion-attack 4 : some vascular patches near close of ileum ; cæcum congested ; small intestine a little congested at the end : patches of congestion in small intestine, ileo-cæcal valve congested, Peyer's agminated glands slightly enlarged but not diseased (Mr. S. W.) : lower end of ileum marked with several minute sub-mucous extravasations which are not situated near Peyer's patches ; in the jejunum a few of the same are to be seen, and several in the duodenum below the opening of the common bile-duct, which is free. Deaths in first apyretic interval 3 : congested patches towards lower end of ileum, no erosion, contents of gut mucus and a few round worms, walls thinned (1) : congestion and spotted patches on jejunum and ileum, Peyer's glands unchanged (2) : ileum, some congestion and possible erosion of Peyer's patches, but the change is barely visible ; mesenteric glands hardly altered (3). Deaths in first relapse 3 : patches of hæmorrhage at base, of several *valv. conn.* in the ileum, and more diffused congestion at the lower end, no change in Peyer's glands, large petechiæ in omentum and mesentery, mesenteric glands turgid and some of the deeper lymphatics contained a fluid dark as blood, thoracic duct nearly empty (Case LXXXVI.) : small intestines unchanged except a small patch of deep congestion (hæmorrhagic) about 3 feet from cæcum, Peyer's glands visible as small sago-grains (2) : congestion and hæmorrhagic spots concentrated around Peyer's patches, especially at one place about a foot above the ileo-cæcal valve, no ulceration ; higher up the *valv. conn.* were of deep hæmorrhagic colour, which gradually became less as the jejunum was approached ; mesenteric glands not particularly enlarged (3).

The following instances call for special notice : the first two were of pronounced typhus type and accompanied with deep jaundice ; spirillar infection was always unmistakeable ; the local lesions described seem to be of a common character, and if not specific yet peculiarly intense and widely different from the typhoid lesions proper.

M., 25, died 6th day invasion : abdomen tympanitic and tender. Stomach distended, congested and marked by a few petechiæ ; duodenum unchanged except by congestion of a few *valv. conn.* : jejunum empty, a pink patch midway : ileum also pale till about two feet from its end, when a deep red tinge abruptly began and increased in depth to a deep purple just above the ileo-cæcal valve, here the mucous membrane was covered with a thin white film, resembling a diphtheritic layer, of somewhat granular aspect after gently washing, and thickest over some of Peyer's patches ; no erosion underneath it, or any enlargement of the follicles solitary or agminate ; no thickening of the muc. membr. over the patches, or deposit beneath it in any part. Villi turgid and folds swollen ; muscular and serous coats of intestine unchanged, and corresponding mesenteric glands pale and hardly enlarged. Slaty patches and a pink stain or two in the cæcum ; no vascularity in rest of large intestine. There was a very large liver and spleen (infarcts) : autopsy after 1½ hrs. Case LXXXIV., page 215.

M., 24, d. at acme of invasion, with the symptoms of *typhus biliosus*. Liver large, pale ; spleen 9 ozs., no infarcts noted ; kidneys large, pale. Congestion and hæmorrhage in stomach and upper part of small intestine, also in cæcum and colon : in the ileum above vascular streaks in direction of *valv. conn.*, at the lower end and at first part of large intestine, the mucous membrane is covered by a granular exudation seated upon a vascular basis, which in the cæcum is often the site of hæmorrhage ; this diphtheritic layer is continuous over Peyer's glands, which are visible but not diseased in aspect (Mr. S. A.). With a lens the granular layer is seen to be formed of thickened epithelium covering the villi everywhere, especially on *valv. conn.* and between the follicles of Peyer's patches (which themselves are rather indistinct) ; in the cæcum the exudation is more evidently granular, and is attended with greater vascularity. Coats of the intestines rather thinned. Case LXXXV.

M., 30, death on 8th day of second interval, at the close of secondary fever : there was some jaundice and a very low state ; abdomen retracted. Liver and spleen little

altered in aspect, muc. membr. of duodenum and jejunum stained with bile; Peyer's patches distinct, but not enlarged: the ileum was marked with vascular patches two inches to a foot in length and of intensely deep blood-hue, only the mucous coat was affected and no sign of peritonitis present (Mr. S. A.). These appearances were regarded as indicative of Enteritis. Case LXXXVIII.

Instances already mentioned above, illustrate the transition stages towards mild degrees of intestinal implication. Sloughing of the mucous membrane or sub-mucous structures was not seen; the mucous and serous coats were involved to a slight, though varying, extent, and only sometimes was the site of the inflammatory patch within revealed from the outside: peritonitis and serous effusion always absent.

Peyer's glands, solitary and agminate.—Having incidentally shown above that these structures in spirillum fever rarely seem to be changed, even when the mucous membrane over and around them is greatly affected, it may suffice to add that on 22 occasions specially noted and referring to all stages of the fever, they were normal in 18, somewhat enlarged in 1 and apparently congested or eroded in 3; these last examples being neither uniform nor pronounced, were at the time regarded as simple accidents. This opinion is abundantly confirmed by wider unrecorded negative experience, and it may be said that the local lesion of enteric fever does not in any degree attend spirillar infection.

In entire accordance with the above observations are those regarding the state of the mesenteric glands connected with the lower end of the ileum; these lymphatic structures being never found affected as in typhoid.

Tubercular disease of the small intestine was never seen.

Histological Note.—Granular (diphtheritic?) on mucous membrane of Ileum. In Case LXXXIV., I found the new layer to consist of translucent material loaded with Micrococci and Bacillary filaments, some red blood-discs and a few fat globules; it was wholly superficial, and formed a complete cast of the surface, furnishing a sheath to the villi and a lining to the follicles of Lieberkuhn: very few nuclear bodies were seen within it, and no peculiar structure on the addition of acetic acid. The subjacent epithelium of the mucous membrane and its appendages, was unchanged; the vascular congestion here was sub-mucous, and neither superficial cell-proliferation nor deep-seated micrococcus-collections were detected: the other coats of the intestine did not appear to be thickened. In the second case, also, the exudation contained little or no fat, being composed of granular matter and epithelial cells. Circumstances did not permit of more thorough scrutiny; so far as appears, the new layer was neither 'croupous' nor 'diphtheritic,' but rather of 'fibrinous' character.

Large Intestine.—Changes here are neither peculiar nor usually pronounced; their relationship to symptoms, also, was less evident and frequent than as regards other viscera.

Of 66 observations 18 showed congestion, 4 hæmorrhage with congestion, 10 ulceration, and 34 (51 p. c.) a healthy or quasi-normal state.

Congestion was proportionately commonest in the cæcum, and there

exists either independently or as a prolongation of disease from the ileum, in the intenser form which has been already described ; this persistent vascularity was neither widespread, nor deep ; it might be scattered.

Hæmorrhagic spots, always in conjunction with hyperæmia, were not often seen in the large gut ; being never numerous except when co-existent elsewhere.

Ulceration was limited to the rectum and adjoining colon ; if old, it was dysenteric, and even if recent might be so, when dysentery supervened as a complication or sequel of fever. There seems not to be any direct tendency to ulcerative inflammation in this fever, nor are the ordinary dysenteric forms modified by its co-existence. Ulcers were always small and shallow, and if numerous they were of inconsiderable dimensions and depth ; a sloughy aspect was very rare, and much attendant vascularity hardly ever seen : acute tropical ulceration was never observed. The rectal changes were sometimes mere erosions of the mucous surface.

In both the cases of gastric hæmorrhage, I found the cæcum congested : in one it was noted also as inflamed and slightly eroded ; contents a thin black mucus, with a vascular patch in the transverse colon, and rectum unchanged ; in the other the cæcum was of a slaty hue, and its contents thin feculent matter of reddish purple tint : the ileum was unaltered in both cases.

Lymphatic System.—Upon many occasions the contents of lymphatic glands chiefly in the lumbar region, and of the thoracic duct at its beginning, were carefully examined in subjects dying during the height of fever : the results were always negative so far as regards signs of the spirillum, and I therefore concluded that the parasite and its germs do not find access through these channels to the blood, as for example by means of *ingesta*. In 9 observations on the lymph in the thoracic duct, the stage was usually that of first attack, the mean hour of autopsy after decease 7, but four times only 2 or 3 hours. The mixed lymph and chyle contents were fluid, pinkish, clear (seldom opalescent), red discs of varying aspect, leucocytes, and very little fatty matter : sometimes large granule-cells, active protoplasmic masses and cells swarming with active granules : also large granule-cells inclosing red discs. Amongst elements probably abnormal, were large endothelial cells, containing fat granules, such as had been found in the blood immediately before death ; once in a girl of 14, dying on 7th day (? acme), and once in a boy of 14 dying about the same epoch ; in this last case blood from the right ventricle contained a few spirilla, at the time when none were seen in the contents of the thoracic duct. (*Vide* Plate II, letter P.) Inferences regarding the comparative prevalence of the parasite at the end of pyrexia (when death is more frequent than at any other time) require unusual care, as the following instance showed :—

M., 30, admitted during a relapse, furnished blood at 8 A.M. swarming with large spirilla, large vacuolated cells, and large fatty endothelial cells ; he died at 10.30, and the body was opened two hours later. The fluid from the thoracic duct did not contain spirilla (though there were some active moving specks), nor did the dark red fluid issuing from large lymphatic trunks joining the *receptaculum* ; spirilla

were not seen after death even in the prepared blood, and none were seen in a specimen taken just before death: here the probable explanation is, that the parasite suddenly disappeared from the blood in the interval between the first morning observation and decease.

The l. glands in the hilus of an infarcted spleen, though slightly enlarged were not materially changed; their contents a fluid with cells of normal aspect, nuclei unchanged and very little fat: at the most, the leucocytes may have been rather unusually numerous.

Once in a casualty during invasion (petechiæ, ulcers in the colon) the lumbar glands alone were enlarged and of a pink colour, their vessels filled with a dark red liquid and small hæmorrhages were present around the *receptaculum chyli*. In another case the mesenteric glands were turgid, and some of the deeper lymphatics contained a fluid dark as blood; the thoracic duct was nearly empty. Highly-tinted lymph I have found in other non-febrile diseases, especially in profound anæmia.

3. LESIONS ACCORDING TO STAGE OF FEVER.

In estimating the frequency of morbid changes in deaths occurring at the successive stages of spirillum fever, it is necessary to distinguish between actual and proportionate frequency; and for this purpose I have compared under each disease-heading, the number at each stage with the number at all stages (or inclusive mean).

Some particulars of weights of organs are subjoined.

The results obtained even if approximately accurate, are not without significance; and they would be usefully contrasted with European data, were such known or accessible to me.

First or Invasion-attack.—Brain: 28 autopsies of which 20 febrile (mostly at acme), and 8 dating at critical fall. Normal aspect 0; congestion alone 1, but in combination with other changes 14, or much above the inclusive mean; it is somewhat seldomer in deaths during crisis than in the febrile series. Hæmorrhages 7 (in one-fourth of instances or much beyond the total mean); probably all occurred during pyrexia; most were petechial and do not account for death, copious and fatal hæmorrhages (febrile) were 2, or proportionately six times less frequent than at first relapse, when, however, cerebral petechiæ were not noted. Serous effusion 16 (1:1.7) found in one-half deaths during pyrexia, and in three-fourths of those during the fall; it is commonly attended with congestion, sometimes with pallor, of the brain-surface. Pallor 3; an old clot 1. The invasion-attack being the severest, death occurs then in highest ratio; at acme of fever, congestion and effusion of serum, sometimes of blood in small amount, are found; and at critical fall, serous effusion, less congestion and very rarely hæmorrhage. The adult brain-weights ranged from 35–50 ozs., bearing no fixed relation to aspect of lesion: with copious cerebral hæmorrhage the weight did not seem above the mean, and perhaps the heaviest brains were the congested and œdematous.

Lungs 38 autopsies: quasi-normal 5 (about the inclusive mean), pale or collapsed 13 (rather oftener than mean), simply congested 9 (about the mean), inflamed or consolidated 4 (or nearly three times less

frequent than the mean), hæmorrhagic spots 4 (much beyond the mean); bronchitis 1 and pleurisy 2. The state of the lungs, therefore, varies without tending to pneumonic inflammation: a quasi-normal aspect was rather most frequent at defervescence, a collapsed state equally during and after pyrexia, congestion of all form and degree was present in one-half deaths both during and at close of pyrexia; whilst inflammation was seen only once in 28 deaths during pyrexia and then near to the end, it was noted 3 times in 11 deaths at the fall (lytic character often apparent); hæmorrhagic spots were found only at deaths about acme of fever. Mean weight of right lung in 18 adults 14.7 ozs. (pneumonia excluded), range $9\frac{1}{2}$ –24 ozs.; the left lung weighed rather less: congested and œdematous lungs were heaviest, the dry and collapsed lightest, but apparently without fixed relationships; quasi-normal lungs being sometimes nearly as heavy as those in the seat of pneumonia. As only one lung, or parts of a lung, may be consolidated, the mean weights are not of precise significance: an inflamed organ has been found to weigh 28 or 30 ozs.; usually it was about 20 ozs.

Heart 39 autopsies, of which 28 febrile and 11 at fall. Quasi-normal; (2 at the fall); clots in the cavities 25 (proportion about the total mean and oftenest in febrile deaths); pallor of the heart-muscle 5 (slightly predominating during pyrexia); hæmorrhagic spots 4, of which 3 in 11 deaths at the fall and in ratio much exceeding the total mean. Mean weight in 19 adults 8.5 ozs., which is decidedly in excess of normal estimates for Natives; range 6–12 ozs., and the maximum not in oldest subjects. In young persons it has been distinctly noted that the heart was large (especially the left ventricle), its substance being both pale and soft. (*Vide* CASE XII., Chap. II.)

Liver 39 autopsies, of which 28 febrile and 11 at fall. Quasi-normal only 1 (early febrile period). Enlargement 23 or in $\frac{3}{8}$ ($\frac{1}{2}$ in total mean); it is commoner in deaths during pyrexia (18 in 28) than during the fall, as might be anticipated on general principles and from clinical study during life, which shows that augmentation in volume of the liver varies at different stages of illness. As estimated by weight, these fatal cases (manifest anomalies excluded) furnish the following figures:—Invasion-attack, mid-febrile period 6 cases, mean weight of liver 43 ozs. (normal mean 42 ozs.); at acme and estimated close of pyrexia, 12 deaths, mean weight 62 ozs.; at beginning and middle of critical fall, 5 cases, mean weight 55 ozs.; and at end of crisis, 2 deaths, mean weight 44 ozs., or again but little in excess of the normal mean: here the rapid augmentation and decline of volume coincident with acme and fall of fever, and with marked changes in the blood, is striking. Simple congestion 5 (slightly less than the mean) and often seen at the fall (3 in 11). Pallor 1, of which only 1 at the fall: a mottled aspect 3, of which 2 at the fall.

Spleen 40 autopsies, of which 30 febrile and 10 at the fall. A quasi-normal state was rare, except when fever had ceased. Enlargements 24 or proportionately as often as the total mean, but decidedly more distinct during pyrexia than at the fall: thus, the mean weight of 30 spleens examined at whole invasion-periods was 13 ozs., at early deaths on third day of fever it was 4.5 ozs., on fourth day 7 ozs., on fifth day 7 ozs., on sixth day 8.2 ozs., on seventh day 10.7 ozs., on eighth day 16.4 ozs., at acme or supposed climax of fever 20 ozs., and when the critical fall had

taken place 11·7 ozs. Here the successive augmentation and diminution of volume of the spleen, contemporary with persistent advance and fall of pyrexia, are in accord with changes of the liver, as already shown; and they are even more strongly indicated as the following analysis will prove:—Comparative weights of liver and spleen, means of 30 cases: in deaths at fourth or fifth day of fever 6 : 1 respectively, in death subsequently until the reputed climax of fever 3, 5 or even 3 : 1, demonstrating the excessive proportion of splenic enlargement at this time; when the fall takes place, the volume of the spleen does not subside immediately, for the ratio still remains at 3·5 : 1.

Infarcts 8 (or $\frac{1}{3}$) are not disproportionately frequent at invasion (general mean $\frac{1}{3}$ of all autopsies), nor are they now oftener found during pyrexia than at the fall. The cases are too few for rigorous analysis, but it would seem that infarcts are usually formed about the time of febrile climax, when also the spleen becomes much enlarged in volume, and the state of the blood undergoes a change. I have already stated that their formation is not necessarily attended with excessive absolute increase of dimensions.

Softening 6 (or $\frac{1}{8}$) *i.e.* in a similar proportion to the total mean: this change was chiefly noted in deaths at the fall (3 in 10).

Kidnies 39 autopsies, of which 28 febrile and 11 at the fall. A quasi-normal state (6) seemed unusually frequent, after fever had ceased (3 in 11). Enlargement 6 (or 1 : 6·5, which is beyond the total mean of 1 : 8·6) and commonest during pyrexia; simple congestion 9, or rather below the mean, owing here to its comparative rarity at the fall; even during fever it was not invariable (1 : 3·5). A pale or fatty aspect 15 times, or in the same proportion as the total mean, and only slightly preponderating at the fall (5 or 1 : 2·2): hæmorrhagic spots were noticed only once in a death during fever. The granular condition seen twice seemed incidental.

The mean weight of right kidney (adult) was $4\frac{1}{2}$ ozs., range $2\frac{1}{2}$ –7, and seldom below this mean. As the normal mean weight is under 4 ozs., a general increase of dimensions with fever becomes as apparent here as of liver and spleen.

Intestinal canal.—Stomach: 6 autopsies, of which 5 at the acme or immediately after, with congestion of the mucous membrane or petechiæ, or both: in a case examined at the end of the fall, there was pallor of the membrane. Duodenum: of 9 autopsies 7 were of deaths at the acme and 6 of these with congestion or spots; 2 were at the fall (1 with congestion). Jejunum: examined with similar results except that congestion was not noticed at the fall. Ilium, the same; in early deaths pallor was found equally as congestion, and alone at the fall; whilst congestion with spots was found in 6 of 7 deaths at the acme. Cæcum: in the few cases inspected there was congestion in both early stage and at fall, it was not so frequent as usual at the acme: similar remarks apply to the colon and rectum.

First Interval.—This post-invasion period could seldom be termed apyretic in the casualty-lists, since secondary or symptomatic fever (always, however, of non-specific character) was almost invariable, and much oftenest it pertained to pneumonia.

Brain 9 autopsies. Organ always changed : simple congestion 3, both early and late, and 2 congestion with serous effusion, so that vascular repletion was met with much oftener than in the mean of all autopsies. Inflammation 1, the event being probably incidental to hæmorrhage : see CASE XXIII.

Serous effusion 5, also commoner than the mean, and found both early and late. Mean weight in 4 adults 44 ozs., or somewhat exceeding that during Invasion.

Lungs 12 autopsies, death in all being attended with vascular turgescence of the lungs. Thus, congestion 2, or about the total mean (deaths on 1st and 12th day) ; inflammation 10, equal to a ratio of $\frac{5}{6}$, or much exceeding the total mean of under $\frac{1}{3}$, so that Pneumonia becomes the special attendant upon this stage ; deaths were pretty equally distributed from first to tenth day, and once occurred so late as the thirty-second. As fatal pneumonia had occasionally begun to appear at the late critical fall, the present series was, in fact, continuous with that of the Invasion ; the influence of a possible second blood-contamination was not very apparent, though there were two deaths on 9th day of first interval and one on the 10th, when recurrent auto-inoculation was at least probable. See CASE LXX., Chap. V. and the Chart 24, Pl. VI.

Heart 12 autopsies. The proportion of cases in which clots in the cavities was found ($\frac{11}{12}$) is above the common mean ($\frac{8}{12}$) : a pale or seemingly fatty state of the muscle is mentioned only once, or less often than the average.

Liver 13 autopsies. Enlargements 4 only (almost $\frac{1}{3}$, whilst the total mean is nearly $\frac{1}{2}$, and mean of the preceding pyrexia $\frac{3}{5}$) ; so that this state is not marked during the first apyretic interval, and taking weight as a guide, I find the mean of 7 average instances was 47 ozs., or not much over the probable normal mean of 42 ozs. Hence the liver may very quickly after the critical fall assume a quasi-normal state, when it happens not to be implicated to a great extent (? structurally).

Congestion 4, or in ratio over the common mean, and also over that of the preceding febrile period ; even if approximately accurate, this datum is striking ; 2 instances of congestion died on 9th day, or about that of possible relapse.

Pallor 3, of which 2 were attended with enlargement, just as 2 of the enlarged organs were attended with paleness : a mottled aspect due to partial whiteness and congestion, was noted once (10th day).

Softening 1, death on 12th day : and once again with enlargement, in a longer sequelar death (32nd day).

Spleen 12 autopsies. A quasi-normal state 3 (2 with some congestion), the proportion being much larger than the common mean ; the instances were seen so late as the 9th and 10th days.

Enlargement 5, seen chiefly in the early days after the fall. The mean weight of 9 organs at this period was 9.7 ozs., as compared with a mean of 13 ozs. during the preceding febrile attack ; and, in general, the weight declined promptly after the critical fall.

Infarct 1, found in a death taking place on 1st day and therefore practically continuous with the invasion series ; the comparative rarity of infarcts at this stage ($\frac{1}{12}$ as contrasted with $\frac{1}{6}$) is evidence of their contingent character : here the usual cause of death was pneumonia,

whilst during high fever splenic disease (as an attendant on blood-contamination) was frequent.

Softening 3 (twice as often as common mean): the organ may be large or small: deaths on 5th, 7th and 12th days.

Kidnies 12 autopsies, of which 2 quasi-normal (seen late in this period), enlargement 2 (seen early), congested 3 (two at late date), pale or fatty-like 5 (distributed): none of these ratios differ much from the total mean, but more precise information regarding the state of the kidney, so far as indicated by its weight, shows that during this period the organ retained a volume (mean w. 4.5 ozs.) considerably above the normal (3.55 ozs.)

Intestinal canal. — Stomach 3 autopsies; congestion of mucous membrane and hæmorrhage 2 at early periods, pallor 1 at a later. Duodenum 7 autopsies, congestion and petechiæ seen twice at the beginning of this period. Jejunum 8, congestion twice at early periods. Ileum 8, congestions 3 distributed. Cæcum 7, congestions 2 at beginning and end: colon 8, congestions 4 distributed; rectum 7, congestions 3 distributed (with or without ulcers); once the congestion was marked, and attended a prolonged fall.

First Relapse.—Brain 7 autopsies. Once, as in the invasion, a quasi-normal state: congestion 2, after the middle, and commoner proportionately than in total or invasion mean. Hæmorrhages 3, seen towards the end and at the fall: the proportion is 1 in 2.3 which decidedly exceeds the other means named: from all these data, therefore, it appears that in the second specific infection with its culminating fever, the brain suffers more than at first. Extravasations of small size are not mentioned, and in each of the above 3 cases cerebral hæmorrhage was copious enough to account for death. Pallor of the brain was noted once at a later period.

Lungs 8 autopsies. Quasi-normal 3, an unusually large proportion: pallor and collapse (at the end) 3, or oftener than the mean total and identical with invasion-mean: congestion 1, less frequent than usual, and inflammation 1 only, in accordance with experience during the first attack.

It appears, therefore, that the lungs do not suffer more on repetition of attack than at first; and no new symptom then appears.

Heart 8 autopsies. In 2 the organ seemed to be normal (above the common mean): clots in the cavities 5 (in proportion about the mean), and 1 was pericarditis with hæmorrhage—a unique case elsewhere referred to, No. LXXVII. p. 208. The weight of the heart was sometimes more, sometimes less than at Invasion; but commonly in excess of the normal, and often strikingly so.

Liver 8 autopsies. The only entries are enlargement 5 and congestion 3: some departure from the normal aspect was always noted, and it seems that the liver is more affected now than in the first attack. Upon a slight re-arrangement the data give enlargement with pallor 3 (seen in early stage), enlargement with congestion 3 (seen in later stages), congestion 2 (at end and fall). In 7 average cases, the liver-weight rose from $36\frac{1}{2}$ ozs. (nearest beginning of attack) to 76 ozs. at climax of fever, and then declined at fall to 46 ozs.; the mean weight of the whole stage

being 57 ozs. These data are, I think, in accordance with clinical experience.

Spleen 8 autopsies. The entries are enlargement 6 and infarcts 2. Enlargement in $\frac{3}{4}$, which decidedly exceeds the means; and as both infarcted organs were also enlarged, it follows that in all these deaths during the first relapse, the spleen had an augmented volume. Tested by weight, I find the mean of 7 average cases to amount to not less than 20 ozs. including all till the climax of attack, which is in excess of the corresponding mean of invasion-attack; at the fall, the weight declined to 14 ozs. (corresponding weight at invasion 11.7 ozs.), thus, augmentation is quicker, greater and slower to subside in the relapse. In clinical study, the spleen was commonly felt more readily, and sometimes solely, in the relapse. Towards the end of attack, the enlarged spleen was twice noted as being of softened consistence.

Infarcts 2, or in proportion exceeding the other means: as in the previous attack, infarcts were detected after deaths towards close of fever and at the fall. The relapse, then, is so far a repetition of the invasion.

Kidnies 8 autopsies. In this series the organs were never entered as being enlarged, and upon analysing the weight-entries I find the mean weight of four kidneys removed at this stage to be 4.2 ozs., which is less than the mean weight of the organ at the preceding apyretic stage, and much less than at the invasion-period. Also in accordance herewith, the kidney is entered as being pale or 'fatty' twice as rarely here as in the invasion-stage, for organs so changed are usually large and heavier than normal. Congestion, however, was more common now (5 or 1 in 1.6) than formerly (1 to 3 or 4), whatever its significance: otherwise, it seems that the kidneys do not suffer so much as during the first attack.

Intestinal canal.—Stomach 4 autopsies: 2 pallor of mucous membrane, 2 congestion and petechiæ both at and after middle of relapse. Duodenum 6, 3 pallor, 3 congestion and spots chiefly towards the end and fall: jejunum 6, the proportions are the same: ileum 6, 2 pale, 4 congestion with spots. Cæcum 6, congested only once at end of fall; colon and rectum, no entries.

Second Apyretic Interval.—The lists here analysed include 10 deaths with autopsies dating at this period, viz. 2 on second day after crisis of relapse, 1 on fourth day and 3 on the eighth (day of possible second recurrence); these 6 may be called post-febrile: there were 4 others dating on fifteenth, sixteenth, thirtieth and forty-second days which, for clinical distinction, may be termed sequelar.

Brain 10 autopsies. Quasi-normal 1 (sequelar): congestion 2 (post-febrile), serous effusion 3 (post-febrile also), pallor 4 (3 sequelar); these last two conditions may be conjoined. Neither inflammation nor hæmorrhage was found at this time, and in this respect as well as by the predominance of serous infusion with or without congestion, the appearances now noted correspond (an exception apart) with those of the first apyretic interval.

Lungs 10 autopsies. Quasi-normal 1 (sequelar), collapse and usually pallor 5 (post-febrile), inflammation 3, viz. 1 dating fourth day and 2 sequelar; there was 1 of late date with old bronchitis and

emphysema. As compared with deaths in the first interval, the predominance of the pale and collapsed condition of the lungs is striking, and so on the other hand the comparative rarity of pneumonia shortly after the crisis (as a sequela pneumonia may at any time occur): the rarity or absence of congestion and absence of petechiæ, are features common to both apyretic periods.

Heart 10 autopsies. Clots in the cavities 3 (1 early and 2 sequelar), pallor of heart-muscle 6 (early and late and 4 with clots, the two conditions being here as previously sometimes co-existent): this last feature is in contrast with all earlier series except the first febrile, and seems to indicate that the heart-substance finally becomes much impaired. There was one death with old valvular disease.

Liver 10 autopsies. Quasi-normal 3 (of which 2 in post-febrile stage doubtfully normal), enlargement 3 (2 sequelar) all with pallor of the organ; a pale and a mottled aspect were seen in 2 deaths happening early in this stage, and there were 2 instances of liver-abscess dying on 4th and 16th days respectively; they were the only instances met with. Congestion of the liver (except once with mottling) was not entered, whilst it was not uncommon in the first apyretic interval: the higher proportion of quasi-normal states is another point of difference, but with regard to this large viscus with its considerable range of normal variation, the naked-eye judgments may need correction. I find the mean weight of 5 cases dying in this post-febrile period to be 51 ozs., that of the first apyretic interval was 47 ozs.; and in correspondence herewith, the mean weight of the liver was found to be higher in the relapse than in invasion-attack: all these data concurring to show a more considerable implication of the liver in second, than in first infections: subsequently, the organ in survivors regains its ordinary volume (if not becoming atrophied), thus in 2 sequelar cases the mean weight was only 40 ozs.

Spleen 10 autopsies. Quasi-normal 2 (post-febrile and sequelar): enlargements 6, distributed and sometimes attended in the later dates with softening, there were 2 instances of infarcts dying early (2nd and 4th days), and in one the spleen was entered as also large. Here the proportion of seeming enlargement is considerable, and upon testing the weights, I find the mean weight of the spleen in this apyretic interval to be 13 ozs., whilst 9.7 ozs. is that of the first interval: hence with this organ, as with the liver, the first recurrent attack induces greater enlargement not only for the time, but also for some days after complete subsidence of fever. The larger proportion of infarcts ($\frac{1}{3}$), contrasts with experience in the first interval: the change which they represent, is probably associated with vascular turgescence and consequent enlargement.

Kidney 10 autopsies. Quasi-normal 2 (both sequelar and indicating a restoration met with also in sequel of first apyretic interval): congestion 2, equally rare as in first interval: pale or quasi-fatty 6, of which 5 in the early post-febrile stage; this change was prominent in the first interval, and is even more so now; it is rarer during the pyrexial stages, which it therefore follows. Enlargement of the kidney is not once entered, and upon testing this rough naked-eye judgment by recorded weights, I find the mean weight of the organ

from 5 subjects was 4·1 ozs. which is somewhat less than that of deaths during the preceding fever (when also there were no entries of enlargement) and decidedly less than that of the first interval (when the same entries were few), and still less than that of the invasion-attack, when enlargement was entered as commoner.

Intestinal canal.—Stomach : congestion with erosion of the mucous membrane was found in one sequelar case. Duodenum—no entry ; jejunum—congestion and petechial spots of each 1 in 5 autopsies ; Ileum—the same with a second instance of more pronounced congestion. Large intestine : congestion and small ulcers (especially in the Rectum and all seemingly chronic) were found in most of 5 cases examined.

Autopsic data for later periods and the sequelæ, so far as acquired at Bombay, have been incorporated with the above ; information being still needed regarding deaths at second and subsequent Relapses, with their corresponding Intervals.

FINAL REMARKS.

The preceding data inclusive, show a 'quasi-normal' state of the organs in the following order of frequency :—Lungs and Kidnies (1 in 7·4 autopsies) ; Heart ; Spleen (1 in 12·3) ; Liver and Brain (1 in 18·19).

The lungs displayed this state oftenest in the febrile stages and especially at relapse, whence appears their more usual implication at or after cessation of fever : the kidneys oftenest at invasion and in sequel, whence appears their late implication and early recovery. The liver hardly displayed the quasi-normal state until the second interval, whence appears its early and severe implication ; the spleen oftenest in the two apyretic intervals, thus showing the comparative rarity of this state during fever and the tendency to prompt restoration after cessation of pyrexia. These remarks are based on a tabular statement of 65 fatal cases of spirillum fever of known duration.

Of the positive alterations recorded, some referring to blood-vessels and their contents are common to all organs—*e.g.* congestion (with its converse pallor) and escape of the blood-serum, impactions and rupture with extravasation, or finally, inflammation. Alterations of volume (usually as enlargement) noticeable in parenchymatous organs are mainly due to extra-vascular changes of the secretory cells, which at first become granular and swollen, and then charged with oil : the heart-muscle also undergoes similar degeneration.

The more apparent of these changes have a relation to fever-stages, as follows.

Congestion (or engorgement, usually venous, found *post-mortem*) whether general or localised, cannot be wholly relied on as significant of disease ; in some degree it was present during invasion-attack rather oftener (58 p. c.) than in relapse (54 p. c.), and in both febrile periods more frequently than during the corresponding apyretic intervals (45 to 30 p. c.) It is rarer at the critical fall in brain and kidney, not so rare in the lungs (where inflammation already tends to appear) ; and in the liver, it becomes the more frequent state at this time. Congestion of

the spleen has not always been discriminated from mere enlargement. Data for the succeeding stages are too few to permit close analysis, but it would appear that in the first interval congestion of the brain and kidneys increases, in liver lessens, and in the lungs is replaced by inflammation (in these fatal cases): during the Relapse it becomes generally commoner, being attended with hæmorrhage in the brain (sometimes also kidney), in the lungs however it is rarer; and during the second apyretic Interval congestion tends to subside, excepting in the lungs, where there is liability to recurrence of inflammation after the first relapse.

Such general survey is useful in showing how the different organs suffer from this fundamental lesion; thus, the brain and lungs which are non-secreting, display as a sequel to congestion, serous effusion, hæmorrhage and inflammation; whilst the liver and kidneys (secretory organs) are affected with granular and fatty degeneration of their cellular elements, and so the spleen, and to less extent the heart.

Hæmorrhages.—The most vascular organs are those in which extravasations of blood were largest and in themselves serious, *e.g.* the brain and, to a less extent, the lungs (pulmonary apoplexy) and striated muscles (*recti abd.*), and the diaphragm. I would add that the remarkable limitation of copious cerebral hæmorrhage to the times of spirillar blood-contamination (*i.e.* to the first and second pyrexial attacks) is well worthy of notice. Hæmorrhagic effusions when not fatal, may excite inflammation at their site.

Inflammation as clinically a primary phenomenon, was seen most prominently in the lungs. Pneumonia attended $\frac{5}{8}$ of the deaths during the first apyretic interval; it is also found at the critical fall, ushering in this stage, and it occurs at subsequent attacks and intervals, but in less proportion (*i.e.* in $\frac{1}{3}$ and not $\frac{1}{2}$ of fatal cases). The few other instances of lung inflammation (seemingly secondary or re-kindled) have been already alluded to. I can hardly doubt that a certain degree of cerebral meningitis pertains to the spirillum fever (see above), and other serous membranes than the arachnoid may be implicated (*e.g.* pericardium and pleuræ): that the liver, spleen and heart changes are inflammatory may be matter of opinion; they, too, are contemporary with specific pyrexia.

The concordance between surviving and fatal cases, as regards visceral lesions at different stages of the spirillum disease, is quite noteworthy; for not only does it furnish satisfactory explanation of ordinary signs and symptoms, but it also aids in diagnosis and prognosis when obscure or latent injury comes into question, and even may guide to prophylactic treatment. Very few of the details here given are without particular clinical meaning, and this, I think, will be obvious if they are carefully considered.

In estimating the pathological significance of anatomical changes there is, however, much room for judgment based on previous knowledge of disease in general; and it may be said that the greater such knowledge the less will be the disposition to insist upon any change as wholly peculiar, always excepting the fundamental blood-lesion itself. As regards structural alterations attending the febrile state, it is to the combination of these changes rather than to any one alone, that special import must be awarded; for it is doubtful if even splenic lesions are

wholly characteristic. Degenerative alterations of tissue have appeared to me as not of primary pathological importance, and I think they were less common or pronounced in India than in Europe. Incidental congestions, hæmorrhages and inflammations must claim the particular notice of clinicians, since the majority of severe illnesses and casualties were referable to them : nor are sequelar changes to be disregarded.

MEMORANDUM ON THE ANATOMICAL LESIONS OF RELAPSING FEVER DESCRIBED BY OBSERVERS IN EUROPE.—I am not able, nor is it requisite, to attempt an exhaustive survey of previous observations ; but from the selected data now submitted, it will be apparent that there exists a close agreement between the Bombay records and those made elsewhere. There is to be noticed here a continuous addition to our knowledge of anatomical lesions, which is strictly commensurate with improved methods of investigation ; and the amelioration of data may hence be capable of large future increase. The older observers did not insist greatly upon apparent changes of a coarser kind found *post mortem*, yet the splenic alterations were constantly noted ; and long ago Dr. W. Jenner sagaciously (as I think) remarked that whilst the liver and spleen are considerably enlarged, there is an absence of marked congestion of internal organs.

Proceeding to details, I have to refer to the later and somewhat more ample observations made in Russia and Germany. Dr. Küttner (Schmidt's 'Jahrbucher,' 1865, vol. cxxvi.) describes the lesions found in 125 autopsies at St. Petersburg : noting that *rigor mortis* came on very soon in those subjects dying of fever with high temperature, and later in deaths after the crisis of fever : the post-mortem appearances differing in the two series. After death at intervals of specific pyrexia, the brain is but little congested, but much serous effusion is present, the brain-substance being pale and soft : so the spinal cord which was examined 13 times. Lungs : œdema of the upper lobes and congestion below : blood-extravasation and hæmorrhagic infarcts at the periphery, and at a later period pneumonia with embolic infarcts, oftenest in connection with splenic infarcts and softening of the diaphragm. Heart : extremely pale and soft, distended with blood : the walls thinned (especially of left ventricle) : pale, grey spots in the heart-muscle due to localised fibrinous infiltration, which may be followed by fatty degeneration and either absorption or destruction of the muscular fibres. It is supposed that the slowness and weakness of the pulse are attributable to these changes of the myocardium. The walls of the arteries themselves are not thus affected, and therefore transudation of blood-serum is probably due to loss of tone in the vessels from altered innervation. The muscles of the extremities are at first unchanged, but afterwards become clouded and their striæ indistinct from granular (fibrinous and albuminous) degeneration : no changes like those in the heart were seen and the *glutæi* were always normal. The mucous membrane of the duodenum and jejunum was sometimes swollen, that of the ileum and colon much thinned. Liver : left lobe often much enlarged and in contact with the spleen, being covered with fresh exudation : white patches both superficial and deep, mottled the surfaces, in which the hepatic cells are found enlarged and much clouded with granular matter. Whether or not

jaundice is due to duodenal catarrh, seems uncertain. Spleen : during fever is much enlarged (once $4\frac{1}{2}$ lbs. and containing small abscesses) ; the pultaceous softening of typhus not met with : after fever, the splenic capsule becomes thickened and infarcts more distinct. Sometimes the spleen is carnified, and then with softening of the infarcts symptoms of septicæmia may appear. The explanation of these changes is not clear : possibly there is coagulation in the large venous channels of the organ. Kidnies : always much enlarged, their capsule thickened and opaque ; cloudy swelling of the epithelium was common, but intertubular cell-proliferation was uncommon and perhaps incidental. In deaths during pyrexia, there is a general injection of the tissues ; the serous membranes dry, and petechiæ are noted in the mucous membranes.

During the same period, 1864-5, Dr. Kremiansky of St. Petersburg observed in some fatal cases, extensive hæmorrhagic inflammation of the dura-mater, or arachnitis with meningeal hæmorrhage, limited to the top and sides of the cerebral hemispheres. At the same time, cerebro-spinal meningitis of severe form was known to prevail ; but I do not gather there was any connection between these two diseases. (See Dr. J. Millar in the 'Edin. Med. Jo.' Sept. 1865.)

Drs. Wyss and Bock ('Studien ueber Febris recurrens,' Breslau, 1868) mention and figure fatty degeneration of the liver-cells, amyloid degeneration of the vessels within the lobules, and suppuration in the centre of the lobules : also cell-infiltration of the inter-tubular tissue of the kidneys ; and in the spleen amyloid degeneration of the capillary network of the Malpighian bodies, with cell-infiltration of the tissue around.

Dr. Pastau (Virchow's 'Archiv.' vol. xlvii.) at Breslau, made 9 autopsies during the same epidemic of 1868. The subjects were mostly emaciated : muscles of the trunk and limbs not always changed in accordance with symptoms (muscular pains) noted during life : sometimes no change being found under the microscope, and at other times alterations ranging between slight granular cloudiness and complete fatty degeneration of all the muscles of the body. Cerebral congestion was noted : sometimes the heart was found empty, its muscular fibres were clouded with granules ; infarcts were seen in the spleen, and minute softened spots in which the cells had undergone retrograde metamorphosis ; the Malpighian bodies might not be prominent. In a case dying at an interval of specific pyrexia, the spleen was smaller, its capsule shrunken, and at the lower end there was found a pale, triangular infarct. The liver was also enlarged from increased congestion ; hyperplasia and enlargement of the hepatic cells not seen ; sometimes fatty degeneration was noted, at spots looking like those found in the spleen. Sometimes there was cloudy swelling of the urinary epithelium, together with congestion of the kidneys. The cases of fever at Breslau were probably not so severe as at St. Petersburg.

Dr. M. Litten ('Deutsch. Archiv. f. Klin. Med.' vol. xiii.) also at Breslau, 1872-73, mentions enlargement and often softening of the Spleen ; parenchymatous clouding of Liver, Kidnies and Heart ; disseminated ecchymoses and swelling of the follicular apparatuses (*sic*), as the ordinary post-mortem lesions, and not of themselves peculiar to Relapsing fever.

The most elaborate researches yet published were made by Dr.

Ponfick at Berlin, during the epidemic of 1872-73 (Virchow's 'Archiv.' vol. lx. part 2, 1874): they are based on 65 autopsies of subjects in good bodily condition, and mostly town-residents. It is considered that certain changes in the Spleen, the Marrow of bones, the Blood (large granule-cells); also of the Liver, Kidnies and Muscles (especially of the heart), pertain directly to Relapsing fever; and taken together, are pathognomonic. The splenic changes are said to be absolutely constant, and this assertion at once disposes of the commonly repeated statement that there are no constant local lesions in Relapsing fever. Liver changes, too, were invariable; but some difficulty here arose from the likelihood of prior lesion due to alcoholism. It is evident that the epidemic at Berlin was a severe one, there being seen several examples of *typhus biliosus*: the following is a summary of Dr. Ponfick's results. Liver: the turgescence ensuing during specific pyrexia, may be greater than occurs in any other infectious disease; the individual lobules become enlarged, their outlines indistinct and tint a greyish red. Microscopically, the increased volume is due to cloudy swelling of the hepatic cells (always present), to their peripheral fatty degeneration, and lastly to an infiltration of small cells in the portal canals: from an anatomical point of view, no distinction here is possible between the mild and severer form of relapsing fever: jaundice was present 16 times (24 p. c.), and it results from biliary engorgement. Kidnies: changed without exception, and in correspondence with alterations noted in the urine: they may be doubled in size: parenchyma flabby, the cortex broad and clouded; the Malpighian tufts pallid. Or parts alone may be changed; and where dark streaks are visible, there not only is the tubular epithelium more or less fatty, but the lumen of the tubes is occupied by fibrinous or blood-tinged plugs. Such cylinders with red discs have been found in the urine (not at Bombay H.V.C.) There is also evident in the extreme degree of swelling, a copious small-cell infiltration of the intertubular tissue; and besides, an amyloid thickening of the vessels which may be attributed to previous *morbus Brightii*. Striated Muscles: lesion of the myocardium is very frequent, its consistence flabby, tint pale grey or brownish, wholly or in streaks, where the fibres have undergone fatty degeneration; such degeneration may be as extreme as in the most virulent kind of infective disease, or even in poisoning by phosphorus. Dr. Ponfick naturally applies these data in explanation of certain fatal cases of fever, where death occurs by syncope and no other lesion is found after death: I have above remarked that the like were not witnessed amongst the temperate natives of W. India. Spleen: changes here are localised or diffused: the latter are always present, and induce a swelling of the organ sometimes greater than occurs in *leuckæmia*. The pulp is then dark, livid and projecting; the Malpighian bodies much enlarged or even effaced, their tint grey or yellowish; at a later stage of fever their outlines become more defined. In cases of unusually rapid turgescence of the spleen, rupture of its capsule may occur and death, with or without peritonitis: this change is compared with that taking place in Enteric fever. Swelling is due to distension of blood-vessels, and to a great increase of the cell-elements, including large multi-nucleated forms in near relationship to the cavernous veins. Dr. Ponfick could not find any *spirilla* amongst these cells. Numerous

pulp-cells were seen containing red blood-discs and pigment; and others filled with bright granules which look like spores, but probably are not such: these structures are not peculiar to relapsing fever, though found here in relatively larger numbers than in other fevers: they may be seen in the blood circulating during life, and when very abundant may be concerned with death of the patient: cases are quoted, such as occurred at Bombay. There is also another contamination of the blood, which can be demonstrated during life in severe cases: viz. by vascular endothelium-cells in a state of fatty degeneration: this, too, is not absolutely peculiar. As to localised splenic changes: the chief pertain to the venous system and comprise the so-called 'Infarcts,' which were present in 40 per cent. of all autopsies: they resemble closely embolic infarcts, but arise from a different cause than arterial obstruction and hence are peculiar to relapsing fever. Dr. Ponfick never found the small arteries leading to these infarcts blocked up, their lumen being always free and open; but in the veins leaving them, were seen coagula not recently formed: on consideration, the only explanation here seems to be that given by Dr. Cohnheim (yet this does not seem satisfactory as there are no capillaries in the spleen pulp. H.V.C.). Infarcts may undergo softening and necrosis, hæmorrhage attending; abscesses may form, which burst into the peritoneum or pleura, or take a much lengthier course towards the surface (nothing of this was seen at Bombay. H.V.C.). Arterial Infarcts (comparatively rare) are yellowish, pale spots and streaks of minute size, which pertain to the Malpighian bodies and small arterial branches in connection with them, and are due to multiplication of the lymph-cells of the former, and to cellular infiltration with fatty degeneration of the two outer coats of the latter: these changes seem to belong to the same series as those occurring in the bone-marrow, being due to inflammatory softening: the pale spots may become soft or diffuent in their centre, the appearances being those of minute abscesses: both arterial and venous infarcts may co-exist in the same case, though not commonly. Bones: their marrow undergoes changes like the splenic; 21 subjects were examined, the *femora*, *tibiæ* and *humeri* being divided, and some minute changes were always found; 6 times there was the gross anatomical aspect of a discrete abscess in the cancellous tissue. The short bones equally display minute changes; when these are marked the unaided eye can detect delicate opaque white lines in the osseous tissue, which are blood-vessels affected with fatty degeneration of their outer coat, sometimes their middle coat, and the capillary walls themselves with the tissue in immediate neighbourhood of the vessels being also implicated. Such lesions are found in white softening of the brain (and I have above described the like in the brain of fever-patients. H.V.C.): the result is impaired nutrition and death of the osseous tissue to a varying extent. Dr. Ponfick thinks a small sequestrum might become absorbed, without there being any external mark of injury; it is possible that the purulent collections in the cancellous tissue of the diaphyses of long bones may become the cause of chronic arthritis in the nearer joints: figures are given of these changes in the head of the *tibia* (showing a large pale greenish spot with a vascular edge), and in the shaft of the divided *femur* (showing minute opaque white spots in the marrow). Similar alterations occur in Enteric fever. The parts were not examined

at Bombay, owing to the sudden stoppage of my enquiries through illness. Respiratory organs : disseminated lobular pneumonia concurring with hypostatic congestion and inflammation of the ultimate bronchioles, was frequent ; the consolidation was not uniform or complete, not being due to cell-growth so much as to blending of small partially indurated areas. Fibroid pleuro-pneumonia was noted in 20 per cent. of autopsies, implicating an entire lobe, and commonest on the left side : it is the same lesion as occurs in typhus : the pleurisy was not marked (exudation serous), the pneumonitis had commonly reached the end of the second stage or that of incipient suppuration : infarcts might be seen in the softened lung. In the Brain, punctiform spots were sometimes seen in the *optic thalamus* and *corpus striatum*. Acute œdema of the glottis was noted in 31 per cent.; the vocal cords might be implicated, and afterwards perichondritis of the arytenoid cartilages occur ; these lesions are connected with the sudden and fatal asphyxia, not seldom seen at Berlin (less so at Bombay. H.V.C.).

Quite recently Dr. L. Lübmoff at Kasan in Russia, has furnished notes on the pathological anatomy of *typhus biliosus* ('Centralblatt. f. Med. Wissen.' No. 46, Nov. 1881), confirming in great part Dr. Ponfick's results. As regards the Spleen, whilst recognising the two main changes of enlargement of the Malpighian bodies and ordinary Infarcts, it is considered that the larger infarcts arise from blending of the swollen Malpighian bodies (see also my observations. H.V.C.). Dr. Lübmoff found some spirilla in these pale bodies, and appears to think they came from the white cells themselves. Transitions from the smaller to the larger infarcts were noted ; abscesses in the spleen are referred to super-added septicæmia, as *micrococci* were found in the blood-vessels concerned. The Liver-changes are two-fold, consisting of parenchymatous cell-swelling and acute interstitial inflammation, with metastatic suppurations. The Heart-muscle showed cloudy swelling. The Kidnies displayed parenchymatous nephritis, or disseminated inflammation and suppuration. The Bone-marrow was altered as at Berlin.

CHAPTER X.

DIAGNOSIS.

THIS heading comprises 1. The recognition of Spirillum fever as a disease separate and *sui generis*. 2. Its identification with the Relapsing or famine fever of Europe. And 3, its clinical discrimination from other febrile diseases. Whilst all these subdivisions are closely connected and of common tendency, the first may be said to concern the differential characters of the spirillum fever, the second to depend on points of resemblance or identity, and the third to include the two others, in so far as clinical diagnosis is sometimes effected negatively (or by exclusion) as well as affirmatively. To enumerate all the characters by which Relapsing fever is to be distinguished and all those which prove it and Spirillum fever to be the same disease, would be to repeat every detail (without, so far as I know, any exception) mentioned in the previous chapters and in some others following: this is impracticable, and I must therefore assume here that many distinctive phenomena are already known.

1. The recognition of Relapsing fever as a distinct disease.—It appears that in the past as at present, such recognition has been slowly effected; and it will be useful to trace briefly the diagnostic history of this fever.

Since specific diseases are not known to rise anew or radically to change, the opinion may be entertained that relapsing fever is old enough to have prevailed 2,000 years ago in Thasos (Grecian Archipelago); and doubtless it has reappeared on numerous occasions since Hippocrates recorded his observations. Yet although the striking characteristics of this disease must always have tended to facilitate its recognition, there did not ensue such clear apprehension of its specific character as might be anticipated; and on consideration, this tardy recognition will be found due to either some unusual quality of the disease or some defect of clinical method.

As to the first point, according to my experience the clinical varieties of Spirillum fever are both many and wide, and doubtless the entire rôle of the affection is not yet ascertained. In a practical sense, however, the disease commonly preserves its special features better than Typhus or Enteric; and therefore some incidental quality may be here concerned. Thus, as known in the British Isles, relapsing fever is pre-eminently of epidemic type, and hence being limited in both sphere and duration, it was never known clinically to most physicians, and carried with it the risk of being forgotten by those who once had

witnessed its presence. That it usually pertained to famine-periods and implicated almost solely the more indigent classes, are other circumstances not conducive to extended practical acquaintance. But most influential here would be its less serious nature as cause of death, and the comparative brevity of its acuter symptoms.

Another obscuring feature of the disease, was its nearly invariable conjunction with a much severer form of fever, namely *Typhus exanthematicus*, which eventually predominating, caused the milder Relapsing type of pyrexia to be often regarded as a mitigated form of typhus. Of late years, however, evidence has accumulated that epidemics of relapsing fever may be nearly or quite independent of typhus (*e.g.* the non-indigenous outbreaks in N. America, in several towns of Germany, and in Bombay itself): as regards some possible confusion of both these diseases, I am able to state that the spirillum fever in its abortive form and more pronounced degrees, presents phenomena closely resembling those commonly attributed to typhus.

There remains to note the retarding influence of defective clinical methods; and here it is observable that whilst clinical phenomena may be correctly described, yet their interpretation may be vitiated by spurious analogy or too vague generalisation. Thus, the often ill-sustained pyrexia of Relapsing fever has been attributed to the Remittent or even Intermittent type of malarious infection, and a seeming confirmation of such analogy was found in the periodical recurrence of fever which is common to malarial and spirillar infection. As regards past epochs, malarious fevers being widely prevalent in the British Isles it may well be conjectured that the rarer relapsing fever would be hardly discriminated from such, for even typhus in the 17th century was classed amongst the endemic diseases of Ireland under the name of 'Irish Ague'; and as regards current times, relapsing fever in Britain has been termed 'Remittent' fever, being also still recorded as such in that part of India I am best acquainted with. As regards 'recurrences' of fever, it was at least possible to confound with true relapses the sudden supervention, in typhus or enteric, of some febrile complication or the abrupt exacerbation of a previously existing complication, as pneumonia or pleurisy: and both remittents and typhoid are, in fact, liable to veritable repetitions. The connecting link here is the plain fact that in countries where certain types of fever prevail, the tendency of professional as well as of popular opinion, is to assimilate all febrile affections to some main form; or, in other words, to create a common type embracing many so-called sub-varieties; and of this statement no better illustration could be had than the past creation of a 'Continued Fever' in Britain, and a 'Remittent Fever' in British India.

Whether or not the preceding remarks be regarded as an adequate representation of events, there will always abide the striking circumstance that epidemic Relapsing fever was unmistakably described in Dublin 50 years (or much longer) since, without its then meeting with due recognition as a separate species of disease. Thus, the accounts of Drs. Rutty (1739) and O'Brien (1826) are so precise as to leave no doubt whatever as to the character of the fever witnessed; the *exacerbatio critica* being particularly mentioned and the 'crisis' (called the

'cool' by patients), whilst the main feature of illness was the liability to relapses; jaundice was sometimes well marked and the post-febrile ophthalmia named. (See 'Brit. and For. Medical Review,' vol. xviii. 1844, p. 188, whence this information is drawn.)

It is not, however, to be supposed that the diagnostic history of Relapsing fever is altogether peculiar; and without further comment, I next proceed to mention some succeeding stages of its identification in Great Britain, the principal indication of which is the fact that differentiation of the malady became clear according as clinical observation grew more precise and methodical. Details are here essential; and those furnished below besides immediately concerning the subject in hand, will serve as ultimate diagnostic memoranda: for intermediate historical passages reference should be made to Dr. Murchison's standard work.

The grounds upon which Dr. (then Professor) Henderson in December 1843, (*vide* 'Edinb. Med. and Surg. Jo.' vol. lxi. 1844) advocated the separation of the 'Epidemic Fever' then prevalent from Typhus, were, first a much greater frequency of the pulse at early stage of fever (*e.g.* 123 per minute as contrasted with 100 in typhus); next, a comparative rapidity of convalescence (the patient rallying a few hours after crisis, whilst in typhus defervescence is so prolonged as to occupy about one-third the entire attack); then, the critical sweats affording relief (there being no such conjunction in typhus); and, lastly, the propensity to relapse, which is 'as great as in intermittents,' and is never displayed by typhus. The presence or character of skin-eruption, or concurrent affection of the splenic, hepatic or urinary organs, or the state of the blood (as then known), are not insisted upon. As other evidence of distinction, the argument relied on the fact that one fever never communicated the other, and that an attack of the one conferred no immunity from an attack of the other.

About the same time, Dr. W. P. Alison pointed out as characteristics

1. The brief duration of the cases, which end by crisis mostly on the 5th, 7th, or rarely 9th day of illness.
2. The absence of the Measles-like eruption of typhus.
3. The frequent occurrence of jaundice, accompanied by more or less fulness and tenderness in the hypochondrium.
4. The unusual degree of sickness and vomiting.
5. The almost constant occurrence of a relapse, generally taking place on the 14th day.
6. The usual termination of the disease by profuse critical sweats.
7. The frequency of severe muscular pains of a rheumatic character during and after the sweating.
8. The very small mortality, not exceeding 1 in 30 cases seen.
9. Every pregnant woman aborted.

(Quoted from the 'Brit. and For. Med. Rev.' vol. xviii. 1844). Dr. J. R. Cormack enumerates, in addition, the sudden invasion of the disease and a bronzing of the countenance before and after the febrile seizure.

Dr. W. (now Sir William) Jenner soon afterwards ('Medical Times,' vols. xxii., xxiii. 1850-51), differentiated the three chief diseases included under the term 'Continued Fever;' furnishing detailed typical illustrations of each, and hereby laying the foundation of present opinion. Seven model cases of Relapsing fever are described, one ending fatally after the relapse from pneumonia; the liver being found large and mottled, and infarcts in the spleen. Perusal of these valuable data has

convinced me that the disease in Bombay was essentially the same as that seen in the London Fever Hospital ; temperature-charts are necessarily wanting, and the anatomical lesions of some organs are only cursorily described, the microscope not being then much used, yet in main particulars the concordance with my own memoranda is very close. Relapsing fever is thus defined—A disease arising from a specific cause, attended by rigors, chilliness, headache, vomiting, white tongue, epigastric tenderness, confined bowels, enlarged liver and spleen, high-coloured urine, frequent pulse, hot skin and occasionally by jaundice, and terminating in perfect convalescence on fifth to eighth day. In a week a relapse, *i.e.* a repetition of the symptoms present during the primary attack. After death, the spleen and liver are found considerably enlarged : absence of marked congestion of internal organs. Variations include gradual onset of the fever, epistaxis as an early symptom, diarrhoea at the crisis, or the menses in women : the relapse may be very imperfectly marked. There is no eruption as in Typhus or Enteric (here the Bombay fever differed somewhat). When death takes place, it is probably rather commoner at the invasion than at relapse. Some of the additional cases detailed show defervescence by lysis : a second relapse may occur in some cases. Death may occur by sudden collapse at the primary or second attack ; the case is here quoted of a girl, *æt.* 15, who died on the 12th day (epistaxis) with typhoid symptoms and lytic defervescence (as it seems) ; at the autopsy liver and spleen enlarged, absence of cerebral and pulmonary congestion and a generally healthy condition of the other organs, which taken in conjunction with the symptoms rendered the nature of the disease unquestionable. This instance particularly interested me, as example of death ‘by collapse’, which, in a strict sense, I had not seen at Bombay : so far as appears, the case was one comparable to those I have described as peculiar to the lytic form of defervescence when typhus-like symptoms are common. Delirium is occasionally the most prominent symptom in the relapse. Dr. Jenner shows that the fever was the same in 1850 as in 1846–7. Regarding ‘Febricula’ as a separate class of fevers, it is remarked there are no absolutely distinctive features or diagnostic points from the primary attack of relapsing fever ; but perhaps by paying more attention to the state of the spleen and to apparent cause of illness, some differential characters may be learnt. Here, most of all, becomes apparent the value of microscopical blood-scrutiny ; indeed, throughout most earlier records, the need of such an absolute test may be felt ; it is an advantage not to be over-rated.

Adverting now to Continental experience, I would first briefly allude to a well-known Article in the ‘British and Foreign Medico-Chirurgical Review,’ vol. iii. July 1851, where in reference to an epidemic recently prevailing in Upper Silesia, it is remarked that the probable reason why the German observers did not distinguish the two component elements (*viz.* Typhus and Relapsing fever) of the epidemic, was the circumstance that ‘they were not so familiar with typhus as English physicians are : this affection being very rare in many parts of Germany, and it was not until 1847 that many German observers believed that such a disease existed. In Scotland during 1843, the relapsing fever presented itself to men who were thoroughly acquainted with the endemic typhus ; and as

it was almost dominant, they were able to study it with great precision.' Yet more is to be said on this point; for not only was typhus hardly known, but relapsing fever itself was practically unknown in Germany until 1867-8. Thus, at Breslau, Dr. Pastau (Virchow's 'Archiv.' vol. xlvii. 1869) remarks that the Germans had no knowledge of the *febris recurrens* of Griesinger, this disease being for them a scientific curiosity known only through the writings of English physicians . . . then came the severe epidemic of St. Petersburg in 1864-5, which as a nearer calamity (that preceding in Silesia being insufficiently apprehended) aroused special enquiry, and the possibility of either overlooking or misinterpreting relapsing fever was gone for ever. . . . Dr. Pastau then describes the epidemic at Breslau, which beginning in April 1868, was one of the earliest recognised in Germany and the precursor of an almost continuous spreading series down to the present date.

Having above alluded to some of the earlier crude notions respecting Relapsing fever in England, I now add that similar misconceptions for a time first prevailed in other countries where the disease was a novel one. Thus, at St. Petersburg in 1864-5, it was suspected to be the Siberian plague, and then to be an abnormal typhus-form attended with a sort of relapse due to unusual circumstances, and the idea also arose that it might be an irregular intermittent; with careful observation and a reference to the literature of the disease, these doubts were removed and the identification of the new disease ('neuen Krankheit') with relapsing fever became established (Herrmann). Or it was considered to be a kind of abortive typhus, ending with crisis and followed by prompt convalescence; until the relapse soon following, the recurrent nature of the malady became recognised (Zorn). Or the first case seen was thought to resemble nearest enteric fever, from the high pyrexia, swelling of the spleen, tenderness of the abdomen and slight degree, at first, of head-symptoms: later on the persistent constipation, absence of eruption, short duration of the attack, and especially the onset of the relapse at a given date, showed the case to be one of recurrent typhus (Zuelzer).

When conveyed by Irish emigrants to the United States of America, the disease spread to some extent (local famine being here, too, absent) and was not immediately recognised. Thus, at Philadelphia in 1869, Dr. Parry remarked that the fever being a new one prompt diagnosis was impossible; not being either typhus or enteric, it was regarded as a severe form of 'remittent fever' and treated as such; large doses of the cinchona alkaloids given just before the primary crisis were thought to have induced this event, and hence the error of diagnosis seemed to be confirmed; 'only after the evident failure of these remedies either to shorten the primary paroxysm or to prevent a relapse, was it considered we had to deal with a new or unusual fever.' At New York, by the older medical men the fever was recognised as being the same as seen there in 1844 (when also it had been imported from Ireland); yet authorities admitted the great difficulty of prompt diagnosis during the first or invasion attack, prior to relapse if such were seen.

Recognition of Relapsing fever in India.—So far as appears to me, the course of events in the East has been closely like that reviewed above for England itself; this disease having been early and often described correctly, without its real character being duly appreciated. The

sanitary history of Northern India, especially, after 1860 may be said to bristle with accounts of local dearth and fever epidemics, both in country districts and the larger gaols wherein pauper prisoners were lodged. I have looked over the more accessible records of these epochs and am disposed to agree with Dr. Murchison, who states (*loc. cit.* p. 320) 'that a disease identical with the relapsing fever of this country (*i.e.* Great Britain) . . . prevails in conjunction with typhus in India and in the Punjab. It is said to have been recognised as far back as 1852 in the valley of Peshawur by Drs. Farquhar and Lyell, and excellent descriptions of it have been published by Dr. H. Clark, De Renzy, R. Gray and others.' Yet notwithstanding their admirably clear apprehension of the symptoms of the disease, medical officers hesitated to adopt the title of relapsing fever; *e.g.* at Agra, 1860, Dr. W. Walker regarded the affection as 'typhus,' other observers as 'typhoid'; at Jeypore, it was termed 'genuine yellow fever:' 'yellow typhus' and 'contagious jaundice fever' (Dr. Bateson) are other names given, and a Sanitary Commission was content with the cognomen of 'contagious fever.' (See an appreciative article by Dr. David B. Smith, in the 'Indian Medical Gazette,' May 1867). Subsequently, however, the recognition became distinct, for Dr. H. Clark, 1869 ('Ind. Annals,' vol. xxv.) expressly named the fever at Buxar on the borders of the N. W. Provinces, amongst a stud-establishment: Dr. J. L. Bryden also freely mentions relapsing fever in his Statistical Report for 1876; and Dr. W. P. Dickson, 1878-9 ('Ind. Med. Gaz.' June 1880) describes it by name at the Rupa Gaols (Punjab). I might, too, here refer to the Reports of the Sanitary Commissioners for the Punjab and N.-W. Provinces, 1876-7, for evidence that this fever has now become well-known to individual observers. Much reticence, however, continues to interfere with its due official recognition; though I am bound to add that the pathognomonic test has not hitherto been furnished in the Bengal Presidency, where the blood-spirillum still remains undetected (*vide* Rep. of San. Commiss. 1879). In the Bombay Presidency at least equal reticence obtains, in despite of the demonstration afforded in 1877.¹ In the more southerly Presidency of Madras, relapsing fever appears to be still unknown.

Lastly, adverting to my own late experience, it is worth stating that upon hospital records still preserved and dating from October 1876 to February 1877 (when famine in the Deccan had hardly attracted public attention), I had made notes designating successive fever cases as a 'typhoid' variety of remittents from the appearance of an eruption of the pink spots described above in Chapter V., together with abdominal tenderness and irregularity of the bowels: the temperature and pulse were daily entered, though too late or for too short a time (patients insisting upon early discharge after crisis) to suggest accurate diagnosis. Sometimes the note was a 'peculiar fever,' 'typhoid' or even 'typhus'; all this confusion resulting from want of the right clue. There are both early and late cases without the spots yet identical in other respects, and

¹ For other remarks see page 27. I would mention here that at Bombay in 1866, Dr. M. B. Colah described a case of relapsing fever seen by him in March (Trans. Grant Coll. Med. Soc.); and this testimony is valuable both in itself and in connection with the late epidemic of 1863-5, and also with a well-known outbreak amongst muleteers from Abyssinia passing through the town about this date, who became severely afflicted very soon afterwards on their way to Kurrachi and Mooltan.

these were marked 'famine-fever.' At last when called upon officially to enquire into the nature of the now prevalent sickness, I called to mind some shorthand notes of Dr. B. Sanderson's lectures I had heard in London, December 1873; and on the first scrutiny made of the blood of a fever-patient (see CASE V. page 21) the pathognomonic *spirillum* was detected, and diagnosis established for good. It is right to add that fever cases in the Bombay hospitals not seldom displaying a course of pyrexia at seeming variance with the descriptions in accepted text-books, some hesitation was at first felt, and expressed, in regarding all instances of spirillum fever as identical with the typical relapsing fever of Europe; but without entering into details of only local and personal interest, I may say that the difficulties met with arose partly from the extremely vague notions traditionally attached to the term 'remittent fever,' and partly from the not inconsiderable irregularity of the spirillar pyrexia itself.

Current nomenclature being indicative of existing medical knowledge and opinion, it is not irrelevant here to quote some suggestive synonyms chiefly from Dr. Murchison's lists. In Ireland, as contrasted with typhus relapsing fever was termed 'short fever,' 'five or seven day fever'; its less sustained pyrexia gave rise elsewhere to the name 'Remittent fever'; its recurrent tendency to that of 'Relapsing fever'; its contagious character accounts for the term 'Epidemic fever,' as applied to certain years and to certain places: e.g. the Scotch epidemic of 1843, the Irish, Silesian, Russian fever, or that of Dublin, Edinburgh, St. Petersburg and Bombay. A connection with want was intimated by the name 'famine-fever,' though this is not a frequent cognomen. The predominance of special symptoms, such as sudamina, and especially jaundice, has given rise to the terms 'Miliary fever'; 'Yellow fever,' 'Yellow relapsing fever,' 'Bilious typhus,' 'Bilious typhoid' and 'Bilious remittent.' At the present day the 'recurrent' character of the fever is generally accepted as its main clinical characteristic, hence the accepted nosological terms—'Febris recurrens,' 'Febris recidiva,' 'Typhus recurrens,' with corresponding names in the vernaculars. In adding one more appellation, viz. that of 'Spirillum fever,' a further degree of precision is aimed at. On the presumption that relapsing fever has long existed in India, it seems likely that some allusion to it would be found in the indigenous Medical works: I have not obtained any information on this subject, but to the North of the Bombay Presidency, the disease appears to be recognised popularly as the 'Gujerath sickness,' and as 'Yerkan' and 'Pilia' (signifying bilious yellowness), and as 'Bēlia' (signifying persistence? from *Bāl*, a long creeper without branches). There has been the same tendency in the East as in the West, to recognise the disease by local names; and it has been conjectured that relapsing fever of the severer icteroid type or complicated with parotid bubo may have been an element of the historic 'plagues' and fatal epidemics in regions where some would locate the home of Relapsing Fever (see page 29).

2. **Identification.**—The Relapsing fever of England is defined by Dr. Murchison as 'a contagious disease which is chiefly met with in the form of an epidemic during seasons of scarcity and famine': and these

features were unmistakeably present during the late sickness in Western India. Other European physicians have defined more strictly the relationship of relapsing fever to famine, and all agree as to its practical limitation to the more destitute classes of people; and so at Bombay the spirillum fever had a collateral as well as immediate connection with public want, being also mainly restricted to paupers and their neighbours. As in European cities, so here, the fever was independent of telluric and climatic influences, its course being regulated solely by movements of the impoverished masses; and with accordant in general characters, special resemblances were noted as regards clinical, anatomical and therapeutic features, without there being apparent any essential diversity or incongruous aspect. Clinical details being most readily estimated, I have enumerated below the successive symptoms named in the latest systematic treatise available, for purpose of comparison. *Vide* Dr. Lebert's article in Ziemssen's Cyclopædia Amer. Trans. vol. i. 1875.

Points of concordance.—An incubation-period of 5–7 days, more or less; mild prodromata or none at all; 'quick onset' of the attack, with or without chills or rigor; from the first high and persistent pyrexia with great debility, headache, severe pains all over, thirst, loss of appetite, sometimes nausea and vomiting, a remittent course with evening exacerbations ($105^{\circ}\cdot8$ to $107^{\circ}\cdot6$ F.), pulse rapid but weak, though with tolerable fullness and tension (*i.e.* non-dicrotic), skin moist at intervals, sudamina occasionally, tongue thickly coated, later on dry, bowels constipated or loose: early enlargement and tenderness of the liver and especially of the spleen, rapidly increasing; the muscles of the body the seat of the most severe pain, which is increased by movement or pressure and keeps the patient very quiet, though without the apathetic expression observed in other typhous diseases; nights sleepless; progressive emaciation and loss of strength. 'A sudden cessation of all symptoms' after 5, 6 or 7 days, attended with profuse sweating and rapid and great fall of temperature to below normal, and with diminution in frequency of the pulse; an *exacerbatio critica* sometimes precedes the fall. 'The patient's strength returns to a certain degree, he leaves his bed and frequently insists upon being discharged from the hospital'; 'this interval of apyrexia lasts on an average for one week,' and, though rarely, 'the disease may be completed by this single paroxysm.' The relapse begins unexpectedly, with or without chills or rigor; it takes a similar course to the first attack, generally however with milder symptoms, except that the liver and spleen become just as much enlarged as before; the remissions are quite as marked and the relief of the patient even greater than in the first attack. The duration of the relapse is usually shorter by two or more days and the critical defervescence is again attended by a profuse perspiration; there may be a pre-critical defervescence or a distinct *perturbatio critica*, and by morning the temperature and pulse have usually fallen below the normal. A third attack (second relapse) is not rare in many epidemics, the interval does not usually exceed 4 to 7 days, though it may be longer; the second relapse is usually milder and shorter than the first; a fourth attack (third relapse) is seen in some epidemics. The emaciated patient remains in a weak condition and suffering from aching pains for a long time, on the average six weeks and not seldom even more: the death-rate has been from 8 to 10 per cent when 'bilious typhoid' was included

in the reckoning ; death may occur from intensity of the fever, from exhaustion or from complications, of which that of pneumonia is particularly mentioned. Lastly, 'spirilla are never absent from the blood during periods of invasion and relapse, although they diminish very quickly after defervescence': the parasite reappears in the blood immediately with the relapse, to diminish once more on the approach of convalescence. I add that a general accord exists regarding the proportion of abortive and relapsing types of the fever, also regarding the proportionate number of relapses ; the concurrence of 'bilious typhus' ; the nature of 'complications,' and of 'sequelæ.' Distribution of disease according to sex and age is nearly the same ; the influence of contagion is equally demonstrated.

Points of divergence.—'Delirium is not a frequent symptom,' this could not be said of the Bombay fever : 'The urine possesses the characters usual in febrile diseases,' in the vegetarian races at Bombay the urine was plentiful and lateritious deposits were rare ; 'the anæmic murmur noticed often quite early in the neck and over the heart during the course of the affection, persists for a number of weeks later,' this description not altogether applying in my experience. Further, the increasing length of the successive apyretic intervals is not emphasised, and the red spots and petechiæ are not fully alluded to by Lebert, who also mentions at autopsies local softenings or abscesses of the spleen, which were not seen in W. India : the death-rate at Bombay was higher than in Europe, and cerebral hæmorrhage more frequent.

Summary.—Whilst the above-named points of accord include all the chief features and some secondary characters of Relapsing fever, those of divergence are neither numerous nor essential ; and the more precise comparisons I have had to make in preparing this work, do not interfere with the conclusion which must be already obvious, viz. that the Bombay fever was practically identical with the Relapsing fever of Europe. Doubtless, more extreme differences and even some seeming discrepancies might be collected in a wider selection of European writings, but they would not, I fully believe, transcend those natural variations which accompany individual epidemics at all times and in all places ; and to expect an invariable or absolute similarity of clinical characters in English or German and Hindoo subjects, would be to disregard the teachings of universal experience. I am able to add the important facts that the aspect and comportment of the blood-parasite are as nearly alike as can be imagined, in both East and West, and that inoculation-experiments in the *Quadrumana* succeed equally in Europe and India.

Reference should be made to the first subdivision of this Chapter, in conjunction with my definition of spirillum fever and the clinical summary in Chapter II. ; also to Chapter IX. where the fundamental morbid lesions are illustrated and compared, and to Chapter VIII. on the Mortality. It will be seen that the evidence now submitted is cumulative and special ; both kinds are equally valid, and each alone is sufficient ; for were the fact of a specific blood-contamination to be excluded from the argument (and I have here purposely laid little stress upon it), still the identification of the Bombay fever with the Relapsing fever of Europe would be completely established.

3. **Clinical and Differential Diagnosis.**—As with other acute diseases, the diagnosis of Relapsing fever is either certain or probable, being at the same time either prompt or delayed. In particular, the conditions of certain diagnosis are met with in cases displaying all the essential characters of the fever free of complication; these are an affirmative history of contagion, a relapse, and the presence of special signs enumerated above: such instances being practically unmistakable, need least to be insisted on here. Diagnosis becomes delayed and less certain in proportion to defects of personal and clinical history, less prominence of peculiar symptoms, and lastly to concurrence of obscuring complications. In hospital practice the completer data are often wanting, and then decision rests upon judgment of the physician: I do not know the usual ratio of instances thus alone estimated, but at Bombay at least one-fourth of all admissions were of obscure, irregular or seemingly anomalous character.

Relapsing fever, however, possesses a pathognomonic test, which never failing in the clearer cases was the only sure guide in the more obscure; and depending upon large experience, I feel justified in making by its aid a confident inference from the known to the less known. Many of the severe and most remarkable instances cited in this work, were recognised solely by careful microscopic examination of the blood; and without the employment of this means they would have been imperfectly understood, or erroneously estimated as belonging to other diseases, or else lost to clinical medicine in that large residuum of unrecognisable cases which has hitherto been inevitably admitted to attend febrile epidemics.¹ I do not say that *Spirillum* or Relapsing fever is to be invariably recognised by the blood-test; but I am able to affirm that when this test is strictly applicable, it settles for good a doubtful diagnosis.

Proceeding to details, I propose to discuss, first, the means and methods of diagnosis; and, next, the discrimination of particular diseases contemporaneously met with during my enquiries at Bombay.

A. MEANS AND METHODS.

Since demonstration is not always practicable and degrees of certainty (*i.e.* of probability) must be admitted, it becomes necessary to recognise gradations of evidence, somewhat as follows:—

1. Adequate scrutiny of the blood immediately before and during specific pyrexia (whether primary or recurrent) is a facile, prompt and unfailing means of detecting the spirillar infection. Its affirmative results have always, within my experience, been confirmed by other positive indications; and sometimes they have discovered infection when other means had failed or were impracticable (*e.g.* in patients moribund on admission), and when the revelation was otherwise unlooked for (as in instances of contagion in the hospital-wards). Non-detection of the parasite, as a rule, shows that the pyrexia belongs to either rebound of

¹ See for example Dr. W. T. Gairdner's 'Clinical Medicine,' p. 134, where it is stated that at Edinburgh, 'anomalous' fevers sometimes exceeded in number those of genuine typhus and enteric together. Probably such obscure cases are commonest in mild epidemics and at beginning or end of the severer; but wherever occurring, their accurate determination is of high practical importance.

temperature, or to fever symptomatic of local inflammation, or possibly to admixture of other idiopathic febrile disease ; and such negative evidence seldom erred, being often of great value in practice.

Soon becoming aware of the facts, it was my custom to examine the blood of every case of fever on admission into hospital, and, so far as possible, of all cases of fever arising within the medical wards during great part of 1877-8-9 ; the labour thus expended leading to the detection of most known instances of errant and diverging forms of spirillar infection, and to the conviction that only by assiduous blood-testing could the full extent of this disease be ascertained.¹

2. Accurate determination of the course of a complete attack of relapsing fever is valid evidence, and until 1873 this was the most certain means of diagnosis known ; it is that most insisted on by medical authors, and its practical sufficiency became fully recognised during the late epidemic at Bombay. Here the drawbacks refer to uncertainty of patient's previous history, when (as was usual) he is admitted after illness had begun ; to the variability of relapses ; and to the delay in diagnosis often incurred. Circumstances under which the evidence fails are—1. When the attacks do not distinctly recur ; and 2 in the majority of the severer cases, which, as shown elsewhere, die before the first relapse is due : opinion then may be formed upon the general history and condition, and on the particular symptoms seen (*vide infra*) ; yet these indications may be insufficient, for well-to-do persons are liable to attack, and the symptoms vary much.

3. Diagnosis becomes confident or presumptive, according to the force of testimony, when patients admitted after a week's illness at their homes show only a few hours' fever, or critical or post-critical symptoms, and no distinct relapse ensues ; when fatal cases are brought an hour or a day before death, and with non-characteristic temperatures ; when fever, though witnessed and uncomplicated, is irregular ; when secondary pyrexia obscures its course, or complications like pneumonia supervene, or apparently malarious or continued fever is blended with the spirillar. In these events, all more or less common at Bombay, I know of no sure diagnostic procedure except that of careful blood-examination, which in the more obscure is an absolutely essential guide. Otherwise, such instances not being amenable to ordinary rule, the hope of accurate diagnosis must depend upon a full acquaintance with each successive stage of spirillum fever, sagaciously applied to detect fundamental characters when but faintly indicated or almost eclipsed by accidental phenomena. The diagnostic features named by authors necessarily refer to average conditions and to pronounced illness (such as that of invasion or first relapse) ; yet it is not here, but in exceptional states that obscurity chiefly prevails. Famine may be absent (as in the late British and German epidemics) ; and I have known even first attacks far too ill-defined for ordinary recognition ; whilst all relapses are liable to extreme modification.

¹ Brief allusion should be made to the experimental pathology of spirillum fever (*Vide* Appendix A. No. 1) as furnishing an additional means of demonstration. It is likely that, before long, structural elements representing other stages in growth of the blood-parasite will be made visible, and thus the period of certain diagnosis become extended. And it may be that the success attending late investigation of this fever, will encourage parallel researches into the pathology of typhus proper and enteric fever.

The following are details to be sought in cases of doubt.

Ordinary diagnostic characters.—General and usually concurrent during epidemics. Non-dependence of illness on seasonal, malarial or insanitary influences, residence in a known famine-fever haunt ; the belonging to a destitute or pauper class, or to races and castes closest associated therewith ; emaciation or other evidence of want ; and especially the proof, probability or possibility of contagion in a family or house.

Particular features.—*a.* Patient's history : in a recent attack, the prompt onset and sustained course of pyrexia for 6–9 days ; in prolonged illness, additional statement regarding critical cessation of first attack, with subsequent debility yet tendency to convalescence, at the end of another week prompt reappearance of fever, to last 3–6 days, and again suddenly subside : about 10 days later, a second briefer recurrence. Initiatory chills are no guide, the fever may at first remit ; characteristic are critical exacerbation and the crisis itself, which is seldom wholly unindicated even with severe complication ; also the advent of first relapse, which may either nearly equal the invasion or be hardly recollected : secondary fever and local inflammations follow the crisis at brief interval. The incubation-period is probably about a week. Should a relapsing history and the general condition be in accord, they would prove a surer guide than actual state of the sick, when the symptoms are very mild or obscured by complications.

b. Symptoms witnessed.—Febrile: objective—high temperature with irregular remissions, pulse rapid yet soft, often quickest in the morning ; heart's systole feeble and often attended with booming sound or murmur, quick breathing ; fulness and tenderness of the upper abdominal zone, with implication of liver, spleen or stomach, tongue turgid, coated, moist ; an aspect dull and harassed. Subjective—extreme thirst, severe headache, pains in calves, knees, the back and upper limbs increased on movement, bilious vomiting sometimes, sleeplessness.

At the acme and crisis (about 7th day) night delirium, jaundice, tendency to the typhoid state, sometimes rigors, with exacerbation of pyrexia and abdominal signs, dyspnœa, restlessness and general distress. At the fall, rapid and deep decline of pyrexia with sweats and exhaustion, yet prompt general relief ; pulse not reduced proportionately to temperature ; aspect pallid and shrunken. Active delirium may now appear, and is perhaps peculiar. Combination of these final symptoms is almost characteristic : the local signs vary much, but pronounced individual symptoms—temperature, pulse, pains and particularly the abdominal signs, without evident complication, are significant. Defervescence in severe cases may be more or less interrupted and gradual.

During apyretic intervals.—Normal temperature restored, pulse for a time declines in frequency and may be quicker in the morning ; emaciation more apparent yet appetite good, if not excessive ; at first aching pains, cough or diarrhœa are common, and the muscular power improves slowly.

During the whole attack, complications most significant were hæmorrhages (often critical) such as epistaxis, apoplexy, ecchymoses of the skin and conjunctivæ, hæmatemesis, melæna, pulmonary congestion

and inflammation, acute abdominal congestions, deep jaundice (*typhus biliosus*). Dysentery at the fall ; later Parotitis.

Residual or sequelar phenomena may alone be seen : these include non-specific febrile reaction, local affections (usually febrile) of head, chest or abdomen, nervo-muscular derangements and post febrile ophthalmia, which though not equally peculiar yet need discrimination from idiopathic events ; an intelligent appreciation of the history of the case being here the guide.

Though the data in this work were all tested by the stricter method, yet it is likely that, after some experience, three-fourths of ordinary cases of spirillum fever would have been confidently identified, sooner or later, by the usual clinical methods. A distinct residuum of more or less deviating instances, would, however, have remained undetected : and if admissions in a serious or moribund state be added, the proportion of overlooked cases at Bombay would have been large. Further, amongst instances of contagion in the medical wards, I found examples with pyrexia so irregular and local symptoms so modified, as to become quite unrecognisable without extraneous aid. Thus led to almost unceasing use of the microscope, my clinical experience (with experiment) soon demonstrated that the spirillar infection was more widely diffused and more difficult of detection than hitherto supposed. This is important, since defects of diagnosis entail erroneous clinical views, overlooked sources of contagion and erroneous statistical statements of the disease.

B. PARTICULAR DIAGNOSIS.

The following diseases were known to be liable to confusion with Spirillum or Relapsing fever, at its febrile and non-febrile stages.

a. Febrile Stage.

Apart from scrutiny of the blood and an adequate clinical history, prompt discrimination based upon actual state of the sick is less decisive than might be anticipated, and personally, in cases of doubt I never omitted resort to the microscope.

General diseases simulated by ordinary uncomplicated spirillum fever. Malarious Remittents and Intermittents, Febriculæ, Yellow fever.

2. Those simulated where there occurs Lytic defervescence and the Rebound or Secondary fever. Malarious fevers, typhus, enteric, cerebro-spinal meningitis.

3. Those concurrent with Relapsing fever, obscuring its diagnosis. Probably all the above, hectic, varicella, erysipelas, puerperal fever, dengue ?

4. Local diseases induced by Relapsing fever and liable to obscure its course. Such are the affections previously described as 'Complications.'

Difficulties here attend chiefly the first or invasion attack of the disease, partly because of the greater severity of illness at that time, and partly from absence of a guiding clinical history. The above list would be greatly extended, if the simulating character of particular symptoms were to be separately discussed ; such as headache, delirium and severe body-pains, cough or dyspnoea, jaundice, vomiting and acute implication

of the liver, spleen or intestinal canal, all of which might give rise to the suspicion of either some other acute specific infection setting in (small-pox is named by Dr. Murchison) or of local visceral inflammation. But it is to be remembered that, then, careful blood-scrutiny will solve all doubts; and, besides, to rely for diagnosis upon a single common symptom or even limited group of such symptoms, apart from personal and clinical history, is to adopt a really unpractical method of procedure. If the blood cannot be examined on the spot, diagnosis should be suspended for a time or provisionally determined by way of exclusion.

1. *Malarious fevers: the Remittents so-called.*—In general characters, are distinguished by never showing the blood-spirillum, seldom relapsing and never at the same intervals, with similar gradations, as noted in famine-fever; their origin is malarious, their production seasonal and endemic, no class of an exposed community being spared and contagion being unknown: they are amenable to treatment by antiperiodics.

In detail, an attack of simple remittent fever can be compared only with the early and more prolonged events of relapsing fever, viz. the invasion and first relapse; the later relapses, as a rule, being comparable rather with intermittents. Clinically, it is necessary to consider separately the uncomplicated and the complicated forms of both fevers.

Simple form.—An uncomplicated remittent very rarely presents a beginning, and especially an ending, so abrupt as to simulate the ordinary spirillar attack; and it is also rare that the limited duration, sustained form and high intensity of pyrexia are comparable in evident degree: further, in the remittent the pulse and breathing rate are less, and the hepatic, splenic and gastric implication are wanting, as well as the peculiar 'facies.' As regards the type of fever, the 'remission,' however pronounced, makes no approach whatever to the prolonged apyretic interval between successive attacks of relapsing fever.¹

As diagnostics, particular states must, however, be estimated with caution; for, as regards pyrexia, whilst it is true that an uncomplicated remittent is seldom so long as 3-4 days well-sustained in form, commonly tending to intermit midway, first and last, and that the ordinary spirillar attack is usually sub-continued in form, only occasionally remitting deeply or intermitting; yet no invariable rule obtains, and many exceptions occur. I have also known the specific first relapse (and even the invasion) to be preceded by isolated paroxysms, and such may occur at its close; when, too, defervescence by lysis occurs. As regards particular symptoms and local signs, I note that whilst remittents of a sustained form are rarely free of thoracic or abdominal implication; on the other hand, the local signs usually characterising spirillar infection may be reduced to a minimum. From these remarks it will be evident that the variability of ordinary relapsing fever is a main source of difficulty, and that in the absence of blood-examinations the best guide here will be practical experience.

¹ This remark is needed because some authors (especially abroad) call the apyretic interval of recurrent typhus a 'remission'; the febrile event, whatever its duration, is also by some called a 'paroxysm.' I have preferred to employ these terms in their more usual sense as indicating respectively an abatement of temperature not to normal level, and an exacerbation of but brief duration.

Complicated Form.—Fever seemingly malarious, lasting 7–10 days, and finally attended with cerebral, pulmonic, hepatic or intestinal implication, is sometimes pronounced, sustained and defined enough to resemble the spirillar attack; and in particular, may pneumonia impart these characters. Under such circumstances I have found resort to the microscope essential to prompt diagnosis, especially when the history or general condition of the patient, or such symptoms as severe headache and pains, splenic or hepatic uneasiness, or early delirium indicated the possibility of specific infection.

At Bombay the complications of 'remittents' above alluded to, were (with few exceptions) such as to be found attending (and in not unlike proportion) the 'new' fever also, and this circumstance did not contribute to easy diagnosis; on the other hand, there were some 'accidents' of the spirillar disease which had very rarely occurred of late years amongst the local remittents (*e.g.* cerebral hæmorrhage).¹

During the periods corresponding to the first apyretic interval (less often than the second) of spirillum fever, there sometimes arose febrile complications which tended to efface those periods, and thereby produce a sort of continuous pyrexial attack. Since in such cases the superadded symptomatic fever pertained to bronchitis, pneumonia, hepatitis or dysentery, that is, to affections equally prevalent in the two fevers under comparison, the whole illness might with difficulty be discriminated from a complicated remittent. Its duration might extend after admission to fourteen days, or altogether to about three weeks (patients seldom resorting to hospital before the end of a week's illness at home); and since the local complication does not become either resolved or proportionately advanced, the co-existence of remittent or spirillum fever is usually to be suspected. If the latter, there would be some exacerbation of the pyrexia at date of probable relapse, and the superaddition of some splenic, hepatic or gastric implication, a quickened and feebler pulse, headache or pains, dryness of the tongue, increased distress and a tendency to the typhoid state; these symptoms lasting 3–5 days, culminating and then subsiding with some indications of a crisis, followed

¹ Murchison remarks on the diagnosis of these two affections (2nd. ed. pp. 394–5), that 'both commence suddenly, run a short course, have a tendency to relapse and are often complicated with sickness, jaundice and hæmorrhages.' At the W. Presidency of India such a statement so little applied, that were 'remittent' fever to become thus characterised, the suspicion would arise (in my own mind) of some confusion of diagnosis, and a conviction that further information was needed. I have not ventured to allude to the definition of severer malarious fever current where my work lay, and will observe only that cerebral, pulmonic, hepatic and intestinal affections occurring independently of obvious climatic influences, are usually regarded as being complications of such fever. Nowhere fitter was this warning of Griesinger (who had personal experience in the semi-tropical country of Egypt)—'Il ne faut pas vouloir faire entrer à toute force les processus pathologiques nouvellement étudiés dans les catégories connues jusque ce jour, et qualifier du nom de fièvre des marais toutes les maladies fébriles que l'on ne connaît pas bien et où la quinine est utile' 2nd. ed. French Trans. by Lemattre, 1868. Respecting the last line of this quotation, I should state that Griesinger thought quinine beneficial in his 'bilioses typhoid' disease, now almost universally admitted to be only a variety of spirillum fever; and as he held that the analogy of relapsing fever to the malarious was considerable, it seems probable that some confusion of diagnosis had occurred, which led to the introduction of the passage above quoted into the 2nd ed. issued after his leaving Egypt, it being absent in the 1st ed. of 'Infectionskrankheiten.' Demonstration of the blood-spirillum itself did not, at Bombay, suffice to overcome the mental prepossession referred to; and 'officially' relapsing fever remained undistinguished from 'remittents.'

by abatement, or greater weakness. The use of the microscope throughout an illness of this kind, first enabled me to see the import of the minor signs here stated ; and I greatly wish it had been in my power to employ other refined means of diagnosis, chemical and physical : increase of daily thermometrical readings did not promise help in discriminating the malarial, spirillar and symptomatic pyrexias. Of instances which remained somewhat dubious to me, CASE XCII. page 226, was nearly the only one : in it the pyrexia seen resembled an intermittent or rather a recurring remittent, but the duration of the attacks was limited to a few days and the relapses were tolerably regular, and besides an organism was found in the blood like to the spirillum : hence I decided the example was one of spirillum fever, complicated by renal disease.

Intermittents.—Though an isolated paroxysm of Ague-like form, never in my experience occupied the position of a primary or invasion-attack of spirillum fever in man, yet such was repeatedly seen as a relapse—seldom the first, and usually second or third. An ague-like paroxysm was occasionally also noted as a phenomenon intercalated midway between the invasion and first relapse ; and, lastly, a brief, isolated pyrexial event was not very rarely found both preceding and following a fully-developed attack, whether primary or (oftener) recurrent.

In the inoculated monkey a single, febrile paroxysm of varying intensity, is sometimes the sole outward manifestation of spirillar blood-infection ; and it yet remains to be ascertained whether a similar ‘febricula’ may not be a primary and sole (as it often is the secondary) result of systemic contamination in man himself.

A series of two, three or more such paroxysms—quotidian or approaching tertian in type—might represent, as an extremely rare occurrence, a primary spirillar attack in the sick ; less rarely, the entire first, second or third relapse ; and not unusually, two or three increscant paroxysms composed the earlier part of a recurrent attack. A few times, aguish attacks seemed to herald the invasion.

As ordinarily estimated, these events varied not less (perhaps more) than ague, in intensity, duration and hour of occurrence, and, as a rule, they were not attended with rigors or sweats so pronounced. Splenic signs were not invariable, and pulse-characters or depth of critical fall were not always alike, general symptoms equally varying.

Viewed alone, such paroxysms single or isolated may be undistinguishable from true Ague ; guides to diagnosis are their isolated position and periodic relationship to other undoubted specific attacks, the statement, condition and progress of the patient, and, independently of other aid, the state of the blood.¹ According to my experience all true periodic relapses, of whatever form or degree, are attended with renewed blood-contamination : whilst the like spirillar contamination

¹ It has been to me one of the strongest collateral proofs of the identity of the fever at Bombay with European relapsing fever, that the facts named in the text precisely correspond to those I afterwards found recorded by German writers of admitted authority. As instances of this kind have in India interfered with acceptance of the view now proposed, I subjoin some quotations showing how they are regarded in Europe.

Dr. M. Litten (‘Deutsch. Archiv. f. Klin. Med.’ vol. xiii. 1874) observed at Breslau during 1872-3, six such cases (temperature-charts appended) which beginning in the usual manner of relapsing fever, displayed at their first, second or third relapses a tertian

is never present during true Ague, in any shape. See Section III. Chapter I.

Febricula: simple continued fever.—Both the varieties sometimes distinguished as 'ephemera' and 'synocha,' might be referred to the simpler forms of spirillum fever; as these have lately become known by experiment on the Quadrumana, and through means of the microscope in Man himself. Nor is the absence of severe body-pains, splenic or hepatic enlargement, jaundice or certain general conditions usually attending relapsing fever, a valid argument against the view that some kinds of febricula are due to spirillar infection; for I have already shown how variable such symptoms may occasionally be. It is not, however, to be supposed that brief febrile attacks are all of one origin and nature; and when a suspicion arises from attendant circumstances that illness is due to contagion, I know of no other sure guide than examination of the blood by the best known methods.¹

Yellow fever.—Within my experience, this disease has not occurred at Bombay; yet since in an official Report the above term was applied to some cases under my care, which I regarded as examples of the icteroid variety of spirillum fever, it is evident that the same liability to confusion lately existed in India as formerly in the British Isles. True black vomit was occasionally seen in my cases, though not in conjunction

or a quotidian type of pyrexia; and inasmuch as during four of these ague-like relapses the blood-spirillum could not be found, he considered the evidence to be in favour of the view that true ague had here followed relapsing fever. It is however admitted that in two instances of similar ague-form, the parasite was plentifully found. It is added that not seldom, discharged relapsing-fever patients had intermittent attacks at their homes; that towards the end of the epidemic at Breslau, in the same dwellings some persons became affected with relapsing fever and others with ague; and that whilst at first intermittents were rare, yet afterwards with decline of the epidemic they became very common. After discussing the possible relations of the two kinds of fever, Dr. Litten then concludes by observing that at all events relapsing fever seems to produce a certain susceptibility to the malarious poison. I need only remark here, that by this competent observer no doubt whatever is expressed of the blood-spirillum being characteristic of relapsing fever; notwithstanding this seeming blending of fevers.

Dr. L. Heydenreich ('Klin. u. Mikroskop. Untersuch. über den Parasit. d. Rückfallstypus,' Berlin, 1877) at St. Petersburg, has equally noted that irregular (though not unperiodic) paroxysms of spirillar pyrexia may supervene in the course of relapsing attacks, which often cannot be clinically distinguished from ague. Many references to previous authors are quoted to the same effect, but Dr. Heydenreich did not regard these irregular paroxysms as being really aguish; nor does he express any doubt as to the significance of the spirillum. Dr. Spitz ('Deutsch. Archiv. f. Klin. Med.,' vol. xxvi. 1880), at Breslau, 1879, states that whilst numerous instances like those described by Dr. Litten were seen, yet by adequate and repeated blood-scrutiny the spirillum was to be detected during these presumed intermittent attacks; and he is not disposed to admit any real connection between relapsing fever and ague.

¹ Dr. (now Sir William) Jenner (*loc. cit.*) remarked a 'close relation, if not identity,' of the symptoms present in the primary attack of relapsing fever and those which occurred in a case described by him as illustrative of febricula; continuing 'I know no absolutely distinctive feature; my observations do not enable me to state the diagnostic points: perhaps by paying attention to the state of the spleen, and to the apparent causes of illness, some diagnostic characters might be elicited.' The obscure cases of fever under notice may now be tested by the means stated in the text; only the aid of an expert is desirable, for visible blood-infection is probably scanty at such times. Dr. Murchison (*loc. cit.*) especially mentions the frequency of *herpes facialis* as a character of 'synocha'; and Prof. Lebert (*l. c.*) the same as being common in 'recurrent typhus' as seen by him at Breslau: I did not notice this sign at Bombay in the dark-skinned natives. Pneumonic signs should be looked for in instances where *herpes* appears; for pneumonia is one of the affections likely to be included under febricula, and it is not uncommon in relapsing fever.

with deep jaundice ; and it never occurred to me, independently of blood-scrutiny, to confound relapsing fever with yellow fever. Not being acquainted with the latter by personal observation, I must refer to other authors for minor points of discrimination : the diagnostic data already given above, and especially examinations of the blood of the patient, should prevent all likelihood of error.

2. *Typhus exanthematicus*.—In the absence at Bombay of true typhus, I am not able from personal observation to add fresh diagnostic marks, in addition to those named by the European physicians who are quoted in the first subdivision of this Chapter. The ordinary clinical rules apply here, and need not be repeated. It is, however, desirable to state that after the height of the epidemic, there were seen cases of undoubted spirillum fever displaying the following characters—a distinctly typhus aspect, an eruption of ineffaceable red spots, a temperature not very high yet sustained, a pulse small and not excessively quick, with cardiac weakness most apparent ; splenic and hepatic implication, body-pains, jaundice, vomiting, by no means exacerbated ; night-delirium very frequent, and in general a low state tending to the ‘typhoid,’ coming on after so short a time as 5 or 6 days. In such cases, the pyrexial onset was reported as not abrupt ; the defervescence seen in hospital was less deep and critical than usual, or it was wholly lytic ; further, secondary fever sometimes ensued prolonging the pyrexial state to 12 or 14 days : and when, as was usual, such symptoms belonged to the first attack of illness, a relapse was not invariable. This remarkable congeries of clinical signs was highly suggestive of typhus ; yet the blood-spirillum was to be found and sometimes abundantly, during the first week of fever. As I have noted nearly equally suggestive symptoms in cases following the entire course of typical and uncomplicated relapsing fever, it must be allowed that the spirillar infection of itself, and alone, is capable of inducing a state closely resembling that of European typhus. Instances of this kind usually occurred together in a family, or in a house ; and upon visiting the dwellings of patients, no other kind of fever was to be seen.

Enteric or Typhoid fever : typhus abdominalis.—This affection is not very uncommon at Bombay ; but it was extremely rarely seen amongst famine-immigrants. The pyrexia of tropical enteric resembles that of malarious Remittent fever, rather than the European, hectic-like form ; and by its paroxysmal character, prolonged duration and ill-defined limits, it differs entirely from the spirillar. The relapses of typhoid are rare, few, of uncertain date and non-periodic ; and often they resemble the reactive phenomena termed in this work secondary fever, rather than true repetitions of fever. The peculiar signs of intestinal ulceration were practically absent in famine-fever cases ; and the blood-spirillum is assuredly never to be found in simple enteric.

With all these points of diversity, there are some of partial approximation, not unworthy of notice. Thus, the skin-eruption regarded in England as peculiar to typhoid, was at Bombay not rarely seen in cases of both spirillum and remittent fever without the least suspicion of a blending of enteric : so that diagnosis based on such spots was there of

no avail. Also the abdominal uneasiness in relapsing fever may extend to the hypogastrium, and even right iliac fossa ; diarrhoea was not rare, and after death signs of local irritation at the end of the ileum were not unusual.

Lastly, in connection with the remarkable Secondary fever following some spirillar attacks, there has been found distinct inflammation of the ileum ; and although in my own cases diagnosis was established by special means, yet with less experience and no aid from the microscope, instances of this kind were distinctly open to misapprehension.

Cerebro-spinal Meningitis.—The single case demonstrated at the larger Bombay hospital, was thought during life to resemble remittent fever ; but the idea of spirillum fever with cerebral complication also occurring to me, the blood was examined with negative results which proved the absence of spirillar infection. The occasional concurrence of cerebro-spinal meningitis with epidemics of relapsing fever, is like the conjunction with typhus, of considerable clinical interest.

3. Amongst febrile diseases incidentally concurring with the spirillar so as to obscure its diagnosis, were seen irregular malarious pyrexia, hectic, chicken-pox, erysipelas, and puerperal fever. In each case, diagnosis was either effected or aided by means of blood-examination ; general principles here applying.

Hectic.—An adult woman, phthisical, had persistent fever with occasional exacerbation, there was a possibility of relapsing fever from contagion, and being led to examine the blood on several occasions, the existence of recurrent spirillar infection was established. CASE LXXVI. page 207. In two other cases hectic was connected with abscess of the liver, and coming after cessation of relapsing fever it occupied the place of secondary fever ; local signs were not pronounced, and the non-specific character of the superadded pyrexia was manifest.

Varicella.—Occurred in patients who had gone through attacks of spirillum fever, and at first it was possible to mistake the fever for a relapse ; the eruption of chicken-pox appearing correct diagnosis became assured, yet before this it had been learnt that the blood was free from spirillar contamination.

4. Local diseases induced by Relapsing fever and liable to obscure its course. I refer to fever symptomatic of parotitis, tonsillitis, pharyngitis, bronchitis, pneumonia ; hepatic, gastric and splenic irritation, diarrhoea and dysentery.

Local inflammation arising in the course of spirillar infection becomes detectible by its own special signs, and is commonly regarded in its true light as a complication of the specific disease. Yet it fell within my experience at Bombay that a precisely reverse view was taken, and most stress being laid upon the local affection (then Pneumonia) the demonstrated spirillar presence was regarded as an incidental occurrence. It has never been shown that pneumonia or any of the local diseases named above display this blood-contamination, except when there is proof or reasonable ground of surmise that they concur with primary and recurrent attacks of relapsing fever : the evidence superabounding that in ordinary pneumonia, &c. the blood is free of such peculiar sign. Usually

complications arise after the crisis, and then symptomatic fever may simulate a relapse or in its course partially or wholly obscure the real recurrence ; so that the only sure guide to diagnosis of events, is such careful systematic inspection of the blood as will reveal the presence and duration of any spirillar contamination. Experience showed the necessity of particular care here, for several of the above-named complications happening also in cases of 'remittent' fever, would have been referred to that heading had the blood not been duly scrutinised ; or in the absence of valid clinical history, would have been regarded as of spontaneous origin. To the end of the epidemic, new forms and combinations of the pyrexia characterised by spirillum in the blood, continued to present themselves, rendering more obvious than ever the need of microscopic aid.

I have very little doubt that my experience was not singular, and that in the Bombay fever was simply repeated the type of many severe European epidemics ; but it happened that professional opinion became divided, and therefore all collateral phenomena were thoroughly criticised.

b. Non-Febrile Stage.

When patients were admitted during the apyretic intervals of fever, the anomalous symptoms occasionally seen (*Vide* Chapter III.), were to be interpreted by the past history, characters present (visible blood-infection not being available) and future course of the case : diagnosis might have to be deferred.

Collapse at crisis.—Destitute subjects brought in from the streets sometimes presented the aspect of algide cholera, but without the other usual signs, the pulse also being perceptible and urine not suppressed for long ; febrile reaction was seldom of a typhoid character, and promptly rallying the rule ; a relapse might ensue. Upon examination of the blood in collapse large pale granular-cells, or endothelial cells containing fat granules, may be found, and to experienced observers their presence is almost pathognomonic of the spirillar infection. Collapse with jaundice has been mistaken for 'bilious colic.'

Nervous symptoms.—The comatose state ensuing on cerebral hæmorrhage at acme, could not be distinguished from ordinary apoplexy, except by the history ; when defervescence had been of lytic character, I have sometimes found the spirillum persisting till its end.

The excitement occasionally seen at crisis, has been known to simulate alcoholic poisoning (the smell of liquor, however, being absent) ; and also 'datura' poisoning, in extreme dilatation of the pupils and peculiar restless tremors.

Active delirium occurring at this time, could be understood on application of the rules above stated ; it might recur at relapse. The mania and fatuity occasionally known to follow spirillum fever were not peculiar in character, and their true relations were indicated by either early clinical history or a prior attack seen in hospital ; so the instances of perverted or defective sensation in the limbs, and muscular wasting. Rheumatoid pains following specific fever were not (as a rule) attended with local heat, swelling or tenderness, or the rheumatic diathesis ; they were not limited to one tissue or part, and not increased by warmth or at night, but rather upon movement.

Local ailments.—Bronchial congestion, hepatalgia or jaundice, diarrhoea or dysentery were to be recognised by the history of a recent severe febrile attack, either single or repeated at definite interval : a still enlarged or tender spleen, traces of an eruption, unusually feeble heart and yet declining pulse, œdema of the feet, severe body-aches and general debility, being the usual sequelæ of spirillar infection, use could be made of such signs in the interpretation of many cases first seen after cessation of specific fever. It is noteworthy that all might be subsiding and convalescence be apparently at hand, immediately prior to a marked relapse ; whereas if no relapse took place, recovery proceeded to its completion.

Since during fever nutrition of the body fails, the nails become thinner and a narrow transverse depression near their root makes its appearance. After an abortive attack of spirillum fever, one such indentation is to be seen (which is not characteristic) ; but a second becomes visible after the relapse, varying in depth according to the severity of illness, and two or more such cross marks would serve as a late diagnostic sign of the attack. I noted this double mark after my own first illness ; it is not invariably distinct.

CHAPTER XI.

PROGNOSIS.

FORECAST of the probable course and termination of illness being required at all stages of the spirillar disease and in all conditions of the patient, the data needed for judgment embrace the entire clinical history of this fever and the general prognostic principles applicable to all fevers. It is here assumed that general rules of prognosis are borne in mind ; and that Chapters V., VIII. and IX. of this Section have been already perused ; see also that on the Blood, below.

Special features of Relapsing fever.—The pronounced character of the pyrexial attacks and their tendency to recur, at first sight augur unfavourably ; yet on the other hand the fever is of short duration, diminishes on repetition, and is mitigated by intercurrent periods of quasi-normal repose. Hence risk to life arises from certain complications, rather than from pyrexia alone : and as there is no foretelling such untoward events (which may be said to be always imminent), prognosis in relapsing fever becomes unusually uncertain. The difficulty is to understand why the great majority of patients suffer so little, as much as why a small proportion ever dies (Chapter VIII.)

Procedure.—The first object is to ascertain the precise stage of illness—*e.g.* whether that of invasion or relapse, or if corresponding to first or second apyretic interval ; and next to compare the symptoms present with those seen both ordinarily and exceptionally, and those indicative of local complications known to happen at the date ascertained.

General Prognosis. As regards the disease. — From a common source of infection results may vary, and that inexplicably if not from personal idiosyncrasy. Pyrexial stages of the fever.—The greatest risk to life attends Invasion, the First Relapse may also be severe and death in the first Interval is by no means rare. Premonitory symptoms and initiation commonly furnish no clear prognostics, nor the first few days of illness ; after the 4th or 5th day the Acme and Crisis becoming always possible, attention is needed, as then exacerbation of symptoms, fatal accidents and serious complications are likely to arise: both prolonged duration of fever and its gradual decline are unfavourable.

The Critical Fall.—If no improvement take place at assumed date of crisis and fever persist, some complication may be suspected ; if after the fall the liver and spleen continue enlarged and tender, there is also risk of pneumonia or dysentery. Crisis may be followed immediately

by sharp rebound, and death in a few hours ; prolonged depression at its end was rarely of serious import, but active delirium may temporarily ensue.

Apyretic stages.—Shortening and interruption of post-critical deferescence indicate risk from complication ; thus, some patients suffered severely from secondary fever and some died from pneumonia, now appearing. Deferred rallying is less serious, but nervous symptoms or dysentery may then supervene. An Invasion-attack may be free from risk, and the ensuing non-febrile period uninterrupted, and yet the Relapse prove fatal ; or the two febrile attacks may leave the patient unscathed, whilst soon after danger and death supervene : pneumonia may ensue even after some weeks.

As regards the Patient.—Famine-stricken, weak or emaciated subjects suffered most, yet not invariably or in proportion to their debility ; amongst the intemperate, aged and infirm, and those previously ill, prognosis was generally unfavourable ; the plethoric ran unusual risk, and even robust subjects died. Amongst cachexiæ, the scorbutic seemed to predispose to pneumonia and dysentery, the malarious and anæmic to cardiac failure ; the syphilitic and leprosy subjects did not suffer most. The influence of fatigue, exposure and neglect of early treatment seemed determined on general principles, and so that of pre-existing heart and kidney disease. No woman aborting died, or was in especial danger.

Special prognosis.—I did not perceive that any symptom of relapsing fever had an absolute significance in itself ; commonly, the particular signs are to be estimated as in typhus : marked irregularity of symptoms usually concurrent at the acuter stages of fever, is indicative of risk. Unfavourable during fever were early typhus tendency, a dusky, haggard aspect, early petechial eruption, diarrhœa, cough, prompt debility, drowsiness and onset of the 'typhoid state.' A tendency to this state coming on late was not necessarily of bad omen, except with pneumonia, dysentery and other post-critical complication ; or when much pronounced. Involuntary evacuation of the excretions and retention of urine, though bad signs, were least so at critical fall, when the prostration implied might be temporary only. I was much impressed with the fact that deep jaundice and a copious petechial eruption, were not inconsistent with ordinary course of relapsing illness and fair recovery. Temperature—on comparing selected data, I found prognostic indications were not to be drawn from means of temperature alone whether high or low, at any stage of illness¹ ; many fatal cases were attended with moderate pyrexia and, generally, their means were rather below those of survivors. Irregularities in course and a prolonged duration of fever, with unusual chills or sweats, implied risk ; and so an irregular

¹ In some more recent observations Dr. Motschutkowsky of Odessa has stated that by comparing temperature-charts of fever cases, it is possible to predict with tolerable accuracy the course of illness : thus, if the temperature rises after crisis of invasion 1° or 2° C. then a relapse will follow ; whereas if after the main crisis, it subsides or remains low, a relapse will not ensue : the influence of bronchitis and other complications must be allowed for. On these data, the author correctly foretold results in 139 out of 194 cases ; being wrong only 28 times. For some similar inferences made at Bombay, see pp. 134-5-6 ; final opinion being expressed in the text. From consideration of the pulse, state of liver, spleen and excreta, and aspect of the blood, no valid aid to prediction was detected at Odessa. ('Deutsch. Archiv. f. Klin. Med.,' Nov. 1881.)

distribution of body-heat, when occurring early or independently of crisis. With such variations, the general symptoms were usually severe. Lysis—this irregular form of defervescence was so commonly attended with signs like those of true *typhus*, that suspicion of a positive mingling of 'continued' fever was almost inevitable, although the blood-spirillum sometimes still persisted : lysis at invasion has been followed by death at relapse. Absence of crisis after the 7th or 8th day of Invasion should excite apprehension, whether or not local complication be present ; and hardly less serious is premature gradual subsidence of specific pyrexia, as the general symptoms will of themselves commonly indicate. I have known an intermitting initiation of specific fever to be followed by ill-sustained pyrexia, and death at the acmé : the general symptoms here, too, were unfavourable.

Pulse.—Whilst mere frequency is free of unfavourable import (especially in the young), a rapidity altogether disproportionate to temperature, or quickness persisting at and after crisis, always excited fear of accident or complication. A failing pulse was sometimes due to intensity of fever, or in the aged weakness ; a slow pulse during lysis, pneumonia attending, was noted in some fatal cases ; intermittency at crisis, or some dirotism, was not necessarily of bad import.

Heart.—The typhus-like character was practically invariable, yet *post mortem* much structural degeneration was comparatively rarely seen : irregularity of action seemed more noteworthy in prognosis.

Lungs.—Quickened breathing is less than usually significant of harm, but persisting after crisis may indicate local lesion ; absence of cough is no guide, if pulmonary apoplexy have occurred ; scanty hæmoptysis has seemed mainly to indicate lung injury. The tendency to pneumonia at and after crisis should be borne in mind ; inflammation then being a common cause of death.

Digestive system.—From the aspect of the tongue few prognostic inferences could be drawn ; a moist, if coated, tongue has persisted till the acmé and until death : or on the other hand a dry, brown, shrunken tongue amongst survivors. Tremulous movements were very rarely noted ; their augury would be unfavourable here. Tympanites was an unfavourable condition, when accompanying other signs of abdominal implication ; also the opposite state of retraction. Hiccup though common in severe cases, was not of itself a bad sign. Irregularity of the bowels and foetid stools, with or without griping and tenesmus, indicated at the acmé probable enteric inflammation : a dysenteric tendency was also observable and might be early manifest at the fall ; both conditions imply risk.

Spleen.—From the state of this organ no sure prognostic inferences were possible, at Bombay : I was not able to detect during life splenic infarcts found after death, and have supposed them to exist when not afterwards so found. Acute inflammation and suppuration of the spleen were practically absent at Bombay.

Liver.—An excessive implication here seemed of more unfavourable import than much splenic fulness and tenderness, particularly in young subjects : yet appearances are deceptive, and patients often recovered with wonderful facility. In general, the pronounced signs attributable to liver and spleen and the intervening stomach, were at acmé of fever so

much alike in both survivors and those dying, that I hesitate to insist upon their prognostic indications taken alone.

Urine.—Few inferences were made from ordinary examinations of the urine ; and in the absence of uræmic symptoms or visible changes in this excretion, there seemed no need of elaborate analysis : the urine often appeared healthy just before the death at acme, but in consideration of some current opinions, I am bound to add that the amount of urea excreted was not then ascertained.

Nervous system.—Severe headache or pains, or deafness, had no unusual import. Delirium always claimed attention: at the acme it was not necessarily of bad augury ; it may be absent at the fall when cerebral hæmorrhage had occurred or was near at hand. Post-critical delirium may aid in the production of fatal, if delayed, exhaustion ; but it happened that no patient died with or from the delirium of inanition. Extreme contraction of the pupils was doubtless a bad sign ; and a pin-hole aspect was once noted in a fatal typhus-like case. I have known a contracted state precede cerebral hæmorrhage and inflammation. In general, however, the appearance of the pupils did not assist prognosis.

Epistaxis at the acme, may concur with mitigation of headache and of delirium ; yet the same results ensue from decline of fever alone: copious epistaxis at the fall has been followed by fatal thrombosis. Convulsions, local spasms and rigidities, with other marks of cerebral irritation, were rare and in my experience transitory ; cerebral hæmorrhage was not apparently preceded by such warnings, when occurring at the acme of fever.

Typhus biliosus.—Spirillum fever attended by deep jaundice, early prostration and acute abdominal signs, with low and irregular temperature and a tendency to lytic defervescence, was oftener fatal than not. The instances were infrequent at Bombay, and I did not perceive that jaundice added to the risk ; for in the more numerous like cases unattended by marked jaundice, prognosis was hardly less unfavourable. At all times, general rules here apply.

CHAPTER XII.

TREATMENT OF SPIRILLUM FEVER.

THIS concerns either the disease in general, or the individual sick ; and is, therefore, either Prophylactic or Therapeutic.

1. **Prophylactic** measures have for their object both prevention of the disease and arrest of its further spread ; and they consist in the employment of certain rules of Sanitation. A main feature of Relapsing fever, has ever been its practical limitation to the more indigent classes ; and I think since its late manifestations, especially abroad, in the form of scattered and recurring epidemics, the opinion becomes admissible that its essential relations are with poverty as much as famine. Hitherto, strictly preventive measures have not been widely adopted. Probably severe famines will not again desolate large areas in Europe ; and in India, with increasing facilities of communication by means of new railroads and highways, the prompt transmission of food to starving people will become more facile : public charity is also universally more active. Other nearer preventive measures would be the discouragement of vagabondage in all its forms, the rigid sanitary inspection of town-quarters where tramps congregate, and the systematic prompt removal of sick individuals or families to hospitals set apart for their accommodation. At the same time the more crowded houses, lanes and streets, should be so improved, that the now dense and indiscriminate herding of the lower orders become less notorious. Baths, wash-houses and all domestic proprieties should be encouraged ; and the people be induced to observe adequate home and personal hygiene. Open Refuges for the homeless and common lodging-houses are known to become fresh *foci* of disease ; and until all strangers entering them are early submitted to medical inspection, the introduction of relapsing fever will not be prevented. It seems to me probable in the highest degree, that sporadic cases of this disease are commonly present in great towns like London, Berlin and Bombay : such cases, at least, have a free access thither, and from the highly contagious properties of the spirillar infection, the occurrence of an outbreak remains a matter of chance. What are the precise conditions leading to an epidemic, is not always apparent ; and at present, the essence of effective preventive measures may be said to consist in a valid opposition to the spread of infection. Once begun, the course of an epidemic of relapsing fever is not to be accurately foretold.

Accommodation of Sick.—The disease cannot be efficiently treated in the homes of patients ; and were this so, still its spread not being

obviated, removal of the sufferer becomes necessary. Patients may be received into general hospitals, or isolated from other sick in special buildings; and the advantages of each plan have been differently estimated. According to some authorities, distribution of the sick in the large wards of general hospitals would, in the instance of an ordinary epidemic, sufficiently meet requirements. Thus Dr. Lebert (*loc. cit.* p. 284) remarks: 'I do not regard it as necessary to force all . . . sick of the poorer classes into an isolated hospital; indeed I hold it entirely unnecessary even to cause the cases of relapsing fever to lie exclusively in separate apartments. In all epidemics of this fever which have occurred here (*i.e.* at Breslau), I have had many cases of the disease in my general wards, where by excellent ventilation and by scattering the patients, I have kept the proportion of cases of contagion at the minimum compared with that occurring in relapsing-fever wards.' On the other hand, it has been urged that it is better not to expose those ill with other diseases to the risk of fever-infection; and that, in point of fact, by suitably isolating the fever-sick in separate buildings neither is their own death-rate increased nor are the ward attendants exposed to greater risk. Thus, Dr. Litten (*loc. cit.* p. 315-16) furnishes statistical details supporting this opinion, which though not numerous are strictly appropriate here; since they refer to the same epidemic at Breslau as that to which the late Dr. Lebert's remarks chiefly applied. In all comparisons of this kind it is essential to deal with the contemporary experiences, for it is well known that different outbreaks of Relapsing fever display amongst themselves much diversity of death-rate and infectivity. By common consent, open asylums and low lodging-houses should be temporarily closed on the appearance of an epidemic. It will be obvious that sanitary proceedings must also be regulated according to the severity and progress of a local epidemic, and that prevention of further disease is the chief object in view. So far as I learn, it is now the usual practice of town and rural authorities to provide distinct accommodation for relapsing fever cases, in the event of an outbreak of this disease; and it has been properly recommended that the sick should be arranged according to the stage of fever they are admitted at. The other necessary details as to ample ventilation and especially room-space for each patient (for minimising contact with other persons); also to restrict visits of the outside population, need only to be alluded to.

Particular stress has been laid on the advantage of evacuating infected houses, and thoroughly disinfecting and cleansing the separate rooms; also upon the necessity of disinfecting with hot air and other means, the bedding and clothing of patients, destroying all the least valuable articles likely to act as 'fomites' of contagion. It has been recommended that the excreta be specially dealt with. Medical men and all individuals exposed to infection should, on these views, seek to avoid the risk of conveying infection in their own persons.

Experience at Bombay was useful chiefly by way of warning. It is true that at the height of the late epidemic a Camp of Refuge for fever-patients was established, yet inasmuch as the friends and companions of the sick were freely allowed to mingle with them, disease continued to spread in the Camp itself during the time it was kept open. That a city

with a population of nearly three-quarters of a million and an enormous death-rate from ill-defined 'fevers,' should be still unprovided with a permanent fever-hospital, is an anomaly which must ere long attract public attention. In the larger Native General Hospital, the medical wards are few, ill-constructed and faultily arranged, and they were often overcrowded with sick; the consequence being numerous instances of local contagion, with a considerable mortality therefrom (see Section III., Chapter II.) Even in the smaller, better-built hospital, there were several examples of communicated disease.

Briefly, although Relapsing fever is not so fatal a disease, and possibly not so highly communicable, as typhus and small-pox; yet for its prevention and abatement, sanitary measures are on all grounds none the less needful.

Principles of humanity, as well as of public safety, require that adequate prophylactic provision be made; and costly as this may be when disease is widespread and severe, yet most communities are beginning to recognise the true economy of valid sanitary preventive measures, early applied.

2. Therapeutic Treatment.—As heretofore, universal experience continues to prove the inefficacy of drugs to either shorten the specific illness or check its recurrence. It is fortunate that the disease has naturally a low death-rate, for to the best of my knowledge the severer cases are not amenable to medical treatment; and, in general, it may be said that relapsing fever is less under control than either typhus or enteric, being also certainly more intractable than malarious fevers.

Specific Therapeutics.—An attack conferring no immunity against re-infection at even early date (*e.g.* two or three weeks in the Monkey, and probably not much longer in Man, the minimum period in either case not yet being ascertained), there is but little promise of help by artificially anticipating infection.

Anti-parasitic treatment.—'To neutralise the poison' has long been a practical aim of physicians, and on the supposition that the blood-spirillum of relapsing fever represents its noxious principle, a not irrational hope might be entertained that this aim is within the bounds of attainment. Already it has been ascertained by direct experiment on fresh blood, that several reagents applied in weak solution promptly induce cessation of movement and apparent death of the parasite; the inference following that if such reagents could be introduced into the living blood in a similar state and amount, the same results would follow with probable arrest of infection and illness. The intricate conditions here intervening are sufficiently obvious upon the shortest reflection, and it will be sufficient to state that as yet there are no precise data warranting more than the hope I have just alluded to. I once found that weak neutral solutions of Quinine seemed to kill the spirillum; Dr. Litten ascertained that a 1 per cent. solution of Carbolic acid quickly arrested the parasitic movements, and that even $\frac{1}{20}$ p. c. solution of Iodine had a similar effect, so a solution of the Permanganate of Potash, a 1 p. c. solution of Nitric and Sulphuric acids, of Caustic Soda and Potash, so Glycerine; not so weak solutions of common Salt, as

Dr. Heydenreich and I, too, have found. Other reagents might be named, but it will suffice to add that Chloroform and absolute Alcohol act promptly in a similar manner; and according to Dr. Riess ('Deutsch. Med. Wochensch.' Dec. 1879) Salicylate of Soda in $\frac{1}{10}$ p. c. solution sometimes acts in like way. In practice, however, the blood-spirillum and the febrile symptoms remain unaffected after Quinine given largely to cinchonism, after narcotism by Chloral and the freest exhibition of Spirituous liquors; also after the administration of the Carbolates, and very large doses of the Salicylates. Hence, as yet, there is no evidence that the drugs named when administered variously and in doses consistent with safety, possess a manifest parasitidal efficacy in Relapsing fever. Dr. B. Oks of Odessa thinks that Calomel has a specific counter-influence. ('Deut. Arch. f. Klin. Med.' Nov. 1881.)

Anti-periodic Treatment.—Owing to the fact that under the simplest treatment the spirillar attacks by no means invariably recur and that a relapse may spontaneously be reduced to a very mild manifestation of illness, some caution is needed in estimating the effects of special remedies. Thus, the non-appearance of a relapse would not warrant the inference that a drug had checked its advent, unless in a long succession of trials the same result should follow; but, on the other hand, a few or even single instances of relapse during treatment would suffice to prove the inefficacy of the drug. Berberine, Arsenic, Benzoate of Soda, Salicylic acid and its salts, and Quinine have thus proved to be inefficacious as anti-periodics in relapsing fever. Perhaps these negative results were likely from what was stated above; but however viewed, there is here a complete uniformity of opinion amongst European practitioners and those of Bombay. Interesting details have been published and some were acquired in India, showing that large doses of Quinine and Sodium salicylate given persistently at short intervals throughout the entire first apyretic period of relapsing fever, have entirely failed to prevent or even mitigate the First relapse; and this on many repeated trials.

Anti-pyretic Treatment.—Attempts to cut short the first or Invasion attack of fever are here most worthy of attention, for the Relapses are so variable naturally that inference from their course would be somewhat difficult to substantiate. Anti-pyretics oftenest tried have been Digitalis, Quinine, the Salicylates and Cold baths. Alcohol might also be named. So far as I perceive, Digitalis in repeated moderate doses (*e.g.* $\text{m xxx.}-\text{xl.}$ of the Tincture) had no influence on the specific pyrexia; sponging with cold water and cold baths often repeated at short intervals, have always had but a temporary effect; so the influence of Quinine, either alone or in combination with other means. It seems that the spirillar pyrexia is particularly intractable, but it is needful to remark that it is subject even during Invasion to marked remissions, and, particularly, that its natural duration is comparatively limited; hence the need of a caution in Inference, which has not always been observed. After the 5th day of Invasion and almost any day after the 1st of Relapse, crisis may of itself supervene whether or not anti-pyretics are being employed; and the plainest cessation of fever may be wholly spontaneous.

Some years since Dr. Griesinger remarked that as either anti-periodic or anti-pyretic, Quinine had probably not been thoroughly tried; at the

present day, this cannot be said. The same excellent authority was of opinion (*loc. cit.* p. 360) that in *typhus biliosus*, it is 'certain that under the action of Quinine the most serious cases change their character promptly, and the spleen goes down': such has not been my own experience, and I cannot but suppose that the cases thus ameliorated belonged to the 'remittent' fever series of malarious character, and were not of the characteristic spirillar type.

Respecting the influence of Sodium Salicylate, it is now ascertained that by its means the specific pyrexia may be decidedly reduced. Thus, Dr. S. Unterberger ('Jahrb. f. Kinderheilk,' v. x. 1876) has seen the temperature brought down 3° C. (5°·4 F.), yet the attack was not apparently cut short, or splenic enlargement prevented, or the active blood-spirillum visibly affected: and Dr. L. Riess (*loc. cit.*), after essay on 26 cases thinks that it is possible to cut short or mitigate the symptoms (especially the temperature) of specific Relapses by very large doses (100 grains or more daily), noting however that even when the heat is reduced to normal or below it, the spirillum still persists. Such doses are said to be well borne, only a buzzing in the ears being complained of; once by oversight 30 grammes (more than 450 grains) were given at once, and no worse result followed. In one of Dr. Riess's cases the relapse set in severely, and the patient died with cerebral hæmorrhage; but in general there was no reason to suppose that the salicylate did harm. The least disadvantages of such preventive treatment are impairment of appetite (a serious matter in relapsing fever), and the need of tedious repetition of doses; there are doubtless others, and from recorded data I confess to a doubt if the benefit attained be a substantial one.

Treatment directed towards the Spleen, as an organ especially implicated by the spirillar infection, has not yet proved encouraging: thus, it is known that the tumefied spleen may be decidedly reduced in volume by means of electricity (the induced current) and cold applications, without obvious beneficial consequences; and this might be anticipated, since the splenic implication is not a cause but rather a result of infection. Tincture of the *Eucalyptus globulus* has failed to reduce the splenic enlargement, or to mitigate fever or prevent relapse. Dr. N. Friedreich of Heidelberg ('Deutsch. Archiv. f. Klin. Med.' v. xxv. 1880) has indeed made the suggestion that specific fever might be truly checked, if individuals exposed to contagion and known by clinical scrutiny to present the incipient splenic intumescence of Incubation-period, were placed under improved hygienic conditions. The data supporting this abstractedly good idea appear to me defective, and in India at least they might not have much validity.

Non-specific Treatment.—It being impossible to strike at the root of an infection once implanted in the body or effectually to check its development, the physician will aim to forward the tendency to recovery to mitigate urgent suffering and to obviate known modes of death. There may be less to do than in ordinary typhus, but there is still ample room for the exercise of practical knowledge and of much sagacity, because the specific fever is very apt to recur, is liable to peculiar complications, and displays in different epidemics a highly varying proportion

of mild and severe attacks. Though very acute, the successive illnesses are comparatively brief and well-defined ; hence if the patient with occasional help and without undue interference can be safely conducted through them, his recovery becomes tolerably assured. It must be remembered that in relapsing fever, the cessation of a specific febrile attack does not constitute convalescence ; for serious reactive phenomena are still possible, and there is no real convalescence until the end of all infective manifestations. The symptoms, too, commonly preserve a certain order of natural sequence ; and the non-recollection of this fact may lead to a therapeutic complaisance which would be, at the least, premature : *e.g.* drugs administered shortly before the periodic crises of fever, are not to be credited with the production of defervescence. Daily use of the clinical Thermometer is throughout an essential guide ; and although at present no treatment specially adapted to the spirillar pyrexia is known, yet in complicated cases it is often desirable to ascertain when specific fever appears or has ceased, and failing a correct reckoning of the attacks the aid of microscopical blood-scrutiny then becomes necessary.

Treatment of Common Symptoms.—An illness at first disregarded or neglected is doubtless likely to become aggravated, especially in debilitated subjects ; and always particular attention is required, as the culminating period of fever approaches. The early exhibition of emetics cannot be recommended ; a mild aperient is usually indicated : then follow the simple remedies grateful to fever-patients, rest, free ventilation and a liberal supply of nutritive liquid food. The mineral acids (Nitric or Phosphoric) and cooled drinks. As a high temperature is less significant here than in other fevers, sponging with cold water, packing in wet sheets or the moderately tepid bath will at first suffice ; the anti-pyretic treatment may be essayed, but I know of no means that will by anticipating check the final acme. Warburg's Tincture has signally failed and possibly done harm, epistaxis following its administration. At Bombay, it was noteworthy how soon medical officers began the use of stimulants : alcohol in moderate doses, ammonia, the ethers and carminative tinctures. Blood-letting was wholly contra-indicated.

As acme approaches, Quinine by mouth or subcutaneously, the Salicylates, or especially the cold bath, sponging with iced water, the ice-cap, and cold drinks or sucking of ice : assiduous watching for the initiation of crisis.

When the temperature begins to decline and sweats appear, the body to be kept dry, warm nourishing drinks, diffusible stimulants and warm applications, as may be needed. Critical collapse is met by artificial heat, hot drinks, the subcutaneous injection of Ether ; alcohol alone seems of doubtful utility.

Throughout, no indication was more clear than the necessity of obviating the tendency to failure of the heart's action : hence, besides the early use of wine and finally spirits, Digitalis, Belladonna and Aconite were exhibited, and, with a double purpose, Nux Vomica or Strychnia : doubtless with benefit.

Headache was treated with laxatives and cold applications ; the pains with fomentation and sedative embrocations ; sleeplessness, from

combined causes, with Chloral and Bromide of Potassium, usually combined, in preference to opiates.

Constipation was rarely obstinate, and cathartics were neither needed nor desirable ; enemata of castor oil being preferable.

Acuter special symptoms.—Delirium : during fever Chloral and the Bromide usually sufficed to quiet the patient at night ; at acme of fever cold to the head, after in the worst cases removal of the hair ; if the patient remain excited, his transfer to a quiet room, with as little restraint as practicable. Stimulants and the frequent exhibition of food may be even then desirable : they become essential in the post-critical form of delirium, which may be the more violent and even persistent. The hypodermic injection of Morphia was frequently practised with benefit, as the only available means of obviating extreme exhaustion ; conium, hemp, or camphor were seldom resorted to.

Excessive tenderness of Liver and Spleen was best relieved by fomentation and poultice, cold application seldom being grateful to the patient ; localised pain by mild counter-irritants or sedative lotion ; after an aperient. Local depletion never seemed called for.

Vomiting and hiccup were sometimes obstinate and persisted after the crisis ; effervescent, ice, sedatives or finally local counter-irritation were variously used.

The state of the kidneys did not attract particular attention during fever, the urine never seeming greatly altered or uræmic symptoms indicated. Salines formed part of the febrile mixtures, but nitre did not maintain a place as being of special utility : considering the known state of the renal secreting structures, I should have hesitated to employ diuretics. Nor in their place the hydragogue cathartics which have been recommended, or vapour-baths. Possibly experience was unusual at Bombay. With partial suppression of urine at crisis, hot applications and dry cupping to the loins were doubtless beneficial.

Jaundice did not call for special treatment ; and mercury was never given.

During the Apyretic Intervals of specific pyrexia, the object was to restore strength and obviate accidents. Fresh air, very moderate exercise, care in diet ; tonic medicines. It never seemed that relapses were due to indiscretion of diet.

Complications.—Local accidents and inflammations being rarely attributable to external causes, their prevention is to be attempted on general principles. Cerebral hæmorrhage seemed always spontaneous, and neither it nor hæmatemesis was seen in the plethoric or robust. Coma was ineffectually dealt with by counter-irritation, enemata and stimulation of the kidneys ; ergotine has been injected subcutaneously. Black vomit was fatal, though similarly opposed.

Bronchitis and slighter pulmonic congestion were met by warm and stimulating applications to the chest, and the addition of Ipecac or Squills to stimulant mixtures. For pneumonia, the free exhibition of alcohol, besides ; antimony, alkalies and diuretics seeming of subordinate need. Support by feeding was always a main indication. Pleurisy might require opiates, but rarely diuretics ; calomel was not given.

Diarrhœa and dysentery were treated by local applications, enemata, astringents and opiates, so far as possible ; large doses of Ipecac not

seeming desirable, and even laxatives not being well borne : these complaints often spontaneously subsided.

The local treatment of pharyngitis and parotitis was subordinate to strengthening of the system ; stimulating gargles, with the permanganate of potash, and poultices with early incision : as in most complications, the Chlorate of Potash with Bark was generally administered.

Epistaxis might require plugging of the nares, but usually ceased on the local application of cold.

Splenic abscess and peritonitis were not met with at Bombay, nor acute œdema of the larynx (which has been known to need laryngotomy).

The more sequelar complications pertaining to the nervous system were dealt with on common principles. Acute otitis in children has, in Russia, been relieved by puncture of the *membrana tympani*. Ophthalmitis needed protection of the eye, atropia, counter-irritants, opiates and tonics.

Anæmia following spirillum fever was not very prominent, though preparations of Iron were generally exhibited, in combination with the nervine tonics Quinine, Phosphoric acid and Strychnia.

Local paresis was met by stimulating applications and electricity (the induced current), as well. Local hyperæsthesia by soothing applications, and aconite was found beneficial. The hypodermic injection of a Strychnia salt has been recommended.

Fresh and pure air, and good diet, with repose, are especially desirable after this blood-infection.

SECTION III.

PATHOLOGY OF SPIRILLUM FEVER.

UNDER this heading are included the nature and causes of the specific disease. Of primary interest is the visible State of the Blood. Then follows the evidence bearing on Contagion, as the main if not sole etiological agency. And, lastly, I shall consider the disease as a whole, and attempt to connect its chief Phenomena, both Clinical and Epidemiological.

CHAPTER I.

ASPECTS OF THE BLOOD, GENERAL AND SPECIFIC.

METHODS OF EXAMINATION. GENERAL DESCRIPTION OF BLOOD: NORMAL ASPECT, APPEARANCES ACCORDING TO STAGE OF DISEASE. DETAILED DESCRIPTION OF BLOOD-ELEMENTS ACCORDING TO STAGE OF DISEASE. ACCOUNT OF THE SPIRILLUM.

SEVERAL striking changes are to be seen in the blood of relapsing-fever patients, and doubtless others remain for detection with improved methods of scrutiny. These changes refer partly to the leucocytes or white blood-cells, and partly to superadded elements which are derived either from the body itself or *ab extra*; and of the truly extraneous ingredients, the most remarkable is a motile filament known as the *Spirillum*, or *Spirochæte*. Although many years past the blood had been carefully examined, no alteration was noted beyond an increase of white cells; and even during 1867-68, Dr. Obermeier, after a special investigation of 82 fever cases did not arrive at more definite conclusions; or at least did not detect the organism, which five years later (when a fresh fever-epidemic occurred at Berlin, 1872-73) he at once saw upon renewed search. This circumstance shows that a skilled microscopist definitely seeking for evidence of visible blood-contamination, may not immediately succeed in finding such evidence; even when to a moral certainty, it is both present and ascertainable. Dr. Obermeier's observations have been abundantly and invariably confirmed on the continent of Europe, and also at Bombay. The main results of my own enquiries are described below, and they have been found to accord with those of other observers.

Methods of Blood-examination.—It is necessary to employ magnifying powers of not less than 500 diameters, and for special investigation

the higher power immersion-lenses ; good illumination is needed and often the achromatic condenser. All glass and instruments used should be scrupulously clean ; and the reagents kept free from incidental impurities.

1. *Fresh blood*.—It is always desirable to examine the fresh blood by daylight. This was usually taken from the washed finger of the patient, by pricking with a needle and after rejecting the first exuded blood, placing a minute drop on the thin glass cover, which is then inverted and put on the slide for examination. All may be done so quickly as to ensure that no substantial blood-change takes place in the transfer ; scrutiny should be made with care, and it is sometimes useful to repeat the examination after an hour or two, when coagulation has taken place. In case of doubt, more than one specimen is to be prepared : such specimens will often remain moist in the centre for a day or more.

2. *Dried blood*.—For preservation dried specimens are needed. A very thin layer of fresh blood is evenly spread with the needle over the glass cover, and allowed to dry under protection from dust (I usually inverted the cover), and free from chance of rubbing. Useful preparations may be made according to the first method described.

a. The thin film of fresh blood is to be promptly exposed to the fumes of a weak solution of Osmic acid ; this is done by inverting the thin glass cover and placing it over the mouth of a phial containing a 2 per cent. solution of the acid, for three or four minutes. The colour of the blood changes to brown, but very little distortion takes place in its structural elements, which thus become permanently preserved in their original condition. No further treatment is needed for these specimens.

b. To the thoroughly dried blood a drop of Glacial Acetic acid (the British Pharmacopœia acid requires a longer time to act) is added, allowed to remain for about a minute and then to trickle off, a drop or two of diluted water following to gently wash away all dissolved parts. The preparation is now once more dried, and there should remain only a barely perceptible, colourless film, which on examination will show simply the nuclei of white cells and the spirilla clear and distinct. If there is much opalescence, acid must be again applied ; care being taken not to remove all traces of the blood-layer. Such preparations cemented with wax at the edges are permanent ; and on exchanging them, Dr. R. Albrecht and myself in 1878 were able to ascertain that the blood-spirillum of St. Petersburg and Bombay is an identical organism. This method originated with Dr. Albrecht ('St. Petersburger Medicin. Wochenschrift,' May 1878).

c. The dried layer of blood (thin as possible) may be stained by a clear aqueous solution of Methylviolet or Anilinbrown or Fuchsin ; after 2 or 3 minutes, according to strength of solutions, water is added to wash away superfluous colour and the preparation again dried. It then shows deep staining of the red and white blood-cells, and of the spirillar filaments ; the plasma in good preparations remaining colourless. A recent improvement of this method consists in heating (or baking) the blood on the glass cover at a temperature of about 120° C. (250° F.), by leaving it for a minute or two on a metal plate made hot by a lamp ;

thus the albumen becoming insoluble, the blood-plasma is less likely to be discoloured and the stained parts are more distinct : I have seen this plan applied only to blood containing ordinary bacteria, but it seems well adapted for spirillar blood. Preparations may be preserved dry (as most of my own were) ; commonly they are mounted in Canada balsam or glycerine. Acetic acid specimens may be similarly stained, and these show very well.

Both the last-named methods are adapted for clinical demonstration, and by a good $\frac{1}{4}$ -in. object glass magnifying not less than 300 diameters, the spirilla are clearly shown. Specimens of blood may be taken night or day, at the bedside and prepared at leisure ; and so a series made, as was often done at the G. T. Hospital. With practice many minor details will be learnt, which I need not describe ; and it will be sufficient encouragement for others to know, that by the use of dried preparations difficulties of diagnosis in doubtful cases were frequently cleared away, and the true character ascertained of latent or obscured specific relapses. It may be needful to examine more than one specimen. The disadvantage of dried specimens is the probability of incidental alterations of structure, with concurrent obscuring or destructive changes of the more delicate blood-constituents ; hence the fresh blood should also be inspected, if possible.

3. *Sections of the Tissues.*—These concern special research, are more difficult to prepare and for examination require the best microscopic apparatus. Small pieces of organs in the freshest possible state (spleen, liver, kidney, brain, by preference) are placed in absolute alcohol (which should be renewed two or three times), to become hardened ; after a fortnight or more very thin sections are made, which are to be sufficiently stained, washed with distilled water, soaked in alcohol (to remove the water), transferred to oil of cloves (which renders them translucent), and finally to Canada balsam. No metal but platinum should come into contact with the sections. After staining, the sections may be first treated with very dilute acetic acid or a weak solution of Potassium carbonate (which abstract the tint from animal tissues, leaving only the bacteria coloured), before soaking in alcohol as before. A large series of preparations should be made, as it is only by chance the spirillum is exposed at full length. Dr. Koch *loc. cit.* has photographed the organism whilst lying within the capillaries.

My observations on the blood at Bombay were carried on throughout the year ; mean temperature of air 80° F. with narrow range, the influence of this warmth being shown in the frequently active amoeboid movements of the leucocytes in fresh preparations : the blood was generally taken morning and evening, before the chief meals of the day ; and in particular cases, at different stages of illness or several times in the twenty-four hours.

Objects noted. Blood-plasma—entered as being clear, cloudy or granular : fibrillation—as scanty or abundant ; free protoplasmic masses—their number, size, movement, vacuoles ; ordinary leucocytes—their comparative number ; red discs—arrangement and form : these, with occasional free granules, may be regarded as constant blood-constituents. Less constant were large granular cells—their number and

contents ; large nucleated cells (simple or multiple)—their number ; free active particles, rods or chains, curved filaments, colloidal crystals—their aspect and numbers ; and lastly the blood-spirillum in its varied abundance, form and dimensions : these are the abnormal blood-elements visible on different occasions, which were regularly noted. Changes in the normal constituents are not peculiar to spirillum fever, nor probably are most of the abnormal elements ; it is well, however, to note their occurrence, for some structures (*e.g.* the large granule-cells and fatty endothelium) are rarely seen except in association with other blood-changes and with general symptoms, distinctly indicating the presence of spirillar infection. The significance of the small protoplasmic masses, free granules and short filaments above-named is as yet doubtful ; but the spirillum itself must be regarded as pathognomonic of relapsing fever.

Atmospheric impurities, including fungus-spores and fibrils, possessing characters of their own, could not be confounded with any of the above ; but I occasionally met with filaments in even quickly-dried blood, which at first sight were puzzling from their partial resemblance to spirilla : such were commonest in acetic acid preparations, and might there be due to the acid having become weakened by evaporation and imperfectly dissolving protein-compounds, others occurring in the stained preparations were of uncertain import. See Fig. X. Plate II.

PART I.—GENERAL DESCRIPTION OF THE BLOOD.

Healthy Blood.—In human adults fibrillation may be slow or quick, and the fibrils of highly diverse aspect ; plasma clear or clouded in various degrees ; protoplasmic masses are not common and may be invisible or several moving masses may be seen ; free granules none or few, pale, active or quiescent, with short rods as noted in febrile blood ; the leucocytes differ in proportion, but at Bombay were seldom more numerous than is usually estimated, amœboid movements commonly active ; the red discs sometimes varying in size, and usually falling into short piles. In healthy monkeys there was identically noted slow or indistinct fibrillation, clear plasma, scanty protoplasm, free granules none or quiescent in rows (as sometimes seen in fever), leucocytes several, red discs of varying dimensions and piled or dispersed. Except the amœboid movements and occasional presence of moving granules, all was quiescent in these fresh preparations ; and nothing peculiar was elicited in dried specimens. The degree and causes of quasi-normal variations, are wide enough to inculcate much caution in making inferences of disease from microscopic examination of the blood ; and in the majority of ordinary illnesses departures from these normal aspects are never uniform, and may be remarkably slight in even severe cases.

1. General Aspect of the Blood during Spirillar Infection.—Appearances differ at different stages of the disease : I will begin with the **Non-febrile** taken in order of occurrence.

a. First Incubation-stage : early part.—In no one particular did the

blood-aspect change beyond normal limits, until the spirillum appeared ; and as then no other constituent appeared to be altered, the impression was inevitable of this organism being truly parasitic in its origin. Examples from man being unattainable, I quote that in 10 monkeys examined at this early stage after inoculation, the blood was usually described as 'normal': once I noted, with no elevation of temperature, the morning blood of second day of experiment very clear, some delicate protoplasmic masses inclosing granules like *micrococci*, and the same evening some rounded granule-cells also containing such granules (quiescent) ; next day, larger masses like those described by Dr. Osler in healthy blood, and active free particles probably derived from them. The Albrecht process elicited small filaments (? fungoid), not unlike immature spirilla ; and the third day after, the true parasite was found.

Final period of Incubation : still no uniform general change. Once my note ran 'blood hardly altered, plasma clear, fibrillation indistinct, very little opacity, and no peculiar change except the presence of a few active spirilla : on further scrutiny with the $\frac{1}{24}$ in. objective (immersion) no other peculiarities seen. The Osler-masses have no evident connection with the fully-formed parasites, being just the same as seen before these appeared, and so protoplasm, white cells and red discs, a few round specks of ordinary aspect and fibrine-bands seen.' By the Albrecht process true initial-stages of the organism were not clearly made out. On the second day of this period appearances not peculiar, though high fever was commonly at hand. Late at the human first Incubation I once noted the presence of large, round nucleated cells, but did not see the spirillum, which may have been overlooked, for there is no valid reason to suppose any difference in experience here.

b. First Apyretic Interval of Man.—Whilst throughout this truly incubative-period the blood may be undergoing modifications, yet amongst the many patients examined at Bombay, I did not perceive any considerable blood-changes upon successive days until shortly before the Relapse. This is a negative datum. Equally in abortive attacks after the first crisis, no peculiarities were to be seen.

Late Incubation-stage of First Relapse in Man.—Going back to the 4th or 3rd day before relapse, I have found in fresh blood 'fibrillation scanty, plasma clouded, white cells not numerous, red discs piled or dispersed some being small, protoplasm sometimes swarming with granules, free granules not many, spirilla not certainly seen' (they might be present). 2nd day—'Plasma clear but clouding on slow setting, much protoplasm large and small, leucocytes not increased ; in dried specimens (acetic acid) clumpy protoplasm and filaments, free granules, reticulated cells (? large granule-cells of the fresh state), spirilla seen.' 1st day—'Protoplasm plentiful both large and small, granular or swarming with active granules, several moving free granules, spirilla present.' On the m. of relapse—Fibrillation distinct, plasma clouding, the white cells vary, so red discs, protoplasm abundant chiefly small, some free specks active or quiescent, no undoubted peculiarity and the spirillum present : once the swarming masses had a yellowish tinge, once some elongated filaments of uncertain character were noted. With advent of pyrexia—'Plasma clouded, active specks, white cells and large granule-

cells common, the swarming protoplasm not increased ; no movement but molecular, and it seems as if the free molecules were growing up into spirilla, they are chiefly indicated by their effects upon the red discs ; t. 102°, p. 114 : the same specimen when treated with acetic acid, displayed a few organisms.'

Upon reviewing all the available instances, chiefly including dried specimens taken at 3-hour intervals, I noted that the parasite always made its first appearance in scanty numbers, being commonly preceded by particles (? protoplasmic) capable of being regarded as initial or immature forms ; and also that in course of the first day of fever, it was sometimes absent for 3 or 6 hours until the near approach of pyrexia, when it became invariably present. Respecting the incubation-stage of later relapses, but few data were procured.

Febrile Stages. A. *First Attack*.—The simpler comparative series agree with the human, and are probably the more typical. During the brief febrile onset, the blood differed hardly from its previous aspect, excepting that the parasite was always present. Thus, 1st day—plasma commonly clear and fibrillation distinct, the leucocytes as a rule not perceptibly increased or changed, small protoplasmic masses common, free granules rare, spirilla several. 2nd day—white cells sometimes increased, spirilla more numerous. 3rd day—no change ; but in a severe prolonged case, the significant addition of very large, pale granule-cells inclosing red discs as well as colourless nuclei (see M, Plate II.), spirilla many ; very soon after the acme (t. 106°·8), when the blood suddenly changed its condition, the parasite disappearing : free granules and large, sluggish protoplasm becoming apparent and crisis beginning ; next day the sharp secondary fever, and the animal near dying. Commonly at crisis the blood simply reverted to its normal aspect, white cells not much increasing, active or quiescent free particles and protoplasmic masses of various size are to be seen : health seems to be at once re-established. In the human (? deteriorated) subject the phenomena are apt to be more complex. By aid of the Albrecht process it sometimes appeared that a few mature and some immature (?) spirilla persisted a few hours after acme ; but it was always possible the temperature had not then passed its climax, and these data did not invalidate the conclusion that about the critical epoch marked blood-changes occur.

In the Human Invasion, during fever remarkably slight general alterations were seen. At initiation, none were invariable, whatever the temperature ; the protoplasm sometimes becomes vacuolated. In 151 ordinary survivors at mid-course, no peculiar change ; in severe cases large granule-cells were seen, and in the worst some fatty endothelial cells. The phenomena of acme seem to be associated with a tendency to this secondary blood-contamination, together with disappearance of the parasite. At crisis, the leucocytes had commonly much increased, active free granules were seen in fresh blood prior to its setting (when they become imbedded and lost to sight) ; the spirillum was not to be found. With a not uncommon tendency to 'lysis,' it may persist and so during pseudo-crises ; with decided lysis, granule-cells and endothelium were noted, just as in the severest cases (generally fatal) at acme of ordinary attacks.

B. *First Recurrence*.—In 156 ordinary cases embracing the whole relapse, inspection of the fresh blood did not show any constant abnormalities, except the spirillum; hence a further resemblance to characters of the Invasion. As variations, fibrillation was oftener indistinct (47 per cent. of cases) and blood-plasma clouded (54 p. c.), free protoplasm often abundant and free granules more frequent (45 p. c.); large nucleated and granule-cells oftener (viz. as 18 : 10); leucocytes more numerous, and the spirillum at first oftener obscured (see below). At the acme and crisis, granules and cloudiness were commoner, but large granule-cells less frequent: the spirillum tended to linger longer.

C. *Second Recurrences*.—The general aspect of the blood not peculiar; plasma and fibrillation various, so white and red cells, free granules uncommon and large granule-cells not seen; the spirillum commonly more sparse, tending to disappear early, but persistent at pseudo-crises; rudimentary forms were sometimes indicated.

D. *3rd and 4th Relapses*.—No peculiarities detected: the spirillum may not be seen in fresh blood. At intercalated, partially suppressed and latent Relapses of various dates, the general aspect of the blood was not more characteristic: its plasma oftener clouded than clear and fibrillation indistinct, free granules comparatively rare, free protoplasm not so; granule-cells were sometimes to be seen and the leucocytes somewhat increased. The chief interest of these cases attaches to the spirillum, which was not always to be seen; immature filaments were sometimes indicated.

In sum, there is a noteworthy absence of marked general blood-changes, concurrent with all stages of the spirillar disease of ordinary degree. Whether or not this negative feature is wholly peculiar to the spirillar disease, I am not able to state from want of similar data for other specific fevers of man. In severe and fatal cases of relapsing fever more positive results are found, which are chiefly noticed below; but these are, in great measure, incidental.

PART II.—DETAILED DESCRIPTION OF THE BLOOD.

The Non-spirillar elements come first under notice; and under this heading are described in succession blood-plasma, fibrillation, the red and white blood-cells, granule-cells and endothelium, free protoplasm and rods, filaments and granules. The relation of these structures to the stages of pyrexia, and in other fevers, is also indicated. Illustrations of their aspect are supplied in Plate II.

The Blood-Plasma.—By this term I mean the medium in which float the more solid constituents of the blood, and its aspect is noted as being either 'clear' or 'clouded.' It was clearest during high temperatures in relapsing fever and remittents: in intermittent and symptomatic fevers (e.g. pneumonia) it was usually clouded, and even in health it may be more or less opalescent. When free protoplasm abounds, the plasma is commonly thick and somewhat granular in aspect. It is comparatively rare to see a clouded medium with many spirilla.

During the invasion-attack of relapsing fever for the first few days the plasma was much oftenest clear, whilst later on it became as often clouded ; at the acme of attack it was clouded in one-third of the cases, and at the fall in nearly one-half.

During the relapse a clouded aspect was met with in upwards of one-half the total observations, yet most frequent at first and less so towards the end of attack ; at the acme, however, a clouded state prevailed in over half the cases and so in the fall. In the typhus-group of cases the plasma had no uniform aspect : in uncomplicated secondary fever it was clear at first (varying individually throughout) and towards the close of high fever tended to become clouded ; this remark applies to remittents, and also to enteric fever in Bombay.

Fibrillation.—The visibility of fibrillation is greatly contingent upon the state of the blood-plasma, and doubtless the fibrine bands may escape notice in a clouded medium ; they may also be missed when forming only after a comparatively long interval. When seen they are general or partial, delicate or thick, radiating or inclined, and disposed in close or open meshes ; the rate of their production may be quick (beginning in 2–3 minutes) or slow. However abundant the spirillum, it never seemed that coagulation of the blood was materially influenced by its presence ; fibrillation in different cases proceeding in different ways and degrees notwithstanding the coexistence of swarms of the parasite, and so far as seen precisely as when these organisms were absent. This remark is not irrelevant as regards the possible production of thrombus or emboli (with hæmorrhages) in relapsing fever.

Commonly the inter-fibrillar spaces were occupied by liquid more or less clear, but occasionally by a substance clouded or pellucid ; which after a time contracting small, rounded, open spaces were produced where the spirilla preferably disported in the liquid serum. Exquisitely clear fibrillation was seen not only during fever, but occasionally at the fall ; the radiating bands in one instance (t. 96°) being almost crystalline in aspect. In young persons fibrillation may be scanty or partial ; amongst adults deficiency was seldom visible in ordinary cases, but in some which proved fatal, its absence (or long-deferred production) was striking as indicating a greatly altered state of the blood : the cause of death was not always alike. In the majority of fatal cases, however, fibrillation was not defective at either high or low temperatures, whatever the prevalence of granule-cells or spirilla.

Invasion-attack.—Commonly distinct at first less often so towards the end, during the whole pyrexial period in about 20 p. c. of cases fibrillation was indistinctly seen : at the acme rather oftener than this, and in the fall it was indistinct as often as clearly seen. During the relapse fibrillation was indistinct twice as often as in the first attack, and most so during the first few days ; at the acme in more than half the cases, and also in the fall. In secondary fever it was slow to form, or absent, and seldom distinct ; in the typhus-like group seldomer distinct than scanty or quite wanting, when, too, the red discs were starred and unusually mobile. In remittents, during sustained pyrexia fibrillation was commonly distinct with clear plasma (sometimes remarkably so), and towards the end more indistinct : and in intermittents generally,

it was indistinctly seen or scanty. In enteric fever it was defective or indistinct.

Red Corpuscles.—The red discs being susceptible of change from many physical conditions, their aspect in spirillum fever could seldom be regarded as significant: thus, their number might be proportionally deficient, their tint paler (with thinness, or peculiar translucency in children), their dimensions variable (especially in young subjects and at close of illness), variations of form might be considerable even in the same preparation (*e.g.* at margins and centre of specimen) and clearly attributable to inspissation or pressure, or alteration of temperature. The bent or cupped, shrivelled or starred aspect was, however, so general as to appear not artificial; and, besides, budding at one margin, throwing out wavy processes from any part of the superficies and a general elongation of the disc into spear-head shapes, were other appearances seemingly pre-existent. Fragments of the red cells might become detached or their hæmaglobin separated, and free pseudo-crystalline forms ensue. The occasional production of long flexible filaments, two or three times the diameter of a normal disc, was, perhaps, the most perplexing phenomenon; especially when these filaments became detached. Variations in consistence were indicated in a flaccid, non-elastic aspect, in a tendency to trail, adhere or blend, or by the opposite condition of unusual mobility and dispersion. As to disposition, that in short piles seemed the normal, the most common departure being a less regular heaping together or a wider scattering: doubtless fibrillation to some extent determined the arrangement of red discs, but not always, and with compact fibrin-bands, the discs might be dispersed and highly mobile. The involution of red discs in various states, by white cells or protoplasm, will be attended to below.

Invasion-attack.—A changed and much varied aspect in 56 p. c. of observations during fever: at acme either a quasi-normal or variously altered appearance, and so in the fall. Relapse—discs seemingly changed in 44 p. c. of observations during fever, but not uniformly; similar remarks apply to the acme and fall. In fatal cases their state though often highly, was not uniformly changed, either when hæmorrhage had occurred or when granule-cells were present; and in the typhus-like group the red discs might be shrunk, bent, blended or dispersed or otherwise irregularly altered. During secondary fever their aspect was not peculiar. In remittents no particular changes were noticed, except a tendency to blend as fever progressed; and in ague the discs were in various states, not alike at high temperatures.

In their relationship with other blood-constituents nothing decisive appears: as regards the spirillum, from both theory (the red discs being oxygen-carriers) and observation (the parasite sometimes seeming to cling to the red-corpuscles), some special connection might be supposed here, but of this no valid evidence was seen; thus, when during fever the organism was present, the red discs were in a quasi-normal state in 54 p. c. (invasion), or 43 p. c. (relapse) of all scrutinies; whilst when the organism was absent, they were unchanged in only 20 (invasion) or 25 p. c. (relapse): during the fall (spirillum absent) some striking alterations, amounting almost to disintegration of the discs, were seen thrice

in thirteen selected cases. The fixed connection of any of the states with prominent symptoms was not apparent.

White Blood-cells.—Leucocytes of the ordinary character were almost always to be seen in the field; the terms few or many, or several, being used to express their comparative frequency in the absence of more precise measurements.¹ It is probable their number was invariably larger than the commonly accepted standard, and the use of acetic acid usually brought into view more than were seen in fresh blood (the cells being often in small groups): yet my impression remains that a great excess of leucocytes was not a necessary feature at all stages of the spirillum fever; and a due allowance should be made in native patients, for a pre-existing leuckæmia due to malarious influence. Another obstacle to absolute precision pertained to the method used of collecting blood, by pricking a finger sometimes previously tied tightly; for in this way lymph became inevitably mingled, and also an excess of white cells from the blood-vessels. The ordinary aspect was retained, and in the warm, moist air of Bombay amœboid movements were usually very distinct. Pigment was not augmented.

The main point of interest here, is the great probability that in spirillum fever the same source furnishing leucocytes (*i.e.* the spleen), also not rarely contributed to the blood similar cell-forms which are larger in size, and granular or even fatty in aspect. A further transition pointed to the large granule-cells described below, and with these a superabundance of leucocytes usually co-existed: finally, it has seemed to me likely that development of the spirillum takes place within these cells.

In the invasion-attack the leucocytes were superabundant in 56 p. c. of observations during pyrexia, and this preponderance seemed to increase with the progress of the attack until the acme: at the fall they were still common. During the relapse they were excessive in 62 p. c. of observations during fever, and early in the attack acquired this predominance: at the acme they were still numerous, and also at the fall. In fatal cases they were common, especially in those instances where large pale cells and granule-cells abounded; also after external hæmorrhage, and in anæmic subjects: in the typhus-like group it is not stated that they were in unusual excess, possibly they were in part substituted by large granule-cells. In secondary fever (the rebound) white cells were sometimes very common, but not invariably. In remittents an excess was sometimes observed, and in ague leucocytes were not necessarily numerous during the febrile paroxysm.

Large Granule-cells (see M, Plate II.)—These abnormal constituents

¹ Dr. Boeckmann of Giessen ('Deutsch. Archiv. f. Klin. Med.' Sep. 1881) has recently furnished measurements showing that the white cells increase during fever, reaching their highest number at the acme and crisis; thenceforward the proportion gradually diminishes to the normal: this course is regarded as peculiar to relapsing fever, and particularly the advent of large white cells at the crisis. The number of red discs distinctly diminishes during fever, and is fewest during the critical and immediate post-critical periods; it does not revert to the normal within apyretic periods, but increases slowly and in correspondence with the slow convalescence after illness. These data entirely confirm Dr. Heydenreich's earlier observations (*l. c.* p. 11), wherein also the late periodic augmentation of leucocytes is insisted on. The simple increase of white blood-cells during fever, was noted by Dr. J. R. Cormack, at Edinburgh, 1843.

of the blood vary in diameter from $\frac{1}{2000}$ in. to $\frac{1}{800}$ in. or more, and are formed of some translucent material in which minute granules are disseminated; their nucleus may or may not be visible without reagents; at first albuminous the granules seem to become fatty, and are then larger and less regular. The shape is globular, ovoid, or elongated: there is no colour; amoeboid movements are very rarely to be detected under ordinary circumstances. The number of these cells ranges from scarcity to abundance; in disposition they are isolated, or in small groups; and commonly many white cells co-exist.

In unselected cases they were seen in about 8 per cent. of invasion-attacks during fever, and at the fall in 14 p. c.: of relapses the corresponding proportions were 13 p. c. and 21 p. c. respectively; whence it appears they are commonest at the close of pyrexia and are oftener found in the second, or often more pronounced attack. They were seen in about one-eighth of acme-periods, being then somewhat rarer in the relapse. Of fatal cases one-half showed those cells near the time of death or much earlier; and if the casualties from mere exhaustion, hæmorrhage or pneumonia be excluded, a still larger proportion. In the typhus-form granule-cells were at least as common as in other varieties of spirillum fever. During the rebound after fall, which sometimes occurs in man, these cells have been seen, but not invariably; in the blood of monkeys artificially infected they were very distinct at this period, as at others corresponding to human experience, especially in the fatal cases; and, lastly, cells like them have been noticed sometimes in the pre-febrile or incubation-period (spirillar) of both human and comparative subjects.

Upon further enquiry, I find the large granule-cells not to be peculiar to this fever; thus, as regards remittents in the case of a young man passing into a state of stupor at high temperature, they were present in the blood for the time, and not so when fever abated: in several later instances, also, when the blood was examined by the acetic acid process some large cells with compound nuclei and granules in them, were frequently found: further, I saw the same in a case of tertian ague, during the febrile paroxysm; and, finally, in three instances (not to mention others) of phthisis, pleurisy and surgical fever. In none of these cases, however, was the number of granule-cells comparable to their abundance in cases like these quoted below.

Comparative Series.—1. R., a monkey at 3rd day of rebound, and $2\frac{1}{2}$ hours before its death; prostrate, breathing quickly and nearly unconscious, temp. 101° , and now falling. Blood-plasma rather clouded, fibrillation indistinct and incomplete; there are many large granule-cells, often in clusters; many free specks and small masses of protoplasm, with others larger and containing many granules; some white cells also granular; red discs in various conditions, some being normal and in piles, others small, shrunken, broken up: some of the large granule-cells contain red blood-corpuscles, the contained granules are mostly dark and all are quiescent: there is no swarming protoplasm, although all the masses seem to change their form very slowly; the granules look like fat. In other parts of the preparation, a few moving, clear granules are visible, but they are rare; the aspect of endothelial cells is nowhere marked, the appearance being rather that of jagged protoplasmic masses filled with minute, rounded granules (as sometimes seen in fatal cases amongst men); not a sign of active movement. A weak solution of osmic acid has not much visible effect, the granules not being quickly darkened.

2. O., a monkey at 2nd day of rebound, dying after two days, t. $106^{\circ}\cdot8$, much

oppressed, blood-plasma clouded, fibrillation indistinct, much large, granular, free protoplasm, many large granule-cells and white cells; no free granules; red discs bent and shrunk: some of the protoplasmic masses and cells enclose a shrivelled red corpuscle, some have a nucleus, or two nuclei, the size of a red disc, but colourless. Many of the granules in the cells resemble quiescent bacteria-forms; nothing of the spirillum is visible: some of the red discs seem to become isolated and to break up into granules: there is a great variety of pale cell-forms and protoplasmic masses: sometimes it seems as if the red discs became reconverted into pale cells (or the reverse): all the pale cells change their shape and throw out processes.

Corresponding instances in the human subject, also dying of spirillar infection at or near the close of invasion-attack, were described above as Nos. XV. and XVI., p. 75.

The relation of granule-cells to other blood-constituents, has been indicated already in the cases quoted: in milder instances of spirillum fever I note that when white-cell production was copious, these larger cells co-existing, then the spirillum was either absent or scanty: this fact seems to indicate a substitution, or at least an incompatibility, of leucocytic and parasitic production. Temperature is not much concerned, for granule-cells were found with high t, at acme, or earlier (invasion), or with low t. at fall or previously (relapse): when very numerous, the general symptoms were marked, severe or fatal, in both man and animal.

The presence of granule-cells in the blood after death from fever of suspected specific character, has often proved a useful aid to diagnosis, especially where confirmed by other post-mortem signs and the history of the case.

As to the source of these structures, there is sufficient evidence to show they may come from both spleen and lymphatic system. Ordinary white cells of various dimensions largely co-existed in 20 out of 29 unselected instances: the granule-cells were sometimes seen to exhibit amœboid movements, they occasionally enclosed red-blood discs whole, shrunk, and broken up, and their contained granules might exhibit a swarming motion. The large nuclei have been seen multiplying by division. On a few occasions it was possible to compare directly the ordinary large granular and endothelial cells of the blood, with the elements of splenic pulp (infarcted) and the contents of thoracic duct; these typical instances need not be quoted in detail, but they were clear enough to indicate the origin and nature of the abnormal cell-structures (see P, Plate II.) And for completer understanding, it is necessary only to suppose that leucocytes destined for the blood may undergo the same turgescent, granular and fatty changes, as are known to occur in the glandular epithelium of liver and kidneys. The amount of change varies in wide degree, and in proportion to its extent becomes detectible during life.

Some granule-cells were quite peculiar: in them the granules were bright, uniform and active, and clearly were not fat; they either radiated round a central nucleus or were disposed in wavy, spiral or reticulated lines, as if prefiguring some filament within. Sometimes the whole cell was occupied with granules in active swarming movement, which probably escaped by rupture.

Altered Endothelium.—The term is here applied to pale nucleated cells having a greater resemblance to the endothelium of blood-vessels,

lymphatics or the lacunar spaces of the spleen, than to other cellular elements likely to find their way into the blood-current ; direct evidence was wanting almost always, but it seems more than probable that the endothelial cells are liable to direct irritation, decay and detachment in consequence of the spirillar blood-contamination. Thus, becoming injured, their tessellated aspect is lost, size altered, and cell-substance rendered granular or fatty ; the nucleus suffers least and last, nucleoli are rarely seen ; sometimes a tendency to division is indicated. Such cells when free in the blood, are colourless and do not change their form : their number was commonly few, but might be considerable : in time of appearance they coincide with the granule-cells, most being seen at or near the acme, or in the fall, of specific fever.

The transition towards granule-cells of less size and with less distinct nucleus is sometimes so complete as to render their distinction impossible, and it must be supposed that a common form is assumed by both : this might be regarded as an enlarged and degenerated leucocyte. The relationship with vacuolated free protoplasm will be considered below ; and I will only add that my notes several times mention the occurrence of red discs in the interior of endothelial cells.

The blood of both man and animal displays these structures and the severest cases oftenest, other abnormal cell-forms always being present : the fatty degeneration may be extreme in individual cells.

As to their origin, when seen *in situ* the endothelium of blood-vessels of the spleen did not seem changed, at a time when the blood was charged with spirilla ; and this might be true generally, whilst in parts the lining cells were affected before or after their detachment : large granule-cells have been seen in the interior of the lymphatic glands, and with them granular endothelium ; also as part of the contents of the thoracic duct, in a characteristic fatal case of specific fever : and lastly, in another marked case, I found a few localised spots of fatty degeneration of the aortic walls, with at one place in the abdominal aorta superficial scales extremely changed and almost detached. It seems possible that these morbid cells are derived from different localities, their varied form indicating as much ; and some varieties may have the same origin as the granule-cells themselves. *Vide* fig. M. b., Pl. II.

Free Protoplasm (See N, Plate II.)—In the earliest fever cases investigated I noted the presence in the blood of free protoplasm distinct from that of the white cells, colourless, mobile and of dimensions varying from a minute speck to masses 3 or 4 times the volume of leucocytes. The smaller particles were homogeneous, rounded or filamentous (then being comparable to incipient spirilla), sometimes clumpy : in general they were less common than the larger forms. Of these one variety was thus described—masses less defined, very faintly seen, changing their shape quickly and finally assuming a radiated aspect and connection at margins with the faintly-marked fibrine-bands ; it is not peculiar. Usually the plasmic particles were irregular in translucency, or enclosed granules of varying distinctness often collecting and swarming in one part where active movements were in progress ; this swarming appearance was sometimes especially distinct at decease of fatal cases, but it may be seen also in healthy blood : occasionally the granules looked fatty.

In some globular protoplasm clear round spaces were to be seen containing fluid or solid matter, and the resemblance to a nucleated cell (*e.g.* endothelial) was then considerable ; and if, with one large vacuole, the amoeboid movements were slower than usual, it might be very close: once large, clear, round bodies like these solid vacuoles detached, as it were, abounded in the field at the critical fall.

It is highly probable all these forms were not identical in character ; not one, perhaps, is peculiar to relapsing fever, and several have been noticed in healthy blood. The influences of diet, and of wasting or reparative processes in disease, could not be estimated in my enquiries ; and I could only connect these protoplasmic changes generally, with those affecting the white blood-cells, towards which, indeed, transitional forms might be seen.

Invasion-attack.—The amount of free protoplasm seemed to increase from first to last, including the fall, *e.g.* a predominance being noted (roughly) in 60 p. c. of cases during pyrexia (most on 6th and 9th day), in 66 p. c. at the acme, and in 70 p. c. at the fall.

Relapse.—The amount is apparently not so considerable, a predominance being noted in 50, 55, and 56 per cent. respectively. In fatal cases the quantity and aspect seldom claimed notice : the most striking changes being somewhat similar to those occurring in more defined plasmic structures—namely, granular swelling and fatty degeneration : the same were observed in monkeys dying in the rebound. The typhus group of cases showed nothing peculiar : nor ordinary febrile rebounds after the crisis. In some remittents I noted that free protoplasm was always present, but in very various forms and amount ; it did not regularly increase or decrease with the progress of the fever, nor were the vacuolated, granular or swarming aspects common or seen at particular periods : in ague swarming protoplasm may be seen during the paroxysm sometimes : in symptomatic fevers nothing peculiar has been noted.

Filaments, Rods and Granules.—The objects included under this heading were both numerous and diverse. The most numerous class of objects were free specks, granules or short rods, and these were noted in 21 p. c. of observations made during fever of invasion-attack ; less often at the acme, but in 80 p. c. at the fall : and in relapses in 32, 12 and 100 p. c. respectively of observations recorded. They were commonest in infants, youths and women ; and amongst men chiefly in the impoverished, anæmic or cachectic. It was always possible that some 'specks' were of extraneous origin or implanted from the cuticle whilst obtaining blood, and I particularly noted that 'rods' and 'filaments' were most plentiful in specimens that had been kept for a few hours ; also that during dessication under ordinary cover, fungoid filaments might arise within a couple of hours : on the other hand, there was the striking fact that these free particles were much oftenest seen at the critical defervescence.

1. *Filaments* or thread-like particles floating in the plasma, of length varying from $\frac{1}{1500}$ to $\frac{1}{800}$ in. thicker than the true spirillum, wavy in outline and if moving, sluggish or drifting only : they were either free or attached. When proceeding from the red blood-discs they were delicate, smooth, bent, quiescent, of a yellow tinge like that of the pseudocrystals

of hæmoglobin often present at the same time, and not to be mistaken for spirillar forms ; as proceeding from white blood-cells the filaments were seldomer seen, but as attached to the protoplasm oftener and of more striking aspect : I quote a few examples.

M., 30 : terminal, ague-like paroxysm of a brief and late specific attack, t. 104°. Blood-plasma clear, fibrillation scanty, not distinct ; the field is remarkable especially for the number of free moving particles seen, there are granules single and in groups, short bent rods, clumps of snaky protoplasm from which twirling filaments proceed, (one, at least, of which is like the spirillum) are actively moving and not unlike the true parasitic clusters ; it would certainly seem that here is proof of new growth from germs in the blood. The t. fell next morning, and there was no other febrile attack.

M., adult : recurrent attack. Blood-plasma rather clouded, all seems quiescent except some free granules and wavy beaded filaments attached, not unlike incipient spirilla : specimen re-examined after a few hours and several fully formed spirilla were seen, also many small clumps of protoplasm giving off delicate, wavy prolongations, possessed of slow but distinct movements : most are knobbed at the end as if still growing, and some are at least as long as the parasites in the same field.

M., adult : critical fall, A.M., many mature and active spirilla, others beaded or nodulated, as if undergoing development or giving off spores ; there are clusters diverging from a common centre, whence also emanates a shorter wavy, bifurcating filament ; there are also delicate, free filaments quite equal to the spirillum in length, but wavy and sluggish ; they are knobbed at one end : other coarser granules are seen, but no strictly intermediate form leading to the true parasitic aspect. P.M. the man is moribund ; blood—plasma clear, fibrillation slow but clear, much granular p.-plasm and several large granule-cells : the granular aspect is so characteristic as to suggest the idea of acute fatty degeneration of both protoplasm and cells : no spirillum. No further development occurred in the A.M. specimen, which was kept for two days longer.

M., 13, a lad dying of the fever with dysentery close of attack, pyrexia intermittent. Blood : no spirillum, the plasma is full of moving particles, flexible chains and even rods ; there are masses of protoplasm the size of a red disc which are in active movement and give off numerous delicate wavy processes ; they look like zooglæa actively swarming, and also like the true spirillar masses : plasma thin, coagulation slow, fibrin scanty, ordinary protoplasm and white cells not common.

In two other fatal cases of relapsing fever long, wavy filaments of protoplasm freely floating in the plasma, were to be seen : such could never be mistaken for the blood-parasite itself, but what changes they might undergo in a brief time was not ascertained. Similar filaments were noticed in the blood of three patients suffering apparently from remittent fever (one death), and this fact points to their non-specific character ; the circumstance also that the formation of spirilla from these fibrils was never actually witnessed is, to a certain extent, against their specificity. Instances of free protoplasmic filaments were rather common, especially at beginning or end of fever : when of independent origin, these bodies were usually attended with granules and shorter rods which represented their earlier condition. One end of the filament was almost always expanded, looking like the point, or germ, whence growth had begun, and a wavy contour, with some capacity of movement, was the rule. I could not ascertain that these growths were certainly specific.

2. *Short Rods and free Granules.*—Minute specks, light or dark, seemed to be the starting-point of these bodies, and extension proceeded by budding alone or combined with elongation of the growing rod ; a

smooth filament might result, wavy or spiral and active or quiescent. It is highly probable these particles were not all of the same nature, and whilst some might well be accidental, others might represent the earlier stages of renewed spirillar development; the fact of their appearing oftenest at the close of febrile attack and with disappearance of the parasite, is indicative of a special connection, for it may be supposed that the spirillum does not become extinct at once, some germs remaining at last which if abortive still may undergo a partial growth. The circumstance of these minute bodies often resembling *micrococcus* and *bacterium*, is not necessarily adverse to their specific character; it is remarkable, however, that they were never seen to grow into spirilla, and in the absence of distinctive characters a final opinion must be withheld.

In fatal cases free granules and short rods were by no means invariably mentioned, and only seldom so as being numerous: but they were present in two well-marked instances of white cell-loading of the blood at the end of invasion-attack, and also in a case of cerebral hæmorrhage at end of relapse; in death at the critical fall they might be few or absent, or concurrent with spirilla numerous and active, yet without definite relation to the parasite. In the typhus group they were not peculiarly common. In secondary fever they were not common: in remittents free specks were not unusual during pyrexia, but never remarkable; in fatal cases here, none were noted. In agues generally nothing peculiar was seen; once, however, many minute quiescent granules of yellowish colour were found in the cold stage, and once others at the last paroxysm of a series. Free particles resembling those under notice have been seen in healthy human blood. They were not particularly numerous in the blood of inoculated monkeys, even at acme or fall, or in fatal cases. In a clouded plasma they may be overlooked, and after coagulation of the blood they may be lost sight of, only the more active particles finding their way into the serum: under these circumstances dessication of the blood and treatment with strong acetic acid will assist in restoring them to view.

The preceding remarks are copied from original notes: since my return to Europe I find several observers in Germany, Russia and Italy have described similar appearances in the blood of man and animals, under varying conditions; without, however, arriving at any definite or uniform opinion.

Specific Blood-elements.—These are represented by the organism here termed the 'Spirillum.' Below these follows in order a description of the spirillum, an account of its clinical relations to the successive stages of Relapsing fever, and, lastly, a memorandum on the natural history of the parasite. Its pathological significance is discussed in Chapter III. of the present Section.

1. **Description.**—Anatomically this includes general aspect and position, dimensions, shape, colour and consistence, numbers and variations: physiologically, the movements and mutual relations of the organism. A note is added on its detection. For illustrations see A to D, Plate I.

General Aspect and Position.—As seen in freshly drawn blood, the

spirillum is a colourless, slender, twisted filament actively moving in the liquid plasma until coagulation begins, when it seeks refuge amongst the red corpuscles and later in open spaces produced by contraction of the clot. This tendency to accumulate in fluid parts, renders the serum around a blood-clot an excellent medium in which to procure and examine the isolated organisms.

Although I am not aware of abstract reasons why the spirillum should not occur naturally outside the blood-vessels, yet at present it is known solely in direct connection with the blood. Thus, it is always absent from such secretions as the saliva, sweat, sputum, bile, intestinal mucus, urine, and from the stools; it is not found in the milk of infected women, nor in the contents of lymphatics and the thoracic duct, even during infection of the blood. Nor in the humours of the eyeball, or in the serum of blisters.

I did not see the organism in the blood of aborted fœtuses (though present in the maternal blood), but Drs. Albrecht and Spitz have been more fortunate: possibly a natural variation exists here. In the menstrual fluid and all hæmorrhagic effusions during fever, the spirillum is to be found.

In recently killed monkeys, I have several times ascertained that the organism is both invariably and uniformly distributed throughout the circulating system; and observations made at human autopsies always indicated the same important fact. It did not seem to me that the spirilla were most abundant in splenic blood, as compared with hepatic or renal, or that of the general circulation.

Dimensions.—Quiescent filaments have a length of about 2 to 6 times the diameter of a red blood-disc or $\frac{1}{1500}$ to $\frac{1}{300}$ inch (0.012 to 0.043 millimetre); when unfolded, they become distinctly elongated. Breadth—uniform throughout, and extremely narrow, being estimated in the fresh state at $\frac{1}{50000}$ to $\frac{1}{40000}$ in.; in stained preparations and those prepared by the acetic acid method, it may seem thicker and varied. Such preparations also sometimes display short and irregular filaments not to be detected in fresh blood, where appearances regarding size are apt to be very deceptive.

Shape.—This greatly depends upon activity. Thus, the more quietly moving filaments (*e.g.* seen in blood-serum) and the motionless (*e.g.* found after death of patient), invariably present the form of an extended, truly spiral rod without any such distinction of parts as head, body or tail. The spiral turns (4–10 in number) are open like those of a cork-screw, not overlapping. The two ends are either blunt or slightly tapering, appearances varying according to position.¹ In their more active state, the filaments unfold wholly or in part, becoming wavy, bent at angles, or seemingly looped; seen obliquely more illusive aspects result, such as those of bright dancing specks (which are the tips or convexities of filaments vertically placed in a medium less clear than usual), or of

¹ My preparations proved to be unsuitable for determining whether or not a *flagellum* is present at the ends of the spiral rods, such as exists in some ordinary *spirilla* and the *bacillus anthracis*: an appendage is, however, sometimes indicated, and there are appearances in the cultivated organisms, which favour the view of temporary flagella being formed.

complete revolving rings about the side of a red disc (due to the superposition of whorls), or of a series of rings ; these appearances being not uncommon in fresh blood specimens at Bombay. All such shapes were transitory, and as movement diminishes the regular permanent form is reassumed.

In specimens of blood drying quickly (as in the tropics) the common aspect was not that usually figured in books, but rather of filaments bent or much contorted, and often looped midway or at one end. Particularly in acetic acid preparations, a uniform curved appearance (as if tetanic) was not unusual. These variations occur during the process of dessication, and I have seen other *bacteria* at such time thrown out of shape (*e.g. bacillus subtilis* (?) in well-water, which became highly twisted when driven along by a receding wave : see W, in Plate II.)

Colour and Consistence.—The formative material of the filaments is homogeneous and colourless (or rarely of a faint yellow tinge), firm, flexible and elastic, but not as I judge either contractile or extensile ; it is translucent and so refractile that when parts of the filament cross (as in loops and at sharp bends) the overlying points appear as bright spots or knots (such points are also apparent in the *spirochæte plicatilis* of Ehrenberg). Like that of other bacteria it resists the action of concentrated acetic acid, and is readily stained by certain chemical dyes, though not by bile or the diffused colouring matter of blood.

Although the tendency to adhere together and to the red blood-discs would indicate the presence of an adhesive outer layer, none such is visible. During decay outside the body, the spirillum very slowly changes by a sort of granular degeneration, in the course of a few days: within the blood an active process of destruction seems possible, since the organism rapidly disappears at the acme of fever in a few hours, or less, though persisting for a day or more unchanged and active in specimens of the same blood taken before febrile culmination.

Numbers.—During specific fever several organisms (*e.g.* 5 to 10) are visible in the field at one time ; not seldom they are too numerous to count, and occasionally they are present in swarms, being apparently nearly half as common as the red discs themselves. Enumeration then becomes impracticable, from both the incessant movements exhibited and the tendency to cluster in dense masses. Small-sized subjects, as infants and the monkey, have in my experience displayed the greatest number of parasitic organisms. During incubation-periods and often initiation of fever, the spirilla were few or even very sparse. My observation did not, however, encourage the idea of an infection of the blood so scanty as to interfere with its tolerably facile recognition ; more than one organism being always found in an ordinary specimen of blood, containing not more than $\frac{1}{4}$ of a drop.

Supposing that in the body of a small-sized individual there were 10 lbs. of blood, there would be about 250,000 millions of red discs (Carpenter's 'Physiology,' 9 ed. 1881) ; and if only 1 spirillum to 1000 discs, the aggregate of parasites would amount to 250 millions. Unquestionably the proportion was commonly higher than this, and hence may be inferred the vast numbers present in early and even late attacks of the infection.

Variations of Aspect.—In fresh blood some differences of dimensions and activity may generally be seen ; other variations are rarer from the conditions not being favourable to precise scrutiny, but occasionally a dotted or knobbed aspect of the filaments was detected. In an instance of specific fever of unusually intermittent type, I saw small curved filaments shorter than the spirillum but equally active (see E. *d.* Plate I.) ; and again in the fatal case of a lad, when transitions to the ordinary spiral form were also apparent. In severe infections and cases of women and children more especially, small bodies comparable to immature filaments were sometimes to be seen.

In dried preparations (particularly after treatment by acetic acid) an aspect of multiplication by fission was not uncommon, and also of many immature forms. Such were, however, as rare after simple staining as in the fresh blood itself : they are further alluded to in the Memorandum below.

In prepared sections of the tissues, the organisms within the blood-vessels retain their usual aspect of long spiral filaments ; this is shown in Dr. Koch's Photographs ('Mittheil. d. Kaiserl. Gesund. Amt.' b. i. 1881, Tab. IV.)

Movements.—According to my observations the extended spiral aspect of ordinary delineations, is that of comparative or complete repose ; whilst the highly varied forms less frequently figured, pertain to the active state which is predominant in quickly-drawn blood. On careful watching, the following varieties of movement may be detected :—
1. Rotation of the spirillum around its long axis, a few quick revolutions in one direction being followed after a brief pause by others reversed, and the organism not necessarily changing its place : the aspect is that of a wave of vibration passing along the filament, part or whole. This variety is limited to the less active state. 2. Lateral movements pertaining to greater activity ; the inflections vary from a slight bend to a quick lashing and unfolding of the twist, with the assumption of the various shapes above described ; such movements seem spasmodic (or tetanic) especially when rings are formed which drift along for a few seconds until reopened. True knots or loops are never formed. Only a part of a filament may be involved, and a jerking action has been noted as of an attempt at division, or throwing off of a segment. 3. Progression in either direction of the long axis, and of varying rapidity : this is usually attended with rotation, and the more active lashing actions do not favour progression. The filaments drift freely in currents set up.

These active movements seem to be spontaneous, but they are stimulated or re-excited by contact with neighbouring bodies, as other filaments, red discs or the fibrin-bands. With the decline of activity, movements become fitful and deferred, a lateral bending persisting longest.

The spirilla are often seen to attach themselves temporarily to the blood-discs, which when isolated then become jerked out of place and shape in a singular manner ; when passing over a group of discs, they communicate a quick thrilling movement which is equally characteristic of their presence.

Duration of movements.—After withdrawal from the body, this

varies from a few hours to one or two days or rather longer, and outward circumstances being the same, it hence appears that the organisms are not always of the same age or maturity ; in the same preparation, some will continue to move longer than others. As none were perfectly quiescent on first withdrawal from the body, I infer that in the circulation movement always persists. Occasionally (in severe illnesses) they have been noted at first sight, as being sluggish and large, and tending to cling together. In corpses with still liquid blood, I have found the spirilla in clusters and quiescent so early as two or three hours after death ; usually movements longer continue, but not always so long as in preparations made during life. The more quiescent organisms often seemed large, stiff and disposed to cluster ; these signs are not necessarily indicative of the approach of death, for they were noted in the early stages of growth under artificial culture ; cessation of movement being, in fact, preliminary to development.

Modifying influences.—Commonly, with great numbers great activity is conjoined. Debility or youth of the patient had no particular influence, nor particular forms of illness. I have found activity considerable at temperatures rather below the normal, and at acme, t. $106^{\circ}8$, it was not necessarily increased ; during experiment outside the body, it seemed to me that heat much above the normal soon led to quiescence, a brief period of excitement sometimes intervening. Local disease complicating specific fever, had no particular influence. In fresh blood-preparations, the application of distilled water at 80° caused only a slight diminution of movement : a neutral aqueous solution of Quinine of strength gr. i- $\frac{3}{4}$, checked movement in 10 minutes almost universally ; and one of grs. 3-3 in less than 2 minutes ; solutions of common salt had much less influence.

Aggregation of the Spirilla.—Outside the body these organisms always display a tendency to cluster ; this was strikingly seen in culture-experiments, and in ordinary serum left to stand the spirilla even when not numerous soon come together, and if active collect into compact groups or if quiescent form open meshes. In the corpse of a man dying at acme of fever with characteristic symptoms, I found $2\frac{1}{2}$ hours after death many quiescent clusters ; so that withdrawal from the body is not an essential condition of aggregation. Whether or not clustering in the loose or dense form occurs in the circulation during life, cannot be demonstrated ; but I am able to state that both forms were seen in blood, with the utmost promptitude taken from the finger and submitted to scrutiny. In such cases the symptoms were urgent, fever approaching its end, and the spirilla numerous. At the beginning of my enquiries I met with such an instance, the blood-plasma being filled with small masses of very active micrococcus-like masses in diameter equalling 1 to 5 blood-discs, in form varying much and devoid of colour ; these masses were composed of spiral filaments moving so actively, as to appear granular or beaded ; the rest of the blood was healthy-looking. Afterwards many other such cases were seen. At other times, the blending of the filaments was not so complete, the aspect being that of meshes with projecting ends ; and, as in a striking case of lytic deferescence with fatal cerebral hæmorrhage, the blood might show spirilla

only partially clustered. Once (also an example of meningeal apoplexy) the tendency to aggregation was so great that in a preparation kept for a few hours, remarkable compound masses had formed.

Some other condition than that of number of organisms was, however, concerned; for in one case where the number was excessively great, all the filaments seemed to be isolated.

Aggregation of the spirilla is not comparable to coagulation of fibrine or apposition of the blood-discs, for it is present in fresh blood distinctly before these processes commence. It seems rather to pertain to the reproductive process, which may sometimes tend to begin in the living blood and so lead to troubles of the circulation of the greatest clinical importance.

Surrounding Relations.—In the absence of any visible affinities, structural or dynamic, with normal blood-elements, the spirillum bears the aspect of a foreign origin; and this impression is strengthened by its abrupt entry and departure.

During fever, other unusual structures are to be seen in the blood, such as free granules, and granular cells or protoplasm; as these are most abundant towards the end of illness when the blood-parasite disappears, they may be indirectly or even directly connected with the specific infection.

When infected blood undergoes decomposition, the spirillar filaments never showed any signs of growth or increase; and slowly disintegrating, they remained quite distinct from the putrefactive organisms proper.

The practically uniform distribution of the spirillum throughout the circulating system, seems explicable from its narrow dimensions and great activity obviating, in general, the accumulation in particular parts of the body.

Some difficulties in Detection of the Spirillum.—That as regards the examination of fresh blood obstacles do exist, is proved by the fact of the organism being originally found only after long-repeated scrutinies; and at Bombay I have met with observers not unaccustomed to the microscope, who could never clearly see the filament. A magnifying power of not less than $\frac{1}{500}$ diameters and a good light are desirable. The spirilla being both colourless, very delicate and in incessant movement, their presence when not numerous is often at first indicated by the peculiar jerking and thrilling aspect of the red corpuscles; later, they may be found only in clear open spaces, around the piles of corpuscles or air-bubbles (which act as magnifiers), and accumulated at a few spots only; whence the necessity of prolonged and complete examination of the specimens of blood. When numerous, less time is needed; and once fairly seen, doubt is at an end. Failing with fresh blood, dried preparations in case of doubt should always be made; and these alone can be depended on for exactitude of research.

The fact that so much care is needed when decision is most of all desirable, made me cautious in arriving at negative conclusions if other clinical data pointed to the likelihood of specific infection. Another difficulty, still insuperable, arose from ignorance of the significance of granules, rods and filaments in blood reasonably supposed to be

contaminated; and a negative record had sometimes to be entered reluctantly, though in strict accordance with appearances seen.

Fictitious Blood-movements.—Amongst the red corpuscles movements partly simulating those due to the spirillum may be caused by active swarming of amœboid protoplasm, discrimination not being however difficult here; swift currents in the plasma may displace and contort the corpuscles in a manner suggestive of indistinct spirillar agency, but then the movement is all in one direction and more or less sustained. Isolated corpuscles in limpid serum may spontaneously oscillate or be moved by active granules, so freely as to excite the suspicion of the neighbourhood of a spirillum; but if present, the filament with patience will become evident however momentarily. Aptitude and practice alone can obviate all difficulties.

Aspects of the Blood-spirillum at different stages of Relapsing Fever.—Observations were based on scrutiny of both fresh and dried specimens; though of unequal value, they are here given as illustrating my entire experience. Since in Europe irregularities and apparent discrepancies have also been recorded, the similarity of data from both East and West becomes hereby verified.

Definition of Terms.—Number—‘very few,’ when one or two organisms only were seen in the entire specimen; ‘few,’ with one or two at a time in the field; ‘several,’ two or four in the field; ‘many,’ ‘very many,’ ‘swarms,’ stand for quantities seemingly equal to $\frac{1}{20}$ th or $\frac{1}{10}$ th the number of red blood-discs, or possibly more. An enumeration in the fresh state was often impracticable, owing to the active parasitic movements; and even in dried preparations, it could not be precise where the organisms were clustered. The filaments were isolated, grouped, or densely aggregated, according to the numbers present.

Their movements were indicated by the terms ‘sluggish,’ ‘active,’ ‘very active,’ in the last case being attended with the aspect of loops, rings and knots; they were ‘weak’ or ‘strong,’ as regards effects upon the red discs.

Dimensions were readiest estimated by comparison with the blood-discs (mean diameter, $\frac{1}{3000}$ th in.): in fresh blood the terms ‘small,’ ‘large,’ ‘long’ or ‘short,’ ‘delicate,’ ‘slender’ or ‘thick,’ referred to actual appearances, but might in fresh blood be illusory. A real diversity of size did, however, exist, especially when the organisms were numerous; the natural inference, then, being that of growth or development.

Peculiarities of aspect were noted chiefly in dried preparations, such as a dotted or beaded contour, knobbed or clubbed aspect, or one of mid-division. The spiral contour as usually displayed, was by no means invariable in even the freshest preparations.

Experience showing that spirillar infection always culminates in pyrexia, the process may be traced as follows:—

Stage of Fever. (a) *Incubation-period.*—During the earlier and commonly longer part of this stage, the spirillum is entirely absent; whilst during the later part, it is invariably present.

First or Invasion-attack.—Data for man are mostly wanting ; in the monkey, I found upon ten occasions that when first seen the spirilla were extremely few, though of usual aspect, and in the earliest hours their presence seemed to be intermittent ; filaments resembling immature parasites were also to be seen. The number of organisms increased moderately (if at all) prior to the advent of fever ; but with rise of temperature it augmented, though not in any fixed proportions. During my own late illness, I was unable to detect the spirillum at this stage, even in dried preparations ; but the examinations were few.

The First Relapse in Man.—In twelve ordinary cases specially treated, the organism was always found within a period of forty-eight hours prior to fever : on the fourth or third day before relapse, dubious (? immature) filaments may be noted heralding the spirillum ; when the organism is first distinctly seen it is very sparse, being, however, of ordinary aspect. An appearance of multiplication by division was sometimes seen, and also a variation in size. The organisms still seem to intermit, and their number hardly increases prior to fever ; nor with the onset of pyrexia is there, at once, a great increase of numbers. In two cases examined throughout, it was observed that when the parasite subsequently abounded, it was commonest (perhaps earliest) also at this stage, and *vice versa*, these differences being independent of temperature : see Charts Nos. 1 and 2, page 358, the former showing filaments present seventy-two hours before fever, a distinct spirillum forty-eight hours before, t. $97^{\circ}8$, appearances dubious during the next nine hours (t. 98° to $98^{\circ}4$), at night a distinct organism, t. $97^{\circ}2$. The day before fever in eight observations at three-hour intervals, t. throughout slightly below the normal, spirilla marked successively as 1, 2, or more, 1, 2, 1, with intermediate forms (?) 1, doubtful, 1 ; day of fever, 1 A.M., t. $98^{\circ}6$, presence of spirillum doubtful ; 4 A.M., several parasites, some dividing. This was the beginning of fever, and at 4 P.M., t. $104^{\circ}2$, the blood showed rather more organisms with developing forms. In the case of the woman, Chart No. 2, the filaments were more numerous, but their first advent was not witnessed.

(b) *Initiation of Fever.*—Invasion of man : in three instances (mean t. 104°) I found the spirilla as very few (2) or several, they were fully formed and active ; in a fourth case, t. 101° , they were absent, but may have been overlooked. In monkeys : eight observations (mean t. 104°), parasite always present, once being few and small (t. gradually rising) ; thrice several were seen (mean t. $103^{\circ}7$), four times many (mean t. $104^{\circ}7$). Succeeding observations at this time showed that with a further rise, the organisms might become fewer, whilst with no rise they might augment ; there being thus foreshadowed the concurrence generally verified, of a post-meridian exacerbation of fever and diminution of spirilla.

First Relapse : Man.—In a first series of thirty-nine ordinary observations of fresh blood, the spirillum was present nineteen times and not seen in twenty instances ; this result illustrating our common experience. Yet in a later series of ten inspections, made with aid of the Albrecht process, the spirillum was always found, and I doubt not its presence

might be thus invariably shown. Of twenty-four selected cases, the organisms were few in seventeen, and several or many in seven; first seen equally at morning and evening. That a high temperature was not necessarily concerned in the above variations, seemed quite clear: the possible causes of absence in the earlier series of cases were extremes of temperature, infancy and youth, a typhus type, local complications, and an idiosyncrasy (?) which seemed to attend some instances of spirillum fever.

Mid-febrile Period.—Human Series.—Invasion-attack.—An unselected series of 112 observations, chiefly of fresh blood, showed spirilla few 13 per cent., several 27 per cent., and numerous 60 per cent. *Relapse.*—A similar series of 130 observations gave like results. On both occasions there was noted a gradual increase in numbers on successive days of attack: thus, during invasion the proportion of spirilla 'many' rose after the third day from 50 to 77 per cent.; and during relapse, whilst on first day 'few' 55 per cent. and 'many' 11 per cent., on second day few 40 per cent. and many 21 per cent., on third day few 21 per cent. and many 60 per cent., and on fourth day few 8 per cent., many 70 per cent.

My notes indicate that abundance of the spirillum is neither so soon nor so strongly marked in the second febrile attack, and then also the terms 'swarming,' 'clusters,' occur much less often; this difference coinciding with a mean shorter duration of attack, and yet higher maximum temperatures.

The number of organisms present appears to correspond with, or determine, most of the other chief differences seen; thus, where the parasites abound they are often large, active, clustered: the converse is not so frequent. Isolated organisms may be large and active, and multiplying by division from the first, smaller filaments being seldomer seen with them; hence the idea of a moderate and gradual development during the earlier periods of visible infection, whilst with progressing pyrexia an increased production, extreme activity, and a highly varied aspect. During the relapse the spirilla in general were not only fewer, but less active and varied: the terms 'delicate,' 'small,' being also rather more frequently used then.

In a few instances carefully examined throughout, the evidence conformed to the above; thus, in the CASE No. IX., Chart 2, on the first day of fever, t. 100° - $105^{\circ}2$, spirilla beginning with several, and at even $104^{\circ}2$ (1 P.M.) still no more than earlier in the day at $100^{\circ}8$; then with rise to $105^{\circ}6$, becoming more common, long, with swellings on them and the aspect of an active production. From this date onwards, with temperature hardly ever higher, the terms employed were almost exclusively many or very many: clustering was noted on second day of fever (t. $106^{\circ}4$) and subsequently; at $106^{\circ}2$ the organisms were once entered as being rather fewer; at the end their numbers diminished before disappearance of the parasite.

In the boy's case, Chart 1, with the first rise of temperature extending over about twelve hours, the spirilla increased from some four in a specimen (t. $99^{\circ}8$) to several, with developing forms (t. $104^{\circ}2$); the daily mean temperatures were 103° , $103^{\circ}7$, $104^{\circ}4$, and $102^{\circ}8$, or about

1° less than in the woman's case, which also lasted a day longer. With this difference the blood-spirillum was much less abundant, and on no occasion marked as abundant; subsidence at the fall began earlier, and in general there were contrasts not easily accounted for. Other instances met with also presented individual characters, and on review of the whole series, it seems to me that whilst the spirilla are more numerous in the more pronounced attacks, yet they do not vary in numbers so uniformly as would be anticipated on the supposition of their being the immediate cause of pyrexia. In some cases, from the first (including incubation-period) they were commoner than in others, and this character was maintained throughout the attack; augmentation until the end being most apparent when their numbers were already considerable. A certain variety in nature or degree of infection, or some idiosyncrasy of subject, was indicated hereby.

Comparative experience.—In the more pronounced induced attacks, the spirilla were so numerous that my reckoning failed to detect perceptible differences at different hours; scrutiny of the blood could not be made as often as in human patients, yet nothing contradictory to the above was elicited.

Intermittency of the Spirilla presence.—Some of my older data seemed to show the organism may disappear for a time varying from a few hours to a day or more; but as the later observations do not confirm such intermittency, I must hold it as uncertain, however likely it be.

Summary.—On scrutiny upwards of 270 ordinary cases of relapsing fever, I am led to conclude that the parasite is practically always present throughout the febrile stages; and although in about 20 per cent. of invasion-attacks, and 21 per cent. of relapses (after excluding the first day) the spirillum remained undetected, yet analysis of the notes commonly showed some such explanation of this absence as the following:—Early or irregular occurrence of the crisis when naturally the organism disappears, this remark being applicable to invasions seen late, as usual, and to relapses which are always of uncertain duration; defective observation was doubtless also concerned; possibly, too, variations of temperature, especially the rise when the organisms may become so few as to be overlooked, or an unusual low temperature; local complications (*e.g.* pneumonia) and an altered state of the blood, as evidenced by the aspect of the red discs, were noted in some cases, whilst in a few the explanation of absence could not be accounted for in the older notes here quoted. In all late observations made by newer methods, and in all comparative instances of high fever, the spirillum was invariably found.

Acme of attack.—By this term is here meant the last ascertained date of high temperature, prior to critical fall; a final perturbation may now occur, and to ascertain with precision all the changes then concurring would require observations made at shorter intervals than was usually practicable at my hospital. Acme was noted much oftenest after noon, and on any day after fourth or fifth of invasion-attack, and third of the relapse.

Human Series.—Invasion.—Fresh blood observations : in 25 entries the spirilla were few 3, several 5, many 7, and absent 10 times : mean t. noted when few or several $104^{\circ}6$, when many $104^{\circ}7$, and when absent $104^{\circ}6$. Had scrutiny been made later than 4 or 5 P.M., the absences would, I think, have been more numerous. The same data showed, I may add, that the spirilla decline in numbers, size, and activity just before disappearing altogether, and, as is evident, the temperature of the body is not regulated by the aspect of the blood at this time. The organism may be found in dried specimens when indistinct in fresh blood on account of smaller size or diminishing numbers. At *First relapse*, in 17 older observations spirilla few 2, several 6, many 6, and absent 3 times ; mean t. when few or several 105° , when many $104^{\circ}2$, and when absent $105^{\circ}9$: hence there was apparent a tendency to persist somewhat longer in the recurrent attack, until approach of mean maximum t. Here, too, the spirilla decline before finally disappearing. Later observations on dried blood, as illustrated in the two cases pertaining to the charts shown, confirmed the rule of decline and subsidence of the organisms at acme ; in that of woman, Chart No. 2, the details being as follows :—Fifth day of relapse, 7 A.M., t. 105° , spirilla many with large nucleated cells ; 10 A.M., t. 104° , many spirilla and clusters, but fewer, some growing (?) organisms ; 1 P.M., t. $104^{\circ}6$, spirilla several, but much less numerous, immature forms ; soon after this hour there occurred a marked *perturbatio critica*, and at 4 P.M., t. $104^{\circ}2$, the spirillum could not be found in the blood, which now contained many large reticulated cells ; at 7 P.M., t. $103^{\circ}2$, at 10 P.M., t. $98^{\circ}2$, and no blood-parasite. In the boy's case (Chart No. 1) the details were these : at 7 P.M., t. $105^{\circ}2$, spirilla not frequent, immature forms ; 10 P.M., t. $104^{\circ}8$, spirilla fewer ; 1 A.M., t. $104^{\circ}8$, spirilla fewer still ; 4 A.M., t. $103^{\circ}4$, no spirillum ; 7 A.M., t. $100^{\circ}2$, and 10 A.M., t. $97^{\circ}6$, also negative results.

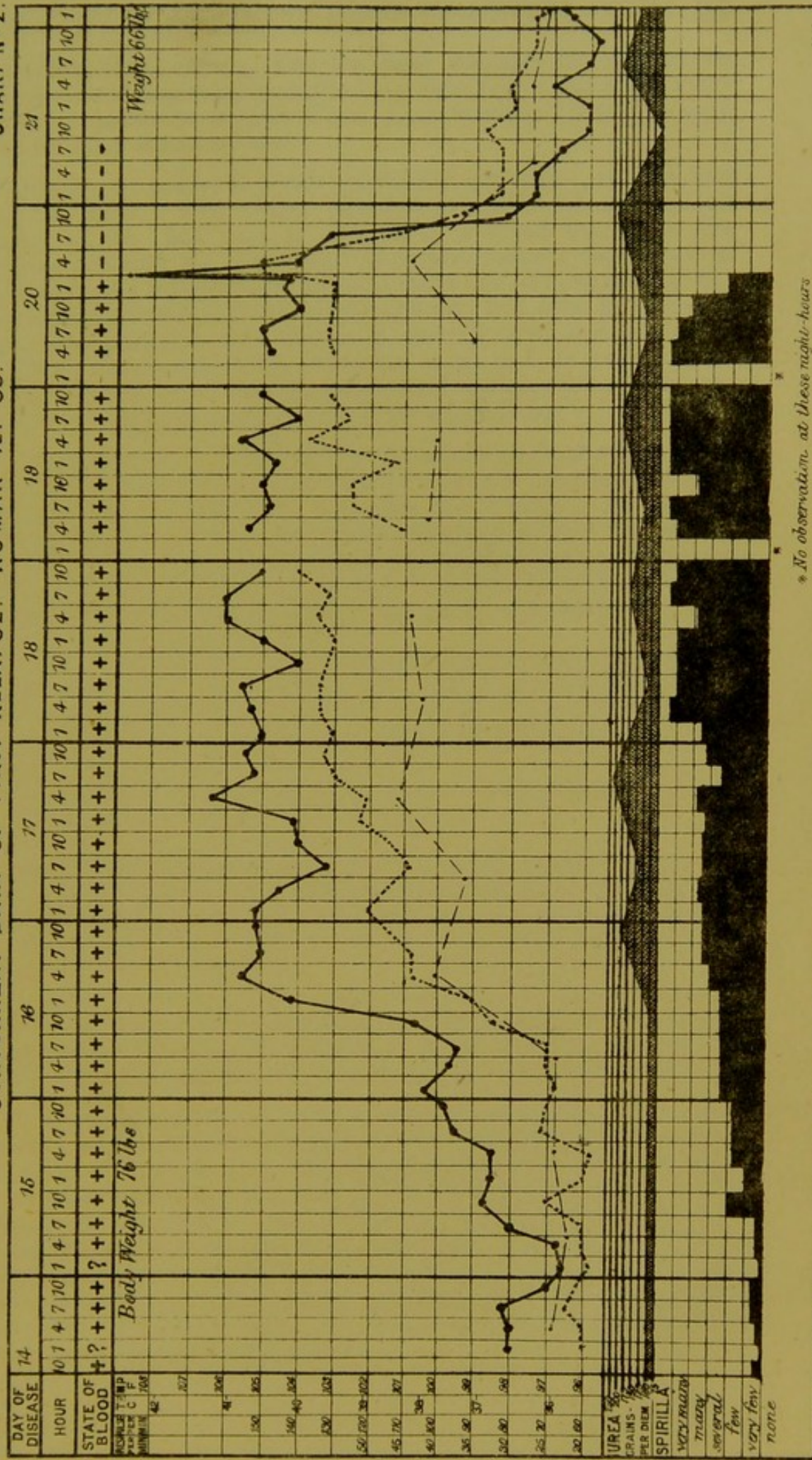
For ready comparison with these instances I will here state some corresponding details regarding acme at invasion-attack. M., 35, day after admission, 7 P.M., t. 105° , spirilla tolerably numerous, with immature forms ; 10 P.M., t. $105^{\circ}2$, parasites still common ; 1 A.M., t. 105° , a few spirilla with some thicker, nodulated filaments ; 4 A.M., t. 102° , 7 A.M., t. 99° , and 10 A.M., t. $95^{\circ}6$ (fall), no trace of the organism. These tri-hourly observations were continued for twenty-four hours longer, also with negative results ; and subsequently for nine days in succession, with continued demonstration that the spirilla disappear wholly and permanently at the acme, not reappearing until the ensuing relapse, if such occur. In all three elaborated instances the temperature had begun to decline before the parasite ceased to be visible ; and when this happened, sinking of the body-heat became more marked. The references in my notes to seemingly growing forms of the spirillum are not meant to be decisive, for it may be that such forms represent a checked development.

Comparative experience.—Here, too, the correspondence of phenomena is quite unmistakable.

The Critical Fall.—This event, abrupt as it appeared, was seldom completed under twelve hours, and therefore might be regarded as a

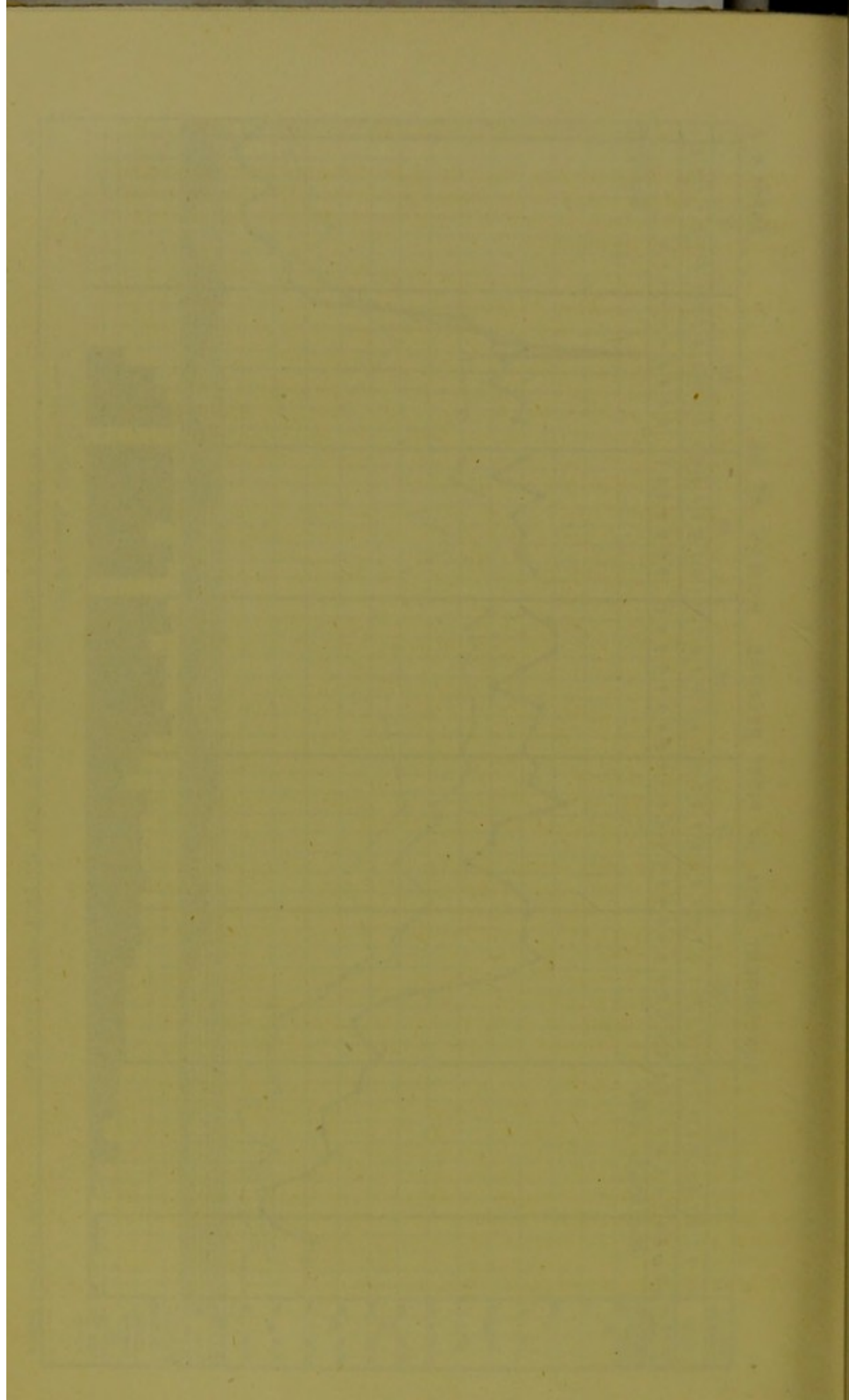
CONCURRENT DATA OF FIRST RELAPSE. WOMAN AET 35.

CHART N° 2.



* No observation at these night-hours

TEMPERATURE IN CONTINUOUS LINE; PULSE IN DOTTED LINE; RESPIRATION IN INTERRUPTED LINE.



stage in the febrile events. The few more elaborate blood scrutinies always furnished negative demonstration at this epoch ; but at the many ordinary examinations of fresh blood some seeming exceptions were noted, requiring attention here.

Invasion.—21 entries in which spirilla few 1, several 1, many 3, and absent 16 times. This datum sufficiently marks the non-spirillar character of the 'fall,' and on analysis, it is found that when the organisms persisted, the crisis was incomplete (mean t. $101^{\circ}2$), whilst when they ceased to be visible it was at or nearer its end (mean t. $97^{\circ}8$) : hence some continuance of visible infection may be looked for when the crisis is more gradual or slow than usual, the rule of its final subsidence at minimum temperature being invariable, so far as I am aware. That temperature alone is not here concerned, is, however, evident from the fact of its varying from $102^{\circ}2$ to 95° in my negative list.

Relapse (first).—16 entries, in which spirilla many and active 2, and absent 14. The two instances of persistent parasite belonged to fall in progress (mean t. $100^{\circ}7$) ; the remainder had a mean t. of 97° , with range $101^{\circ}4$ to 97° . These data show a disappearance of the parasite at the fall, quite as evidently as those of invasion-attack, although the spirillum was longer persistent at the acme ; and hence the true character of the crisis is better shown in these prompter and somewhat deeper critical events of recurrent attacks.

Variations are still marked enough to prove that the phenomena at crisis are not quite uniform ; and it is found that acme and fall pass by almost imperceptible degrees into the gradual or lysis-like mode of defervescence. The only available examples of lysis belong to the fatal series alluded to below.

During all febrile manifestations immediately following specific defervescence, the spirillum was always absent.

The different forms of Specific fever. 1. *Brief, isolated paroxysms.*—In the monkey these short culminations of infection invariably displayed the spirillum prior to the acme, though still not in fixed ratio to degree of temperature. A distinct specific incubation-period preceded these events.

In man, at first and later relapses, appearances usually accorded with the above, but sometimes I failed to see the parasite in fresh blood ; probably this circumstance meant that the organisms were very few. It occasionally happened that they were numerous. See Chart 8, Plate IV.

2. *Prolonged attacks.*—In the monkey visible infection was always copious ; intermittency of the parasite or temperature was never seen, and never the fall by lysis. In man the amount of visible infection varied considerably, and not in fixed relation to temperature or general suffering of the patient. The increase in numbers with progress of pyrexia seemed to be invariable, and with many organisms in the blood some degree of accompanying fever was always found. Two unselected cases are shown in Charts 1 and 2, which will furnish the necessary details on inspection : they are not absolutely alike, and probably every febrile event has its own peculiarities.

3. *Intermittent attacks*.—Complete intermittency of temperature during specific fever was not observed in the lower animal. In man several remarkable instances were met with at both invasion and relapse, which were accompanied by persistence of the spirillum, and so distinguished from true critical phenomena. Charts 5, 6 and 7, Plate IV., belong to cases displaying these *pseudo-crises*, with persistence of the spirillum at low temperature occasionally met with during first or recurrent attacks. Details (here omitted) proved that the organisms were not numerous at the lower temperature reached (*e.g.* 95° – 96° F.), and sometimes they were best, or only, seen in dried specimens of blood: sometimes, however, as in Chart 5 of an invasion-attack, they were abundant at a temperature of 98° . The general result of this series, including examples not now mentioned, is that during marked febrile intermissions the blood-parasites do not necessarily disappear: at specific relapses, especially, they may not even diminish in numbers then.

Chart No. 9, Plate IV., illustrates the blood phenomena at a quasi-latent or suppressed relapse. It happens that detailed observations were made in this case, anticipating the relapse; and whilst the ordinary chart showed but little indication of fever, general symptoms concurring, the more elaborated chart proves that infection ended with a smart, if brief, febrile paroxysm occurring at night. There was a distinct incubation-period in this instance, just as in the comparable short spirillum fever of monkeys.

Fatal cases.—Death from 'fever' alone, took place generally at or near to close of first febrile attack; and as then the blood-parasite was not necessarily of different amount or aspect, the fatal event seemed commonly independent of spirillar changes. Of forty-three selected instances, the organisms were, at last observation, present and more abundant than usual in nineteen; and eleven times they had but recently disappeared. The remaining thirteen cases were those of casualties from complications, arising subsequently to cessation of specific fever. In cases fatal at or near the probable acme of attack, I have found shortly before or shortly after death, the spirilla either many or few; the temperature had then been high (*e.g.* $+105^{\circ}$) and I inferred the crisis to be at hand. In a larger series, the mode of defervescence indicated was the gradual and prolonged lytic form. The last mean t. was about 100° , general condition depressed, often jaundice and peculiar complications. In three cases the decline of temperature was referable to copious hæmorrhage (one nasal, two gastric), these showed the parasite shortly before death at $98^{\circ}6$, 98° , and $97^{\circ}5$. Once a man was admitted with a temperature of $97^{\circ}5$, dying soon after, whose blood was charged with the parasite; and other instances pointed to not only a close association of critical blood changes with death, but a contingency of the detection or non-detection of the parasite about this time according to precise date and method of blood examination. It also seemed that soon after the infliction of injury they might be associated with (*e.g.* meningeal apoplexy, or other hæmorrhage, the patient lingering for a time), they may *ad interim* disappear from the blood and not be found at or after death.

In about one-third of the casualties a tendency to lysis was evident;

and the spirillum might be found until lowest temperature point ; its numbers declining, and its aspect seeming large, sluggish, and sometimes granular or knotted. These variations are interesting from their resemblance to changes taking place in organisms under artificial culture outside the body, and being such as might lead to obstruction of the living blood current.

When deep jaundice or a typhus type of fever was present, or local inflammation, I could seldom perceive any peculiarity in aspect of the spirillum : the number might be few, or very few, and the maximum did not occur in such cases.

Summary and conclusions.—The following data point to a real connection of spirillar blood contamination with the pyrexial attacks of relapsing fever :—(1.) Infection is always followed by fever. (2.) With the advent and progress of pyrexia the blood-parasites increase. (3.) They disappear with the cessation of fever. (4.) By contact with the sick and by inoculation of blood containing the spiral organisms or their germs, the disease may be conveyed to new or old subjects.

The following data point to conditions modifying the connection above implied :—(1.) The presence of the blood-parasite during several hours, or for one or two days, prior to fever. (2.) The sudden onset of pyrexia is not preceded by or attended with, a proportionate visible augmentation of the spirillum. (3.) The absence of any fixed relation between variation in form and intensity of fever, and varying numbers of the organism. (4.) The persistence of the parasite during pseudo-crises and defervescence by lysis.

The above remarks apply to the earlier and more pronounced specific events ; during accidental pyrexias of all kinds the blood-parasite is invariably absent ; and even at the third and fourth periodic relapses which are occasionally seen, I was commonly unable in fresh blood to detect the organism ; an opportunity of blood examination by the newer methods did not, however, present itself.

MEMORANDUM ON THE NATURAL HISTORY OF THE SPIRILLUM.—

Classification.—The organism found in the blood belongs to the vegetable kingdom ; by recognised authority it is referred to the class of ALGÆ, and more particularly to the Oscillatorial group. It is ranged with several other pathogenic organisms amongst *Bacteria* (Schizophytæ), forming (like *bacillus*) a member of the Nematogenous subsection, in which the component cells are arranged in rows ; and, lastly, it comes into a small group characterised by colourless, screw-shaped threads, and embracing 3 genera—namely, *Vibrio*, in which the filaments are short and slightly undulating ; *Spirillum*, in which they are short, spiral and stiff ; and *Spirochæte*, in which they are long, spiral and flexible (Dr. F. Cohn, 'Beiträge z. Biologie d. Pflanz.' heft 3, 1875). At first, the blood-organism was referred to the genus *Spirillum* ; but now it is regarded as belonging to *Spirochæte* (being named *S. Obermeieri*, Cohn), which contains only one other species, viz. *S. plicatilis* Ehr. found in river and well-water. By the kindness of Prof. F. Cohn, I am informed that the genus *Spirochæte* is doubtless a parasitic repre-

sentative of the genus *Spirulina*; and that beyond their external characters and habitat, nothing certain is yet known of the life-history of either the *Spirochæte Obermeieri* of relapsing fever or the *S. plicatilis* of common water. According to my observations at Bombay, the blood-organism does not precisely correspond to the above definition of *Spirochæte*, differing in its remarkable faculty of unfolding during its more active movements; whereas the organism I found in tank-water (which closely corresponded with *Spirochæte plicatilis* Ehr.) preserved its well-defined spiral character in all its varied contortions (see V., Plate II.). As it was also much larger, being thick enough to display a double contour, I did not conceive it possible to confound it with the blood-organism. By general admission, a certain degree of variability belongs to all those minute plants; and when one whose natural habitat is outside the body becomes introduced into the blood, some change of aspect might be anticipated. Still, in the present uncertainty of knowledge, and for the sake of euphony, I have preferred to retain the name *Spirillum*, originally applied by Dr. Obermeier. Respecting the spiral organism found in saliva, I cannot regard it as being identical with that of the blood; although the resemblance is certainly considerable (see T., Plate II.).

Identifications.—That the blood-organism seen at Bombay is identical with that found in Europe, was promptly recognised by means of specimens sent during the late epidemic to Drs. F. Cohn of Breslau, Koch then of Wollstein, and Albrecht of St. Petersburg, who have each expressed to me their conviction that no essential difference here exists. Nor is any apparent in the photographs, drawings and many full descriptions of the European form, which I have had access to; and, besides, I have myself directly compared specimens from both East and West without finding any real diversity. Since the whole range of clinical phenomena proves to be strikingly alike, it might be expected that the identity would extend to the characteristic blood-parasite also.

I am able to assert, too, that the blood-spirillum of man is the same as that of the monkey inoculated from him; the proof of this identity is equally manifold, and now as generally accepted. For latest illustration, I may refer to the photographs in Plate IV. of Dr. Koch's late article, published in the 'Mittheil. d. Kaiserl. Gesund. Amt.,' Band 1, Berlin, 1881, which upon comparison will sufficiently confirm the present statement. Under culture, the organism from man and animal equally extends. Clinical phenomena of infection, so far as comparable, are alike.

Clinical Memoranda.—There not being available any direct information for guidance in the interpretation of pathological phenomena, I should add that assistance from analogy with other pathogenic bacteria does not yet furnish trustworthy aid. Hence there remains only to briefly describe what was learnt during pathological enquiry at Bombay, of the apparent development and reproduction of the spirillum.

Development and Growth.—The fact that the spirillum is commonly reproduced at least once within the body, shows that it here undergoes a certain life-cycle; whether or not this be the complete process throughout

is unknown, but probably not so ; since, when placed under other conditions (as in culture-experiments), appearances are seen diverse from those detected in the blood, and, *a priori*, it is supposable that the growth of a foreign organism implanted in the human frame would be more or less erratic.

Whilst the ordinary aspect of the spiral filament is fairly uniform, there are seen chiefly at beginning and end of their sojourn in the blood variations of aspect considerable enough to warrant the idea that their germination and reproduction are not always effected in the same way ; or at least may take place in association with quasi-normal blood-elements of different kinds, and thereby assume somewhat different phases. Such association might seem close enough to suggest that the spirillum was originally a direct product of some one blood-element, did not the phenomena of relapses and the whole history of propagation require for their comprehension another conception. That the spirillum is strictly an autochthonous element of man is primarily inconceivable, and the bulk of evidence tends to prove that it is a truly parasitic growth.

Germ.—That there exist spores or germs of the spirillum is highly probable, because other bacteria possess them ; and it is quite possible they have been seen in the blood, without being recognised. Still more likely is it that certain short filaments seen (described below) were spores germinating, the argument here resembling that for the existence of other germs in the air, which are commonly known by the growths they give rise to. Further, the practical facts of contagion require the presence of fertile spores, since spiral filaments are absent in the *secreta* and *excreta* of the body, and propagation by blood-inoculation is obviously not the rule in common life. In my opinion, it is more than probable that all spirillar blood contains germs as well as filaments ; and, also, that these may persist in the blood somewhat the longest, being of themselves capable of producing infection. For example :—

H. V. C. scratched a finger at autopsy of a woman, who died of fever with cerebral hæmorrhage, at close of first relapse ; the temp. then declining, and no spirillum in the blood. A few days afterwards (*i.e.* three or four) specific fever came on (the blood contaminated) ; and critically subsiding, was followed by a periodic relapse.

C. E. scratched a finger at autopsy of a woman dying of epistaxis and femoral thrombosis, which followed in two days a pronounced specific attack ; temp. at death high (secondary fever), but no spirillum in the blood. Three and a half days afterwards he showed high fever, which in a typical manner relapsed twice. Date July 1879.

In the first of these cases I had noticed and figured minute granular bodies actively moving, which $3\frac{1}{2}$ hours after death had grown into beaded rows (spleen) : see E.c., Plate I.

I should observe that H. V. C. had been exposed to infection in the medical wards for several months, without acquiring the fever ; whilst C. E. was a surgical assistant not in the way of ordinary contagion.¹

¹ Dr. Motschutkowsky of Odessa made a series of experiments on the human subject, which seem to prove that the inoculation of relapsing fever in man is successful only through blood taken during febrile periods. I quote at second-hand, but it is stated that the presence of the spirillum was not needful to ensure the success of such inoculation. 'Centralbl. f. med. Wissen.', 1876, No. 11. Much experience induces me to remark that as the blood was taken at febrile periods, it probably contained spirilla which were not detected in the fresh material used.

The chief objection to this view of germ-infection, was furnished by my unsuccessful inoculation of the monkey with non-spirillar blood taken after critical defervescence ; but it is possible these animals are less susceptible than man. Inoculation with dried spirillar blood failed in them, whilst it is the general belief that relapsing fever amongst men may be communicated by fomites. I do not infer that all cases of fever are equally infective ; and it may be severe or fatal cases showing characteristic blood-lesion, like the two above, are such as are most likely to give rise to post-febrile infection. For other remarks, see the next Chapter.

Growth of Germs.—In both man and animal, I have on successive days carefully examined the fresh blood for evidence of spirillar development ; dried specimens were also as often scrutinised, and series of special trials were made with the venous blood issuing from the spleen, liver and kidneys, the portal blood, that of the inferior vena cava and jugular vein, and the contents of the cardiac cavities, of monkeys killed at different days of infection and promptly opened to insure the least possible post-mortem change. The results of these prolonged essays were often interesting, but seldom decisive ; and I concluded that the blood is too complex a medium to be suitable for this kind of research. The less fallacious appearances noted were those of minute free particles of rounded form, others elongated, tailed, clubbed or knobbed in the middle ; often distinct filaments, short, bent, or even spiral, and in fresh blood not devoid of movement (see E, Plate I.). It is noteworthy that these aspects were commonest near the end of specific fever, when the spirillum is about to disappear, and when increased germ-production might be anticipated. In several severe and fatal cases, too, the blood was unusually charged with such particles ; and this is remarkable. The chief objection to regarding the granules and filaments as immature or abortive spirilla, was the fact that they seemed too coarse, thick or irregular, and did not appear to advance beyond a certain stage ; so that no forms were seen clearly leading up to unmistakeable spiral organisms. Besides, in non-spirillar blood similar granules and even filaments might be seen, which were formed of compact protoplasm only.

Not being acquainted with the normal aspect of incipient Spirochæte, I had not the essential aid of comparison with standard forms ; and this was a serious drawback to inference.¹

Briefly, whilst it is highly probable that the spirilla grow from germs in the blood, yet the fact of such growth has not been fully demonstrated.²

¹ The complex phases in development of a common spirillum, as described by Drs. Geddes and Ewart (Proc. Royal Soc., No. 188, date 1878), are not indicated in the blood of relapsing fever, or reproduced during culture of the blood-organism ; the finally liberated spores assuming a tailed or 'comma' aspect, might, however, be not unlike. In some of my culture-trials the spores of a true Spirillum must have accidentally found an entry, for I saw several stages of their growth remarkably well ; they did not correspond with the above-named observations.

² I find that the experience of observers in Europe has been similar to my own. One of the latest records is that of Dr. R. Albrecht ('Deutsch. Arch. f. Klin. Med.,' July 1881), who preserved and examined at intervals preparations of fresh blood taken 1, 2 or 3 days before expected relapse ; with the result that sometimes the spirillum was not seen at first, but was seen soon afterwards, having apparently become developed in the preparation itself. There is no intimation of the author being aware that often, if not invariably, a specific incubation-period precedes all febrile events ; and I know that during such period,

Endo-cellular production.—Since at their earliest detection the spirilla had generally a matured aspect, I was led to consider if they were not developed in structures connected with the blood, entering the blood-plasma only when liberated therefrom. Such structures would be the white blood-cells and the vascular endothelium; and, as matter of fact, large cellular and plasmoid bodies containing granules and filaments (visible on staining), are to be seen in infected blood. They were very numerous in splenic venous blood of the monkey, and also in other perfectly fresh venous bloods; and my first conviction was, that this evidence sufficed to demonstrate the real mode and place of parasitic reproduction. It was supposed that the free germs (in the form of granules) liberated in the plasma, are (like other foreign particles) often taken up by the white cells and endothelium, multiply, grow and at definite dates become free and dispersed by rupture of these cells: the original source of germs would be contagion, and for the relapse either relics of the same or those freshly formed during invasion-attack and liberated at its crisis. Thus regarded, the granular aspect of certain large cells found in blood during, before and after fever, is not due to albuminous or fatty degeneration alone; there being no objection to the surmise that many spores or granules not undergoing development, do become degenerated. Only some of the white cells would thus become the *nidus* of spores, and the vascular endothelium at some places only (commonly of the spleen); the periodic development is a rapid process, and sometimes so sustained as to furnish successive broods of organisms in connection with prolonged febrile attacks.

Further experiment with the blood of subjects free from visible infection and of individuals affected with other diseases, has shown that large granule-cells undistinguishable from the above may be present here too; that is to say, no means are known of discriminating between them.

This conflict of evidence notwithstanding, I cannot lose sight of my earlier views.¹ (*Vide F, Plate I.*)

Maturation and Multiplication.—Indiscriminate measurements of the blood-organisms furnish no help in estimating their rate of growth; during culture, I found the filaments in a very few hours might grow to double or treble their ordinary dimensions, and there seem no objections to admitting a rapid growth in the blood. Increase of numbers may also be prompt, and this from either multiplication by fission or accession of new broods. I considered that the filaments do really multiply by subdivision, this process being oftenest indicated in acetic acid and stained specimens (D, Plate I.): as opinions regarding appearances may differ, I need not add details convincing only to the observer.

the spirillum in fresh blood is very liable indeed to be overlooked upon first or even second inspection. There are no illustrations to this very interesting article, but judging from the description, appearances seen were such as are shown in my figures.

¹ Recently Dr. L. Lübmoff of Kasan in E. Russia (*Centbl. f. med. Wiss.*, No. 46, Nov. 1881) thinks he saw moving spirilla in close connection with the pale cells composing the substance of enlarged Malpighian bodies, in a fresh spleen. The organisms seemed to be attached to the cells, and sometimes more than one to a white corpuscle. The case was one of *typhus biliosus*, in which death occurred with rupture of the spleen; the pulp of this organ being marked by disseminated white spots. Dr. Ponfick (*l. c.*) had not seen such appearances, nor have I; although I long since met with the like in splenic blood of the monkey.

When the spirilla are numerous, their variety of aspect is also commonest, this fact suggesting a varying activity of growth : sometimes they quickly reappear after temporary absence ; and I have found them disappear outside the body, whilst still continuing in the blood, which therefore must have been replenished.

The ordinary duration of individual organisms has not been ascertained. The length of visible blood-infection varies from 2 or 3 to 8 or 10 days ; and probably the longer periods are due to continuous or successive broods of the filaments. That all spirilla have not equal vitality, is shown by the varying periods for which they remain active outside the body, conditions being similar ; and by their varying state after death of the patient. When first seen, though looking alike they are not equally long-lived.

Life-term.—Observation of fresh-blood specimens distinctly shows that the individual organisms have not always either the same or an indefinite duration.

Monkey B. T. at second day of induced fever, t. $105^{\circ}\cdot4$, its blood full of active parasites : oil preparations made : next day—animal's blood still swarming with spirilla ; in the preparations none to be seen, all having disappeared without leaving a trace behind, no change in the other blood-constituents or sign of decomposition. Such an abrupt disappearance is rare, and fallacy is possible from the tendency of the organisms to cluster in one place, which might be overlooked.

Same animal on third day ; P.M. blood full of active spirilla ; specimens made. Next morning—the blood quite free, crisis having occurred ; in the specimens quiescent organisms distinct, and so for three days longer. In another instance, at acme of fever they were distinct and active in preparations, at an hour when they suddenly ceased to be visible in the blood upon final rise of t. to $106^{\circ}\cdot4$.

Phenomena at subsidence.

Experiment.—Oil specimen 2 hours after preparation : the spirilla quiescent, clear and uniform, amoeboid plasmic masses with contained granules, free granules not numerous, red discs shrunken, white cells unchanged ; some protoplasm is vacuolated, as it creeps along it pushes aside the stark spirilla irritating them to brief spasmodic movements, and sometimes it seems to absorb or destroy the spiral filaments. Examination with $\frac{1}{24}$ in. immersion lens : temp. of air 80° F. On second and third days—some spirilla still visible, though faintly seen, plasma vacuolated, its granules are elongating into bacterium-shapes, free granules few : fourth day—plasmic granules increasing : fifth day—same : sixth day—spirilla barely visible, bacteria common and multiplying, some gloeoid masses present : seventh day—the field full of bacteria and micrococci, spirilla not visible.

In another like specimen, the spiral filaments began to grow (?) ; but as wet preparations are not adapted for continuous search, I made more use of dried blood specimens. From these it is learnt that at febrile acme, with the mature spirilla many plasmic particles and filaments like immature forms are seen with sometimes granule-cells, so that the aspect becomes that of splenic venous blood ; soon the spirilla become fewer and disappear ; and then pale cell-forms become frequent, the larger showing a reticulated structure on the addition of acetic acid. Neither aspect of blood nor temperature and intensity of symptoms, are always the same at the end of specific fever, but the following events are usually apparent :—an increase of immature parasitic forms (seen also at beginning of attack), a prompt and final disappearance of the spirillum, the entry into the blood of white-cell forms large and small. This last feature in some

cases was distinctly connected with a simultaneous prompt reduction of the splenic tumour, and the inevitable inference was that the fresh leucocytes and large nucleated cells had come from the spleen. In severe and fatal cases of fever, this secondary contamination of the blood was often very striking. Respecting the presence of free granules at acme, many might be seen in fresh blood, but few in the dried specimens.

Briefly, I infer that at the close of fever the spirillum ceases to be developed, it is no longer produced and appears to come to an abrupt end: its final suppression might seem indicative of an active counter-influence rather than of normal subsidence, but according to my data the end is more likely a natural one.¹

¹ It is well known that in healthy subjects the blood displays a certain intolerance of non-pathogenic organisms (as spores of fungi) and inorganic matters (as cinnabar in fine powder), which are speedily got rid of; but this datum does not here apply. The striking observations lately made in China on the periodic disappearance of the *filaria sanguinis hominis* from the blood of infested persons, offer a nearer analogy. (LANCET, 14 January 1882).

MICROSCOPICAL ILLUSTRATIONS OF THE BLOOD AND TISSUES.

DESCRIPTION OF PLATE I.

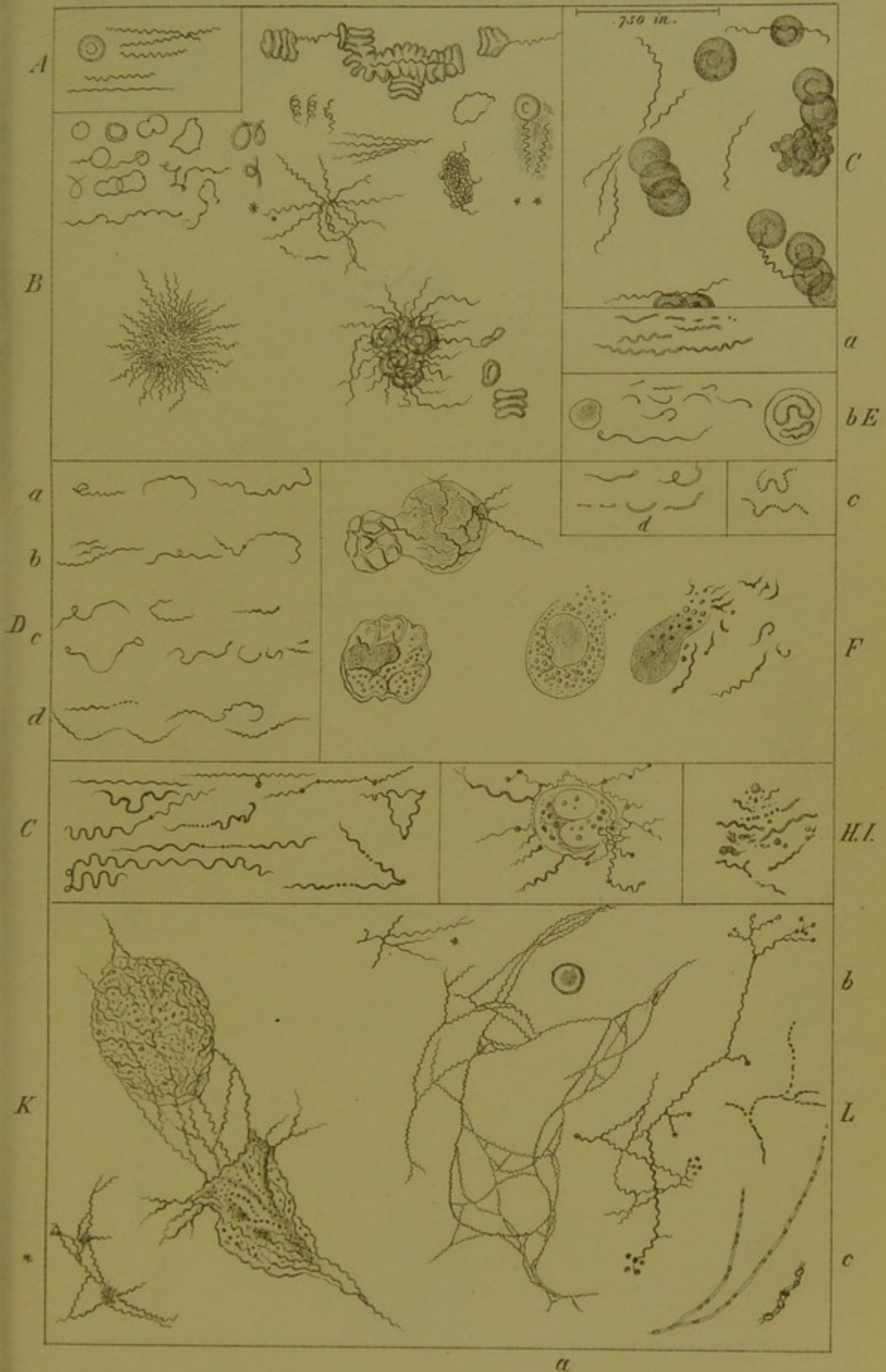
- Compartment A.—The spirilla as seen in blood-serum: a red corpuscle is present; below it is indicated the lengthening which ensues, when the filament is unfolded. $\times 500$.
- B.—Their varying aspects as seen in fresh-drawn blood, and when still active. Shows their multiform contours and aggregations: at * diagram to illustrate the appearance of small knots: at ** spirilla in the corpse, quiescent and with marked twists. Their relation to the red blood-discs is indicated above. Human subject. $\times 500$.
- C.—As seen in dried and stained blood, along with blood-discs; from the monkey. $\times 750$.
- D.—As seen in Acetic acid preparations: the series from above downwards show aspects at specific incubation *a*, during fever *b*, at acme of fever *c*, and at *d* occasional dotting and division. $\times 500$.
- E.—Apparent development of the spirillum: *a* in fresh blood, at 2nd day of 1st Relapse during a temporary decline of temperature: *b* in blood treated with acetic acid, at near acme of invasion (a red and a white corpuscle are added): *c* dotted filaments in blood of a woman dying of cerebral hæmorrhage at close of relapse: spirillum not present, but this blood proved to be infective. At *d* are filaments seen in a case remarkable as simulating recurrent ague: they resembled immature spirilla, especially the two in the upper line. $\times 500$.
- F.—Appearances of development in Splenic venous blood of monkey: to the left two large cells with reticulations visible after the action of acetic acid, granules are present in one: to the right, large granule-cell and granular contents, with immature spirilla attached and free: stained preparations. $\times 500$.
- G.—Hypertrophied state seen in artificial culture of blood-serum, with apparent dotting and division. $\times 500$.
- H, I. H.—To the left: a white blood corpuscle with active spirilla attached in various conditions, from specimen of cultivated serum. $\times 500$. I.—To the right. Apparent development of the spirillum out of free granular matter; from specimen of cultivated serum at 6th day. $\times 500$.
- K.—Aggregated and blended masses of spirilla at 2nd day of cultivated serum: they display a micrococcus-like aspect. At the left corner, a similar granular aspect in another specimen at 6th day, occurring at the angles of a spirillar network.

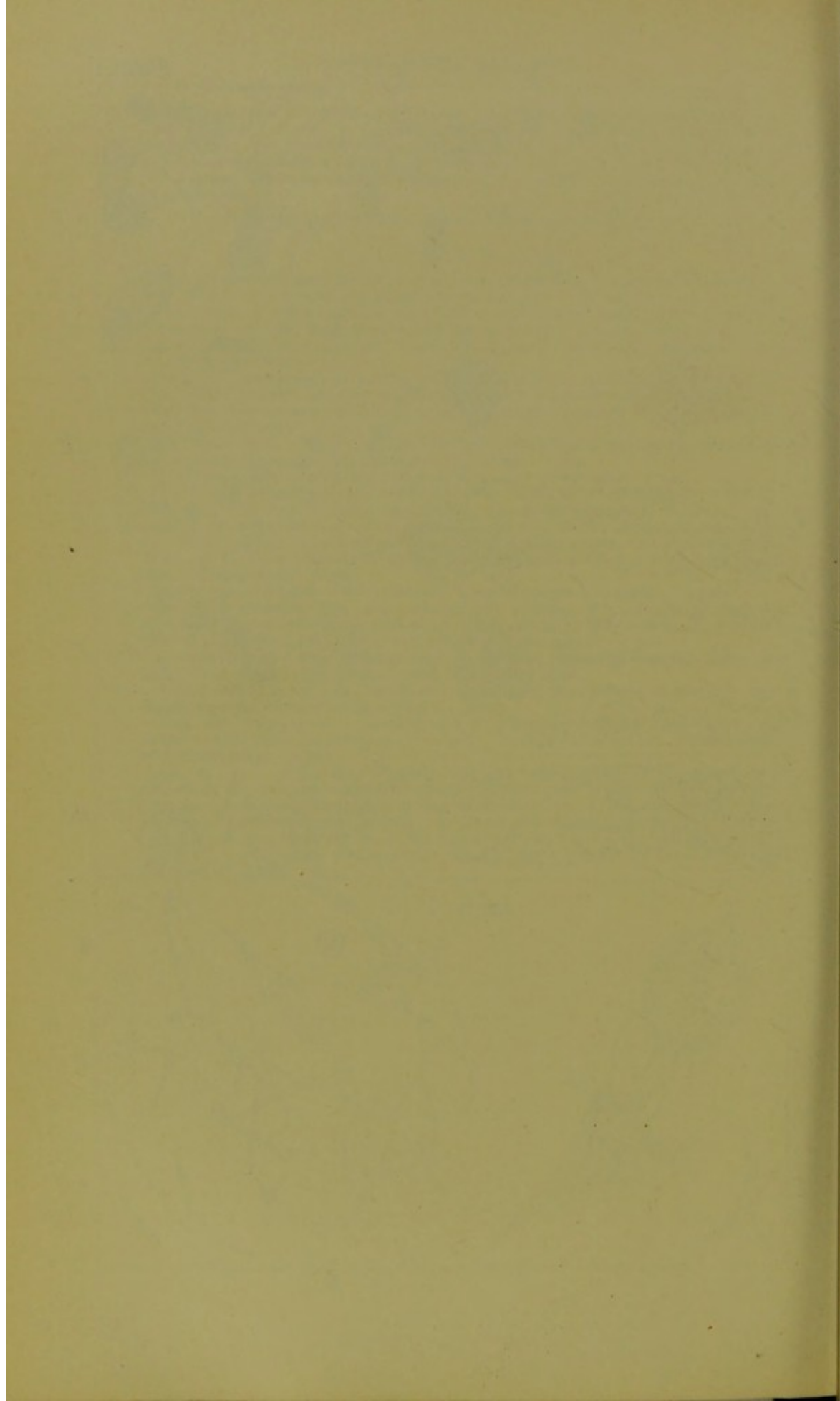
- L.—Another aspect of the spirilla under culture. *a.* Open network seen in warmed serum, after 24 hours. $\times 400$. *b.* Fragment of network in diluted serum, after 24 hours' warming; to show terminal production of bright granules. $\times 500$. *c.* Dotted aspect of filaments composing a network, 20 and 24 hours after dilution and warming of the blood-serum: the lowest figure is diagrammatic. $\times 600$. * To show the appearance of ramification.

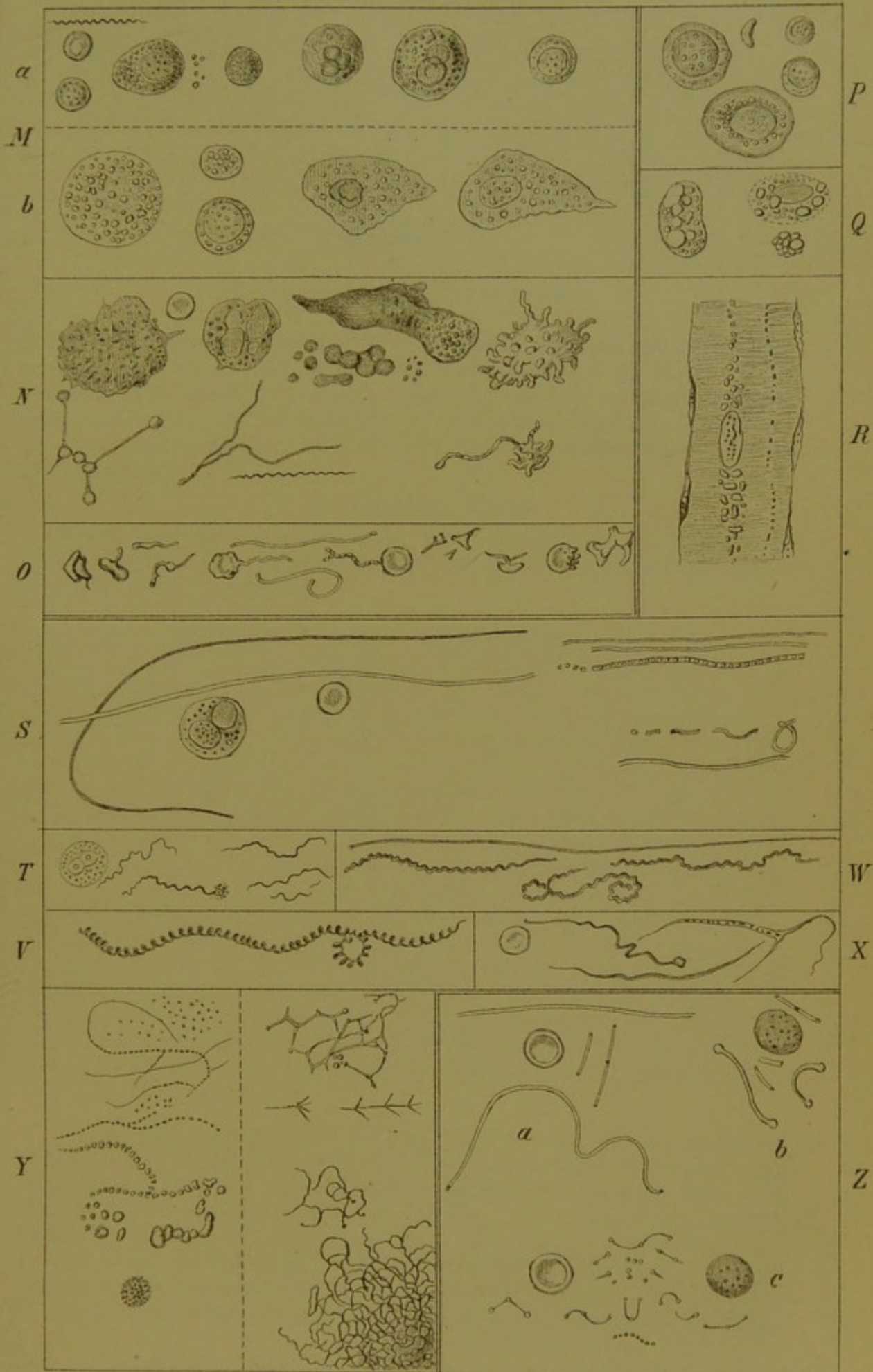
N.B.—The red blood-discs furnish a scale for measurements.

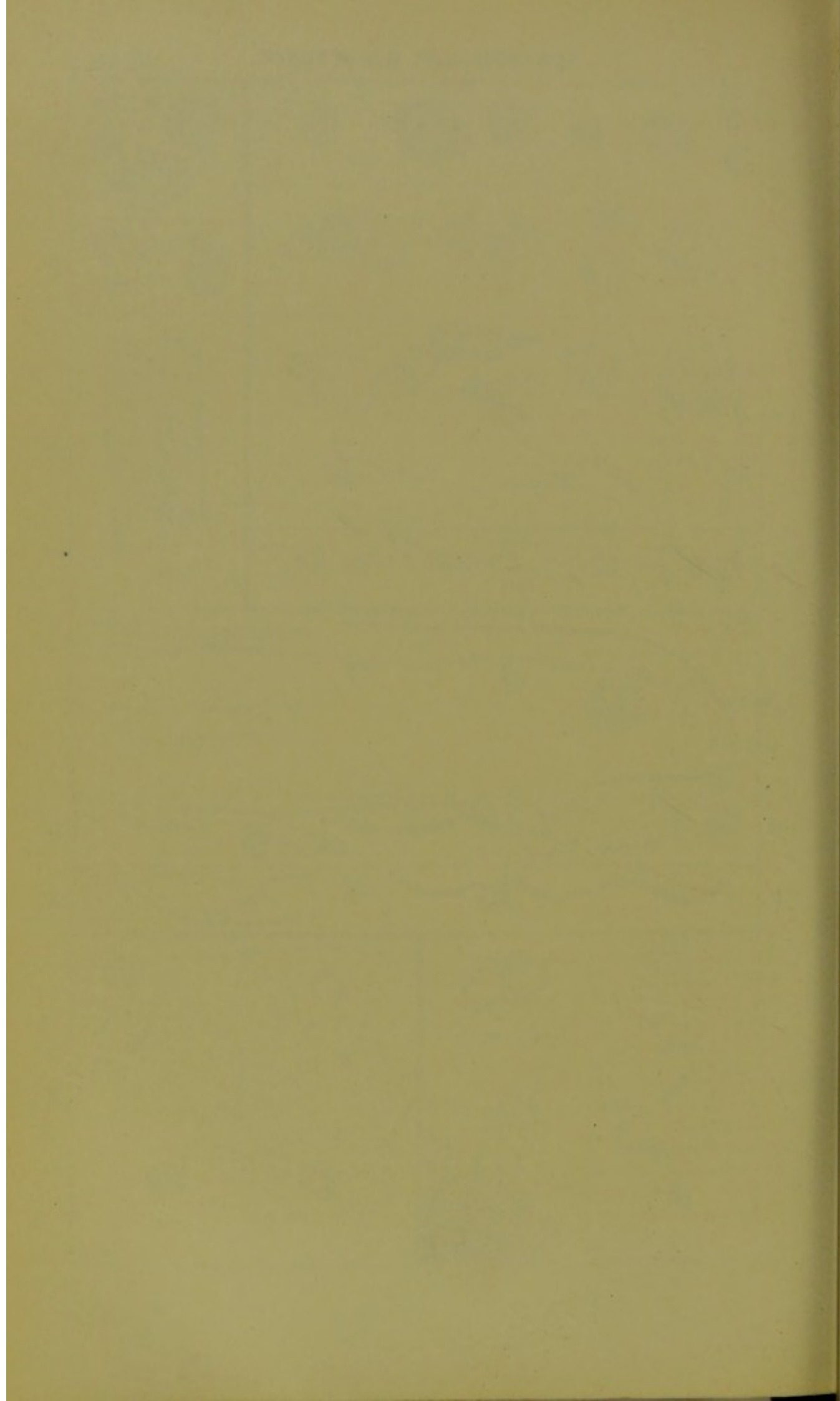
PLATE II.

- M.—At *a* granule-cells seen in the blood about acme of fever, in man and the monkey: they vary in aspect, and sometimes enclose red discs: an ordinary red and white blood-corpuscle, with a spirillum, are placed at the left-hand corner to furnish scale of dimensions. *b.* Very large granule-cells and endothelial particles, from the blood of a girl dying at acme of invasion. CASE XV., page 75: same scale: the granules are usually oil-globules. $\times 500$.
- N.—Forms of free protoplasm seen in conjunction with the spirillum, during fever: all change their contour; some are dotted with granules; others give off long prolongations, which may become free; some are vacuolated. Scale by means of red disc, and the spirillum introduced. $\times 500$.
- O.—Red blood-discs in fever, showing their occasional aspect, and forms assumed by hæmoglobin liberated: some forms are filamentous. $\times 500$.
- P.—Lymph cells from thoracic duct, after death at acme of invasion. $\times 500$.
- Q.—Cells loaded with oil-globules, from the spleen: to the right + acetic acid. $\times 500$.
- R.—Muscular fibre from the heart, + acetic acid: granular degeneration. $\times 500$.
- S.—Bacillar filaments from the blood, seen 5–7 hours after death; the larger ones to the left moved actively; others to the right (above) contain spores: the filaments below these last are from contents of lumbar lymphatic glands, taken 7 hours after death from spirillum-fever. $\times 500$.
- T.—Spirilla from the saliva of fever patients; a mucus-corpuscle is added. $\times 500$.
- V.—*Spirochæte plicatilis*, Ehr. from tank-water at Bombay: it is much larger than the *Spirillum Obermeieri* Cohn, and does not unfold during its movements; its contour is double, and bright spots are seen at the flexures of the spirals. $\times 500$.
- W.—*Bacillus subtilis* ? from drinking-water at Bombay: shows the contortions assumed whilst the contents of the specimen were drying up. $\times 500$.
- X.—*Fungi* ? seen in acetic acid preparations of the blood, freshly made, at Bombay: they sometimes closely resemble blood-spirilla. $\times 500$.
- Y.—Forms and constituents of networks seen in cultivation of Entire blood diluted with *humor aqueus*, under the influence of warmth. Specimen of blood taken at critical fall of spirillum fever. $\times 500$.
- Z.—Blood in Ague. *a.* Bacillary filaments seen after cultivation of blood, taken from a man dying of pernicious ague; and reported as once seen in blood aspirated directly from the spleen of a patient suffering from malarious fever. *b.* Other filaments, termed bacillary, from blood of fever-patients. *c.* Others seen oftener in the blood at initiatory cold stage of ague (regarded as similar to spore-products of *bacillus malariae* Kl. and T. Crud.). \times Oc. 3 obj. 10 Hartnack. Copied from a Memoir by Drs. Cuboni and Marchiafava, Rome, 1881.









CHAPTER II.

ETIOLOGY OF SPIRILLUM FEVER.

THE single cause of Relapsing fever is a specific poison, the nature and properties of which will be most suitably discussed in the following chapter. The conditions under which this poison operates concern either individuals or the community: individual conditions constitute the Predisposing causes; and the common determining agency is that of Contagion.

A. Predisposing Causes.

These comprise the states under which individuals are found, who become affected with the disease; and they pertain to either the subject himself; or his surroundings.

Personal Conditions. 1. *Sex.*—Of 594 patients 488 (viz. 82·15 per cent.) were males, and 106 (17·84 p. c.) females; the respective proportion being 4·6 of the former to 1 of the latter. Such a preponderance of male subjects is not approached in the normal population of Bombay, but it does not exceed that noted in hospital admissions for 'fever' generally; since on account of the prejudices of the native inhabitants, women seldom resort to public hospitals. Indeed, during the late epidemic more females than usual came or were brought for treatment, in the proportion of 27·2 p. c. of all fever admissions in 1877 to 13·2 p. c. in prior years. I have already stated that the cases in females were the more serious. In European countries also male subjects are the oftenest attacked, but there, as in W. India, this predominance is attributable to the circumstance of males being oftenest vagrants or wanderers in search of work. Lastly, I note that though in the list of 68 known instances of accidental infection, only 11 were females, yet there is no anomaly here, since a similar proportion obtains of the total subjects liable to contagion in hospital, whether patients or servants; and, therefore, it would appear that the predisposing influence of sex, if it exist, must yet be slight.

2. *Age.*—Circumstances in Bombay were too peculiar to permit of comparison with the local normal or European medical data, a large proportion of the sick being immigrating male adults, some alone and some with their families; hence the ordinary proportions of age, if they obtained amongst the sick, were not likely to be maintained. In general it may be supposed the ages of most affected would approximate to those of the mass of immigrants, and thus there arose a predominance of young adolescents: of 594 cases, 158 were of the age of 26–30 (or

26.6 per cent.) ; and 472 (or 79.1 p. c.) were at between 16 and 40 inclusive : only 72 (or 12.1 p. c.) were under 16 years, and only 50 (8.4 p. c.) over 40 years. There were 3 cases under 1 year, the minimum age being 5 months ; and 11 between 55-60, the last-named being the maximum age stated. There was a predominance of female patients at ages under 11, and again after 36 ; with a deficiency at the mid-periods, although here a tendency was notable to the maximal ages of males, viz. 21-30 yrs. Other statistical details are contained in Table XIII., page 244.

3. *Race and Caste.*—Of 498 patients the Hindoos numbered 248, Mussulmans 226, Native Christians 21, Indo-Briton 1, Jew 1, European 1 ; and upon comparison with the normal ratio of Races in Bombay, the greatest divergence to be noted here is the large proportion of Mussulmans affected, for this class is only about one-fourth as numerous as the Hindoo section of the community. The explanation is not that they were most predisposed to famine-fever, but that in 1878-9 the numbers of their sick (from N. India chiefly) resorting to hospital much exceeded that of the Hindoos, who, however, predominated in 1877, when immigration from the Deccan was largest : and these comparative data show that the epidemic tended to spread with the arrival of fresh paupers and the invasion of fresh quarters of the native town by them, or by house migration of those already sick seeking healthier dwellings. There was no restriction upon such internal movements, and could be no more favourable contingency for diffusing sickness. Remarks on the other Races attacked are not required, nor additional comment on the several affected castes of Hindoos (see the Chapter on History), which were in sum those forming the bulk of Deccan immigrants and the poorer residents. Personally I saw no case of famine-fever amongst the better fed and lodged Parsee community (numbers 50,000 in Bombay), nor any amongst the 10,000 European inhabitants of the town : there is, however, some other evidence that no race or caste was exempt from susceptibility to this infection, and probably none was *per se* exclusively predisposed.

4. *Occupations.*—The list is a long one, and includes many of the minor sections of the lower classes. During 1877 when the Hindoo immigration predominated, most of the sick were termed labourers, who worked on shore and aboard ships at unskilled though hard labour ; some women were thus employed, but the majority of these kept house : many sick professed to have no occupation, yet very few acknowledged themselves to be beggars. At this date, weavers of the Mussulman race were few : cooks, barbers, washermen, domestic servants, tailors, shoemakers, embroiderers, and the low caste sweepers, may have been in common contact with the sick, though special evidence of contagion here was seldom clear : cart and cab drivers, grooms, mill-hands, carpenters, bricklayers, petty shopkeepers, hawkers, gardeners, painters, firemen in steamers, sawyers, dyers, butchers, police sepoy, and courtesans are named. During the following year when Mussulmans were oftener admitted, the majority of the sick were weavers of cotton or silk coming from Upper India ; yet many occupations, temporary and unskilled, were taken up by these and other persons ; and there then

occurred a large influx of low castes from Kattiawar, who also did any work entrusted to them. During 1879, the range of occupations was still considerable, amongst even the 50 sick at the G. T. Hospital, labourers and weavers predominating ; the evidence being thus complete of the general diffusion of infection, and hence of the non-predisposing influence of mere occupation. At this hospital some of the earliest and latest sick seen, belonged to the scavenger community employed by the Municipality, and it might be supposed that their unclean employment, joined to bad lodging and sometimes bad food and habits, would specially predispose these individuals to disease ; yet if so the demonstration was imperfect, and I think the most exquisite samples of spreading infection were seen elsewhere as well as here. Upon several occasions more persons than one were admitted, at the same time, who were similarly employed and worked together ; as, for example, young inmates from a Reformatory, hands from a large cotton-mill, or from private establishments of silk or cotton looms ; but in such cases the spread of infection seemed referable not to the character of employment, so much as the aggregation of workers.

5. *Habits*.—Intemperance was not so common a vice amongst patients, as to allow of its predisposing influence (if any) becoming apparent ; and the number of sick persons indulging in alcohol, opium or hemp, or in debasement generally, did not appear to exceed that noted of patients suffering from other diseases. Occasionally it was stated that relapsing fever came on after a debauch ; but nothing was then learnt of the attendant circumstances, tending to show contemporary increased predisposition to infection.

6. *Destitution and Bodily Fatigue*.—The connection of spirillar infection with individual want, was often so evident as to become remarkable, and a few times it seemed as if fever might have arisen spontaneously amongst the destitute ; yet it was never known by competent observers so to arise, and statements of the sick as to their personal movements at times and in places where famine-fever might be lurking, were not always to be depended on. Besides, the fact of so large a proportion of town residents (who were not starved) becoming affected, negatives the idea that bodily want was very influential in the production of the disease. That exposure to much heat or wet, with or without excessive physical exertion, had a special predisposing effect, did not appear upon review of all the data ; however striking some individual instances might seem.

7. *Mental Anxiety and Depression* had not probably a decided, or particular, influence in the causation of this disease ; it being allowed that all depressing agencies may have exerted their usual effect, to the full, during the late events at Bombay, and that the type of actual fever may have been modified thereby.

8. *Previous Illness*.—The interesting series of cases observed at the J. J. Hospital, of sick persons in the wards becoming affected by the spirillar contagion, presented an opportunity of considering if pre-existing disease does or does not favour this infection ; and I am led to infer

that the evidence did not indicate a particular influence in such cases generally. Bombay was not a suitable field for noting the possible predisposing effect of ague, on a sufficiently large scale ; only if individual instances be regarded, there were some indicating such result of previous malarious infection.

9. *Idiosyncrasy*.—Of three workers long employed together daily, at hospital, in examining the blood of fever patients, and all in fair health, only one became ill with relapsing fever. Again, while in spite of innumerable contacts with the sick, of temporary impairments of health and frequent great fatigue, I remained unaffected until inoculated by accident ; on the other hand an adult ward servant, well nourished, a few days after being taken on as extra hand at the G. T. Hospital, began to show characteristic symptoms, without the suspicion of any inoculation. Instances of such diverse import might be multiplied greatly ; they are well known to happen in all contagious affections ; and opinion regarding them varies, there being no means of testing personal idiosyncrasy apart from its supposed effects. Among closely associated persons, *e.g.* of a family or occupants of a common chamber, when contagion entered it was rare that any escaped illness ; and this fact indicates the subordinate rôle of idiosyncrasy, apart from any acquired predisposition.

Conditions pertaining to the surroundings of patients were as follows :—

1. *Climate*.—Upon comparing the late experience at Bombay with records of epidemics in Europe—*e.g.* in countries reaching towards the Arctic Circle, it becomes difficult to conceive that even great differences of climate exert a fundamental influence in the production of spirillum fever.¹ Nor is this statement modified by accepted views of the climatic limitation of typhus proper.

2. *Season of year*.—The dates of fever admissions at the J. J. Hospital are shown in Col. E of Chart 1 ; and there it is seen that in 1877 most cases were entered in May and September, and fewest in the cold months (December) ; during 1878 the maximum was in April, and the minimum towards the end of the year. From local acquaintance with almost all these instances, I am enabled to state that at no time could the predisposing influence of season (if such exist) be eliminated from more direct influences, and especially that of successive immigrations of paupers from various quarters of the Presidency. Cases of hospital contagion occurred, as a rule, when the medical wards were most overcrowded. During 1879, at the smaller hospital, 10 admissions were in the hot season, 11 during the rains, and 29 in the ensuing cool weather ; and here, also, the detection of a house-epidemic and the prompt transfer

¹ The clinical characters of relapsing fever at St. Petersburg are practically identical with those seen at Bombay ; yet these two cities are separated in N. latitude by 40°. There the mean annual temperature is 58° F., at Bombay 80° F. ; and the disease flourished during winter with a mean temp. of 14° below freezing point, or at least 70° below the corresponding mean t. at Bombay. The race of people is diverse (though apathy is a common characteristic), and their diet (though chiefly vegetable) ; but a common feature is the crowding of the poorest classes in badly ventilated buildings, and overcrowding during the initiation of the fever-epidemic in both localities seems to have been alike excessive. This fever has not left the northern city since its first date of 1864-5.

of sick to hospital, served to raise the admissions, which but for such incident, might have remained low. Under these circumstances, useful comment on the possible direct influence of temperature and moisture changes in the air, becomes impracticable. In Europe, 'season of the year has little influence.' (Dr. Murchison.)

TABLE XV.—SEASONS. FEVER ADMISSIONS, APRIL 1877 TO DECEMBER 1878, J. J. HOSPITAL, BOMBAY.

Cold season	Hot and dry	Rainy	Hot and damp
December . . 20	April 68†	June 68	October . . . 43
January . . . 10*	May 78	July 46	November . . 15
February . . . 21*	—	August 65	—
March 22*	—	September . . 53	—
Totals . . 73 146 232 58
Grand total . . . 509			

* For one year only.

† For part only of April, 1877. Remaining months for two years.
Maximum monthly admissions 78—during May (two years.)
Minimum monthly admissions 10—during January (one year.)

3. *Soil*.—This subject being of special interest in connection with the possible co-production of malarial and spirillum poisons, the following details are subjoined ; and reference may be made to Chapter II. on History, for a brief description of the island of Bombay.

Quarters of the town chiefly implicated in 1877–8, whose geological character is alluded to in the Municipal Commissioners' Report for 1875 :—

- A. Southern point of the island—low, little overcrowding : structure of trap rock, calcareous sand and clay shale ; famine-fever rare.
- B. Eastern foreshore—high, overcrowding in many parts ; structure of hard trap and trap breccia : fever very prevalent.
- C. Western side—low and densely peopled ; calcareous sand, littoral concrete, in parts trap rock and tertiary clay slate, and marine alluvial mud : fever very prevalent. The adjoining foreshore is, like the eastern, high and formed of hard trap ; but the inhabitants are chiefly Europeans, live in detached houses and did not suffer from fever.
- E. Further north on the eastern side, is a somewhat raised though inclining area, densely peopled, and formed of trap, shale and marine alluvial sand : here the fever was very common.

The instance of B as contrasted with C and E seemed, in my opinion, conclusive that geological composition, elevation or sub-soil drainage, were not concerned *per se* in the production of the late epidemic.

4. *Locality of Residence*.—No fact was clearer than the limitation of the great bulk of sickness within defined quarters of the town ; and undoubtedly it may be inferred that dwellers in these localities were

especially liable to contagion. As such liability arose solely from the increased chance of contagion, this topic is more fully alluded to below: there was nothing apparent in the places themselves to predispose to relapsing fever.

5. *Period of Residence in Infected Localities.*—Of 442 cases seen in 1877-8 at the larger hospital, 42, or 9·5 per cent., were said to have been in Bombay less than a month, and of these 14 (3·2 p. c.) less than five days and 39 under fifteen days; the earliest of these dates, at least, pointing to occurrence of infection prior to arrival in the town. The majority, however, of hospital patients had been some time resident before becoming affected; thus, 136 (or 31 p. c.) named 1-3 months, and altogether 220, or 49·8 p. c., had resided for various periods within 12 months. The large residuum of 168, or 38 p. c., had lived in the town even longer than this: thus 75 (or 17 p. c.) named from one to three years, and no fewer than 68 (or 15·4 p. c.) upwards of five years, in some cases mentioning 20-30 years, or even longer periods, without necessarily being town-born.

These data rest upon the patients' statements, being probably least uncertain as regards the shorter periods named; below they are arranged like Dr. Murchison's statistics of the London Fever Hospital; and upon comparison I note the imported character of the Bombay epidemic becomes strikingly apparent, for whilst in London 86·6 per cent. of patients had been resident longer than 12 months, at Bombay the proportion was only 38 p. c.

TABLE XVI.—LENGTH OF RESIDENCE IN BOMBAY ON ADMISSION.

Under 15 days	.	.	.	39	or	8·8	per cent.
„ 3 months	.	.	.	139	„	31·4	„
„ 6 „	.	.	.	52	„	11·7	„
„ 12 „	.	.	.	32	„	7·2	„
Over 1 year	.	.	.	168	„	38	„
Unknown	.	.	.	12	„	2·7	„
Total	.	.	.	442		99·8	

Whilst the figures also show that residence in infected localities induces a liability to attack, the absence of uniformity in ratio of attacks according to length of residence, indicates that the element of time is not here essentially concerned; and I should interpret the above percentages as properly corresponding with those of the entire population during the years 1877-78, when large and repeated immigrations took place. From this point of view the ratios of sickness under notice become intelligible, for the numbers of patients seen must in chief measure depend upon the number of persons contemporarily liable to infection.

6. *Overcrowding.*—Infected localities proving to be those whither pauper-immigrants most flocked, habitual and excessive overcrowding of apartments and whole houses may be said to have become the rule; and from personal inspection, I know it was so generally prevalent that the difficulty arose how to account for any member of a household

escaping illness. In a practical sense, overcrowding was inseparable from some degree of destitution; and during the height of the epidemic these influences could not be rigidly estimated apart. Moreover, whilst the term in question strictly means an insufficiency of air; yet it also implies the massing together of people, the concentration and accumulation of noxious effluvia, bad hygienic conditions and resulting unhealthiness. Owing to this complexity of conditions, the influence of overcrowding as a potent predisposing cause of spirillar infection, is open to varied interpretation; and knowing that such influence of itself has not at Bombay during past years produced so highly specific a disease, I cannot but conclude it latterly operated most injuriously, by entailing close contact of the sick with the still unaffected. Obviously, this inference does not exclude a certain indirect, and less understood, favouring of infection; such as is admitted with regard to some other zymotic diseases.

B. Contagion.

The determining condition of illness being an effectual entry into the system already predisposed, of the specific febrile poison; there remains to consider the evidence illustrating this process, which is variously termed 'contagion' or 'infection.' It appears to me that by this means alone is the spirillar infection acquired and propagated, and that through its operation the late epidemic at Bombay arose.

The evidence now offered was procured by myself, with the aid of a few Native students; it might have been much more considerable had time and other aid permitted. The data are arranged as follows:—

1. Inference from identification. 2. Deductions from the number of town-residents who were affected. 3. Results of house-visitations and family histories. 4. Particular instances of disease acquired in hospital.

Whilst each of these items is open to separate judgment as valid evidence, all are connected enough to stand together; and their conjoint testimony seems to me both sufficient and conclusive.

1. Argument from Identification.—The spirillum fever of Bombay being clinically identical with the relapsing fever of Europe, accepted etiological inference respecting the latter should apply also to the former. The identification would still hold good, were the evidence of contagion at Bombay either scanty or not forthcoming (the presumption then being that observation was defective); but this is not the case, and in every particular experience in the East tallies with that of the West.

Collateral testimony furnished by comparative experiment is also emphatic, Dr. R. Koch having promptly confirmed my positive inoculation-results on receipt of intelligence sent to Germany ('Deutsch. Med. Woch. schr., No. 25, June 21, 1879).

Local modifying Influences.—Regarding the testimony following, it is desirable to state that the pauper population of Bombay chiefly consists of Hindoo and Mussulman races, who do not intimately associate in their dwellings or social customs; and the larger Hindoo section is again subdivided into castes which also maintain a definite isolation, the inferior grade living wholly apart from the rest. In the same house there

may reside people of different castes, or even of different race ; and except hospitals, there are no Asylums or Refuges indiscriminately resorted to by the whole community. Hence the operation of contagion tends to become more restricted here, than in European towns inhabited by one race practically undivided in the mass. The spread of miasmatic diseases like the Malarious, and also of Cholera or even Small-pox, would not hereby be so essentially modified as that of Relapsing fever ; the propagation of which whilst practically inevitable in a family, was beyond domestic limits certainly contingent. Owing to the people being often reticent towards Europeans and not always exact in their reckoning of dates, difficulties arose in the collection of verbal testimony. The sphere of enquiry was too large for a single worker to get more than samples of evidence. Lastly, the possible interfering influence of a tropical climate and of concurrent malarious infection, has to be taken into consideration.¹

2. Argument from apparent Origin and general Course of the Epidemic.—Difficult as it may be to prove a negative, there is in the present instance every reason to conclude that the late relapsing fever was practically unknown in Bombay prior to 1876 ; for current opinion is to this effect, and I have looked through the records of the same hospital as that where my recent experience was mostly acquired, without finding any clear evidence to the contrary. Even if it could be shown that there had occurred in some previous years sporadic instances of the fever in question, this circumstance would by no means invalidate the conclusions I am about to draw from late experience, regarding the extent to which persons resident in Bombay prior to 1876 became affected in 1877 and '78.

The data available for analysis commencing in May 1877, reach to December 1878 ; and desirable as it was to have the facts concerning the beginning of the epidemic in October or November 1876, I do not think their being wanting affects the present results, for by far the larger period of the epidemic, including its termination, has actually come under notice.

I find already amongst the earlier hospital patients several residents of even long standing, and others who had been in Bombay 1, 2 or 3 years before being attacked with this fever : thus, out of 37 discharged in May '77, no fewer than 18 had lived in the town from 1 to 30 years, whilst the remaining 19 affected individuals were immigrants of a date ranging from 2 hours to 10 months. Now, as it is wholly impossible that the residents were attacked in this number and proportion before the latter half of 1876, no other inference can be made than that of their becoming affected from some special influence of present date ; and I am of opinion that the details with case described at pages 9-11 of a woman who was brought from the Deccan Railway Station direct to hospital, ill with the fever and her blood charged with the parasite,

¹ I was informed by the Health Officer of Bombay, Dr. T. S. Weir, that local 'cattle plague' did not seem so highly contagious as in Europe. Small-pox and measles, however, spread very promptly, and cholera is probably never quite absent. Enteric fever is comparatively rare, and I have not heard of it as an epidemic. Typhus is supposed to be absent altogether, but skilled enquiry has yet to be made regarding the existence and actual extent of both these last-named fevers.

sufficiently show what that influence was, which disseminated by contagion speedily reached persons of all periods of residence. That immigrants themselves suffered most, is clearly explained by their known tendency to herd together ; and also by their miserable condition predisposing them to attack, when not already affected.

Next, I note that residents of both the main classes of the native community, were implicated in very nearly equal proportions ; thus, out of 402 consecutive cases of spirillum fever 109, or about one-fourth, are classed as residents, and this ratio holds good equally of Hindoos and of Mussulmans. Add that both were of the same low social status, or but little raised above the condition of the immigrants who flocked into their midst ; and it will be apparent that the resident poor were subject to some uniformly acting agency. Further evidence to this effect is derived from analysis of the localities in Bombay inhabited by sick residents and sick immigrants ; these I find to be exclusively the same, and some districts ordinarily furnishing many 'fever' patients were almost or entirely spared. As examples, I may mention Khetwady whence 18 immigrants and 5 residents came for Hindoos, and for Mussulmans Nagpada with 26 immigrants and 12 residents admitted into hospital. There are, of course, seeming exceptions amongst the numerous minor localities ; but some at least of these are explicable from local knowledge already acquired, and others must be put down to the vagarious movements and isolated positions of many homeless and sick immigrants, who can hardly be said to have 'lived' anywhere in particular. *E.g.* Two of the four patients named in the Inoculation-series quoted below, were picked up in the streets by the police ; and both the others are also entered as being without fixed residence in the town.

Being personally acquainted with by far the majority of cases under review, and having their written records before me, I may further observe that the course of the epidemic is calculated to furnish other confirmative data : thus, we find that, on the whole, most sick residents were seen during those months when most sick immigrants were admitted. For Hindoos, these periods were November '76 and from May to July '77 ; for Mussulmans they were December '77 and April and May 1878 ; these dates corresponding to those of the main influxes of famine-stricken persons sufficiently closely to demonstrate, as far as is possible, how closely allied in time was the implication first of immigrants and next of their settled fellow caste-men and neighbours. Further, with decline of immigration, residents became less often affected ; but the last instances seen were usually resident Mahomedans. These facts must speak for themselves, yet I would invite special attention to the circumstance that whilst Hindoos and Mussulmans here live in tolerably close apposition and in habits do not materially differ (so far as concerns the subject in hand), still the two classes were not simultaneously implicated with regard to their resident population ; the distinction in time being such as of itself points to other than so-called 'local' influences—malarious and the like, being solely at work at these periods of public ill-health.

Such are the J. J. Hospital data, 1877-78 ; at the G. T. Hospital,

1879, of 43 outside admissions 21 were immigrants and 22 residents; and it is further shown here that when once the fever is introduced, the resulting house- or street-epidemic may implicate residents only or chiefly. There is no way of accounting for this fact except by supposing the sickness to be communicable, at brief successive periods; and the working of contagion furnishes the sole adequate explanation known to me.

Should it seem that the above remarks are founded upon only a narrow basis, I can only express my regret that the vast field of observation lately offered in Bombay, could not have been better explored by more enquirers and for a greater length of time; but *equidem verbo* I would state that my data are really typical, and have been rigidly scrutinised. They are capable, too, of furnishing minuter evidence of the operation of contagion; for example, in the seeming recrudescence of disease amongst groups of residents contemporarily with a fresh local influx amongst them of famine-stricken poor; and, besides, in all this sort of proof, it is not general evidence so much as particular instances which will carry most weight.¹

3. Argument from the direct evidence of individual instances.—This heading includes cases illustrating the operation of contagion in spirillum fever, such as are usually regarded as demonstrative in infectious disease.²

The examples may be arranged as follows:—A. Spread in a family. B. Spread in a house. C. Hospital experience.

A. FAMILY SERIES.

Few facts in the natural history of the Bombay fever, earlier claimed attention than the frequent occurrence of the disease in several members of a family; and in no considerable collection of cases which I have had access to, has this feature been wanting. Thus, in the famine-camp of April 1877, in the medical wards of the J. J. Hospital 1877-78 and in those of the G. T. Hospital 1877-79, such testimony of contagion was to be found so often that I am unable to allude to every instance; and, while selecting some of the completer examples for illustration, I would express my conviction that the spirillum fever tended invariably to spread amongst families situated as were those of the poorer classes of the native population here, during the late famine crisis. Many patients, it is true, were solitary individuals, and probably caught or propagated their disease in a casual manner; but as regards the associated, the mode of origin and spread could not have been one of mere accident. Direct evidence of the successive attacks in members of a household may be difficult to obtain from native reticence or appre-

¹ Epidemics in European towns where there exists no suspicion of famine, are universally attributed to the spread by contagion of imported disease. At such times the proportion of residents affected varies considerably, according to local circumstances: compare Table XV. above and remarks, with Dr. Riess's statement (*l. c.*) that at Berlin, 1868-9, of 77 ordinary cases 20 were tramps, 35 persons of unsettled home, and 22 town-residents.

² The printed Official Forms have a column for 'locality of residence,' and to this added information of birthplace and length of time in Bombay.

hension, and from a certain waywardness or obstinacy which sometimes interferes with full investigation; yet indirectly the manifestation of contagion will not be obscure, and were I to include all instances of family series named in the total of 650 cases, my list would become a very long one indeed.

Families often include other relatives than parent and child, and such complicated examples being proportionately difficult to follow out, except upon hearsay, they are mostly excluded below: I will, however, mention what was said, with every appearance of veracity, in these selected illustrations. A family occupies one room; the following instances, therefore, are examples of tenement ('stuben') epidemics: see also B below.

1. Family of Luximon Govinda: husband, wife and son, famine-immigrants lately resident at 'Dongri,' a well-known fever-haunt: admitted together in May 1877. The husband ill some time, was emaciated, delirious and reduced to the typhoid state, dying after 48 hours: the wife exhausted, jaundiced and also delirious, just as occurs at the end of the chief or invasion-attack, quickly rallied; but 9 days after admission had a brief though short relapse (spirillum in the blood), 11 days afterwards and again after other 11 days, she was attacked with a febrile paroxysm (seemingly non-spirillar) which left no permanent ill effect. The son, aged 12, had been ill a still shorter time, namely 4 or 5 days, his temperature was 105° , he was delirious and his blood was charged with the parasite; next morning the critical fall took place and apyrexia ensued, lasting for 7 complete days; there then occurred a short but typical relapse (spirillum), and subsequently convalescence. Here there was an obvious successive order in the attacks; only that of the husband being too far advanced for specific demonstration.

2. The Anniabas. Four brothers also famine-immigrants recently from the Deccan, were admitted on sequent dates in June and July 1877; all lived together, and it is reported there were two other strangers in the same 'chawl,' who had suffered from fever and had returned to their native place. The cases were quite typical, all showing the spirillum, and on comparing the dates of pyrexia, which Mr. Sukharam Arjoon was careful to enter at the time, I find that the first attack of the second and third man was contemporary with the relapse of the first, while first attack of the fourth man coincides with the relapses of two and three: and our decided opinion was that the fever was communicated from one to the other in evident series. The Marooti family seen in June and July 1877, also of four individuals, was a similar instance.

3. Hari family: husband, wife and two young children. Natives of the Deccan and resident prior to the famine in Khetwady, whither flocked many immigrants from the desolated upland plains. The parents and one child had fever on admission in July 1877: the husband's case is mislaid, those of the wife and son show them to have had a specific relapse at about the same date, and very like in the unusual character of a deep mid-cleft (? whence a common source of infection); the infant in arms was unaffected at first, but just after the close of its mother's relapse and 14 days after admission into hospital, it was seized with fever attended with a profusion of the blood-parasite, and the family then took alarm and fled. This baby girl remained by her mother's side on the same bed, and the strong presumption must be that she was infected by her parent.

4. The Gopal family of seven persons, viz. parents and five young children, famine-immigrants, were admitted together in September, 1878: three of them had 'fever,' viz. the husband and two children named Seeti and Sawalia, the wife had just recovered from a relapse: her little son Bhao, and the infant at breast became affected 4 and 3 days respectively after admission: the remaining eldest daughter, aged 11, was not seen to have the fever (there is some doubt if she had not before had it). On comparing notes of the cases now before me, it seems most probable that the mother infected all the rest, and that their first attack began with or soon after her relapse; and as it is known that two of the children (one a suckling) were well upon admission, speculation as to the source of their disease does not seem difficult. As regards the

existence of some special 'local' influence in their home, the place was visited at my request and report made that with the exception of two women of the same low caste, from the same village and in close friendship with this family, no one else had fever in the house; these women suffered at brief intervals a short time previously, and the adult patients volunteered their belief that their family was infected from them. I could find nothing to contravene such opinion, and may add that while this young family was in No. 10 ward, the nurse and a native medical clerk simultaneously acquired the same fever, both dying shortly afterwards. The husband in another ward suffered very severely, yet strangely the children very little. It is true there was another woman in an adjoining bed to the Gopals, in No. 10 ward, who was convalescent from spirillum fever; but on the whole it seems far less probable that she, and not the huddled children, was the source of infection of the two attendants.

5. The Arjoon family of father, mother and three young children, entered hospital with ague acquired in a notoriously malarious district (the Toolsi lake) during the early part of the rains 1877: the father's case I am now unable to trace with certainty, but the mother 19 days after admission was attacked with veritable relapsing fever: her case will be afterwards quoted with reference to contagion in hospital, and I now mention that her suckling infant was seized with the same ailment just after the close of her relapse, or 1 month and 10 days after their resorting to hospital; it died on the 7th day, and I will leave the question to be considered by others how this came about, and also whether or not a family might be successively involved outside hospital as well as within.

6. Members of the Vittoo family likewise famine-immigrants lodging in a recognised fever-haunt (Dongri) gave the following account of themselves, the father first suffered outside hospital with fever; his two sons and the wife and child of one of these young men, then came for aid (August 1877): Narayan V. has spirillum fever, and so his brother Rowji V. whose wife was previously to him attacked and whose child of 10 months was afterwards affected.

Further separate enumerations would hardly add to the force of the above examples or I might allude to the Dhondi family of father and two sons; the Deobas husband, wife and two children, and the families of Rowji and Suntoo.

The notes are before me of Wamonrao Bala and his wife, a middle-aged, childless couple; she first had the specific fever with relapse and recovered; he was admitted in a state of great debility and 5 days afterwards underwent an attack which left him at death's door, and in that state he was removed by his friends. Whether or not this was a relapse may be questioned, but the event occurred subsequent to his wife's illnesses, and at least may have been connected with them otherwise than by mere sequence.

There are several other instances of husband and wife suffering about the same time, and oftenest the wife first, which present similar evidence; and also of other forms of relationship besides those above-mentioned.

The series above enumerated belong to the Hindoo community, and in order to satisfy requirements I now mention a few instances met with amongst Mussulmans; who none the less have furnished the same kind of proof of contagious influence.

1. The Bala family.—In August 1877 a woman and five of her children were admitted into hospital with fever, and it is stated that her husband (Sheik Mussulman) was suffering from the same; his case is not with the rest but to the best of my belief he recovered, and I saw him waiting upon his son Mahboob who died in No. 12 ward: the other records are before me. Sherifa, the mother, a very thin, enfeebled and gaunt-looking woman had manifestly suffered of late from some severe illness; she had no fever in hospital: the five children all displayed the blood-spirillum, so that the nature of the case is beyond doubt; three probably had the relapse in hospital, two probably the first attack: two died, namely the eldest and the infant youngest. The whole series was carefully scrutinised at the time and noted as evidential of a common source of infection (doubtless the mother) for some individuals, and also of successive infection for others: as entire proof of these statements would require the publication of each case and chart, and an analysis of undesirable prolixity, I will

offer in part verification the report drawn up for me by a very intelligent native friend, who was quite competent to undertake the task. It is dated the 16th August, '77, and runs precisely as follows :—

The woman states that she is an inhabitant of Sattara. Her husband is a copper-smith. As they were reduced to the utmost destitution, and were starving there, they left Sattara about two months ago (according to her relations' statement on or about the new moon of 'Jesht') and removed to Poona, where her husband Ba'abhai, and her eldest son Mahboob, who is about sixteen years of age, left her to take care of the children and came to Bombay. She remained at Poona for some time (about one month and five days) supporting her children by grinding corn on hire and doing other jobs. Fifteen days after her arrival in Poona she was laid up with fever, where she remained ill for about a fortnight, when her eldest son Mahboob, hearing of her illness came from Bombay and removed her with the children from Poona.

She was at that time delirious or in an unconscious state, and was brought by her son to Bombay by the G. I. P. railway train.

They put up in Bombay in Peeroo Havildar's Street with a relation of theirs, who is also a copper-smith, and who supported them all for some time. A few days after her arrival here, her husband was attacked with fever. She does not know how many days ago as she was at that time delirious. Shortly afterwards her eldest son, Mahboob, was also laid up with fever.

He was followed by his sister Anvarbee, who is about eight years old, and is lying in the hospital at present in a state of collapse.

After Anvarbee her younger sister Nyazbee, a girl about 6 years of age, was also laid up with a similar complaint.

Both of the above had it on for some days (about a week or ten days according to the mother's statement) when the fever left them for some time, but has relapsed again about a week ago.

The next one attacked is the woman's eldest daughter, who is about 20 years old and is an idiot.

About a week ago her son Dada, a boy aged about 10 years, was also similarly attacked; at this time his eldest brother Mahboob, whose fever had before left him, had a relapse with chills.

Last of all Ellahi the infant at breast got it, and is very ill as yet.

She has been in Bombay since the last month. Her eldest son Mahboob and her husband are, according to her statement as yet ill, in Peeroo Havildar's Street. But on enquiries being made of her relation at whose house they had put up, he states that the fever has left both father and son about 3 days ago.

The house where the whole family have resided is not in an unhealthy locality, and though not very cleanly kept, has no offensive smell about or around it. The ground where they slept is a little damp.

None of their neighbours or other members of the same house have or had any fever. They look perfectly healthy and robust.

No one, not even the relations at whose house this family had lodged for the last one month, had associated or eaten with them, nor did they sleep in the same room.

Their food while in Bombay consisted of rice, dhal, and some vegetable; mutton or any other sort of meat they could not digest.

Their appetites have become abnormal, and they (especially the women) crave for food. She and her family have a peculiar musty and very offensive smell about their persons.

They are very much reduced in flesh, have vacant, stupid and hungry looks: they do not like to be questioned, and their temper has become a good deal irritable, and their mind somewhat deranged.

The whole family is in a state of the utmost destitution.

2. The Ahmed family.—Was composed of 8 persons, viz. parents, 4 sons and a son's wife and child: the old people and 2 younger sons were admitted together in May 1878, and prior to this the young wife, the old mother and one elder son had had the fever; the last-named dying of it. On admission, the old man and his two lads all displayed the spirillum: he died presently in the stage of collapse and both lads underwent a relapse in hospital, one of them indeed having two relapses: an elder brother, Sulliman (whom I saw well) used to visit his young brothers, and shortly after the conclusion of their relapse was himself attacked at home, and died

there; I had the opportunity of ascertaining that there were many spirilla in his blood. Thus, 7 out of the 8 were about one date known to be affected, and 3 had died upon successive occasions. On reviewing the series, I saw and still see nothing improbable in the supposition that there had occurred a spread of fever by infection from first to last; the mother, for example, communicating her disease to her husband and two younger sons who all suffered about the same date, and it seems likely that one of the latter infected Sulliman the last one to sicken and die. All this is not quite the entire account, though sufficient for my purpose, for there is mention of another relative being fatally attacked, and I have reason to believe that the grandchild died also; the notes distinctly stating, and that more than once, that the whole of a family of 9 were attacked and no fewer than five died; this had all occurred within a period of six weeks. What was the nature of the 'fever' may be inferred from our finding the specific sign in every instance examined during a pyrexial period. Two separate accounts were obtained for me at the house which quite concur, and I will quote from the one by student-apprentice Farrell which gives important items at the end, procured on a second visit:—'There were 8 persons living in the room or rather sleeping in it at night (they are the above-mentioned. H.V.C.); the fever was first contracted by the eldest son's wife, a young woman of good bodily condition who caught it about 2 months ago, it only kept on her for 8 or 9 days; before she was well the fever attacked one of the sons, her brother-in-law, and he succumbed to it after only a few days; it then attacked the woman Amina to-day admitted (the old mother. H.V.C.). The next persons attacked were the father (old man) and 2 sons at present in No. 11 ward, these 3 being simultaneously affected. Of the 8 persons who used to inhabit the one room there are 4 in this institution now; 1 is dead, 1 has been attacked and 2 have entirely escaped, these last are the eldest son who works at some distance from his home and only comes back to sleep (he is the Sulliman who visited his sick brothers in hospital, caught the spirillum fever and died: see above. H.V.C.), and the other is his young son. The room occupied by this family is one on the 2nd floor of a two-storied building, the first floor (he means ground-floor raised on a plinth. H.V.C.) is occupied almost entirely by spinning and weaving machines: the room is a small one about 10 feet square, it has only one window ($2 \times 1\frac{1}{2}$ ft.) which looks out on to a number of dirty gullies and drains; the door of the room faces a closet and at the side of the closet is a small bathroom having in it one pipe which leads up from a drain below and in its course passes through a range of closets situated on the first floor of the house. A boy died on the first floor of the same building about 3 days ago, after having had fever for 7 days.' This was written a few days after the family was admitted, and I sent Mr. Farrell a week later to learn other particulars; he then reports—'In same house Sulliman Ahmed the eldest son was attacked on Tuesday with fever . . . (specimen of blood brought to me and found to contain the parasite. H.V.C.) No one else sick with fever in adjoining rooms, nor in same house nor in any houses of neighbourhood' (*i.e.* the immediate vicinity. H.V.C.)

I am well aware that here is not a rigid demonstration, nor are any of these data offered as so many proofs positive of contagion: they possess, however, a corroborative and an accumulative testimony, which is nearly all that facts of the kind can be expected to furnish. Before proceeding to the next sub-heading, I will allude to experience at the famine-camp of April '77, the notes of the 47 fever cases seen there being now before me.

In these notes there are references to at least 12 families, members of which were successively affected; and although there is only ordinary evidence, and sometimes imperfect, regarding the nature of the 'fever' here, yet I cannot fail to recognise a majority of specific instances. It was notorious that individuals were attacked with 'fever' at the camp itself, and here limiting this remark to families, I saw myself in the brief period of three weeks, the wife and children of Gunnoo Dhondi affected after him (his own father was previously ill); and I also saw three little children of the Tooka family attacked on succeeding days, verifying by the microscope their cases also. As in the abundance of other completer material there is no need to lay par

ticular stress on this interesting and valuable series, I will pass over other details, and proceed to mention very briefly what was noticed at the G. T. Hospital.

In April and May 1877, Gunsha Mahadeo and his son, and the two brothers Amruta (one attacked after attending on the other) are names of related individuals occurring in the list of 40 cases: here, however, were more instances of destitute persons found ill on the public roads, and few of those of families; this circumstance being explicable from the fact of the hospital not standing in the midst of a dense population, but rather on the outskirts of the native town and near to markets and wharves. In 1879, there was the instance of 4 young negroes, brothers and cousins, who successively caught the fever in Bombay, as well as an elder relative who returning from Kurachi in good health (seen at hospital), was seized in less than a fortnight himself, and underwent a typical attack; of 8 men living in one room 6 came to hospital and 2 left the house in fear: here the evidence of contagion was unmistakable. Of 43 patients admitted, 4 only occurred as isolated examples, whilst the remainder were associated in small groups of related individuals: this is a fact significant of contagion, and well illustrative of events at the close of an epidemic.

Intermediate series.—Coming between the above examples of more than one member of a family seen to be attacked, and the next of people not related becoming successively affected, I find a large number of instances of only one member of a family being assuredly known to present the spirillum fever, whilst as regards the near relatives, also seen by us, it is almost certain from the history and symptoms of their cases, that they also had had the same complaint: and there are even instances of a patient admitted with fever only presumptively specific, when it was possible to learn for certain that some relative not in hospital, yet living in the same house, had, about the same time, veritable relapsing fever. The last case may be regarded as completing the chain of testimony which binds together the two main groups of my collection, namely, those of ascertained and of presumptive specific fever; and which thereby renders both almost equally significant.

1. Spirillum fever in a family and in a house.—A Mahomedan woman came in on the last day of the invasion-attack, when many parasites were found in her blood; as there was no definite relapse, no further opportunity occurred of verifying the nature of the fever. Her brother entered hospital 3 days later in the same month of November '78: he was in a state of depression, and the symptoms were those of the apyretic interval following specific pyrexia; his blood did not contain spirilla (as might be *a priori* anticipated), and there was no true relapse, so that hypothetically there seemed room for doubt as to the nature of the fever which he states, without being in a condition to furnish details, to have preceded his low state on admission. Like the sister, he had an eruption of petechioid spots; and if any doubt existed (which was not the case with Mr. Sukharam Arjoon and myself) about this matter, it may be said to have ceased with the admission into hospital of another brother living in their common home, who presented the characteristic signs of spirillum fever.

2. The brothers Kusba were admitted in August 1877, in a critical condition; one of them speedily died in the typhoid state without furnishing direct evidence, in the condition of the blood, of specific infection, and so far his case may be regarded as of doubtful nature; but when the other brother who survived, was found to show the blood-parasite, I could not but conclude that both men were affected with the same disease, and there was nothing whatever to gainsay the inference. The form of fever was that of *typhus biliosus*.

Instances of this kind are of much value in aiding diagnosis, and were by no means uncommon; indeed, had those of presumed specific fever (which are as valid as any described in Works published prior to

the discovery of the blood-spirillum) been added, my lists would be greatly extended.

3. A lad, famine-immigrant like the preceding, was admitted in June 1878 with high fever (said to be persistent) of 10 or 11 days' standing; he had also the symptoms of double pneumonia: on the 3rd and 4th day afterwards the temperature quickly fell to normal, and subsequently to a brief rebound it remained level for the month longer he stayed in hospital. Nothing peculiar was seen in the clear blood upon his admission.

Suspecting from the long duration of the febrile attack, when regarded in connection with the comparatively moderate amount of lung condensation on admission, that the case was not purely pneumonic: I had enquiries made as to family history and residence of the patient, and learnt that he was 1 of 10 lodging in the same room, 2 persons having had 'fever' before him and there being other history of such ailment. Besides, in a room next but one to his friends, was a woman then ill with fever (she was 1 of 4 there), and on sending for a specimen of her blood, I found it to contain the pathognomonic spirillum: all these individuals lived on the same floor, were of same caste, being weavers and working at home together. This instance is a frequent one of patients coming to hospital at too late a stage of their prolonged attack to permit of microscopical verification; yet I felt entitled to draw from it the inference that the boy had probably suffered from spirillum fever acquired by infection, subsequent to which, as is so often the case, lung disease had become developed; and this view seemed to be confirmed by the remains observed on admission of the copious sudamina sometimes attending the critical defervescence.

B. HOUSE SERIES.

Upwards of 40 instances of 'fever' reported in the houses (*i.e.* dwellings) of hospital patients, were enquired into by myself and native assistants chiefly during the months of May, June and July 1878; and the results noted are now before me. The foundation, at least, of this work was valid, for we started with the definite fact of a man or woman being admitted into hospital whilst displaying the spirillum fever; and we visited the dwelling of such patient, taking a series of questions I had drawn up to guide the local enquiry. Sometimes only hearsay evidence of other co-residents being affected was to be had, and very rarely indeed was this wanting; often other sick were seen in the houses, but it was seldom these could be induced to come to hospital, and I suspect the rumour of our post-mortem examinations had operated as a deterring influence at this period. Commonly but two or three house-visitations could be made in each case, and a particular prejudice existed outside as regards taking blood from the finger for microscopic examination; hence from these drawbacks and the inevitable predominance of hearsay entries, the series of instances now under review becomes defective if it be regarded as proof of contagion. With this admission, I cannot lay stress upon the data as likely to be demonstrative to others; but still as corroborative evidence of contagious influence and also for collateral information, I think my notes have a certain value. Most of them refer to spread of fever in a common chamber or tenement; and some to propagation from one room to another in the same house, whenever intimate contact was possible.

In Europe, ordinary testimony regarding the prevalent fever of an epidemic is held as fairly good, and much of the current evidence of contagion seems to be based upon such general statements as 'that fever was in the house' at a certain time: the personal impression of

the physician also is counted as a trustworthy datum, without his being constantly called upon to give details of exact conditions, date and symptoms. And different as circumstances may seem to be in Bombay, I am disposed to maintain (*mutatis mutandis*) that 'fever' during the late epidemic, usually meant 'relapsing' fever in those houses we inspected; and I also hold that our personal impressions are not to be disregarded, except upon practical grounds wider or better than our own. The main difficulty here concerns the likelihood, according to some local authorities, of confounding 'remittent' fever with other diverse types of pyrexia; yet I claim that we had acquired knowledge enough not to do this, and for my own part, hesitation would depend rather upon a possible confusion between the specific pyrexia and fever due to pure or consecutive pneumonias, especially, or sometimes to cerebral implication, or hepatitis and occasionally dysentery. Not seldom did it also appear that a secondary 'typhus'-like fever, was apt to be intermingled towards the later stages of the epidemic.

Finally, if it be said that my enquiries were few as well as imperfect, I would answer that they were carefully made and not prejudiced by selection; and, therefore, may stand as samples of a larger number. We gain a glimpse from them of the state of this town in these famine years; for the Mussulman localities which, as it happens, were mostly visited were certainly not worse off than the quarters inhabited by the poverty-stricken Hindoos.

Subjoined is the list of queries drawn up for preliminary use, the replies in writing are still preserved.

Question 1.—On what date was the patient attacked with fever?

Q. 2.—How many persons living in the same room with him have had fever and on what dates before or after he was attacked?

Q. 3.—Have any persons living in the adjoining rooms on the same floor ever had fever, and if so was it before or after the patients' illness?

Q. 4.—Have people living in other parts of the same house ever had fever, and if so when did this occur?

Q. 5.—Had the people in adjoining houses, or other parts of the street, been attacked with fever?

Q. 6.—Did the fever attack those of the same or different castes, and have the people mixed together or not?

Q. 7.—When and where did the first case of fever occur, and where did the sick person come from?

Q. 8.—How many came to hospital and have any died?

Q. 9.—Occupation of the sick?

Q. 10.—If strangers, their native place and period of residence in Bombay?

Q. 11.—General condition: cause of migration to Bombay: any fever at their native place; fever on the road: fever how long after arriving here?

1. Famine-fever in a small community.—In May 1878 a number of sick were brought to hospital, respecting whose origin I made particular enquiry and here copy my notes:—Omerkari. Visited the house of these Hallalkaris (public scavengers) who are Gujerathis from Kattiawar and Ahmedabad, living in two short rows of 'chawls' (low buildings), the doors of which are less than 5 or 6 feet apart, leaving a narrow passage between, always crowded, and by sleepers at night particularly. One room entered measured about 15 × 9 × 6 ft. and lodged five adults with children: others are larger, but equally crowded. There are many daily visitors, who are mostly new-comers looking for accommodation: the men sleep outside in the fair season: privies are in close proximity, some being open. Their food is dried fish, rice, onions and chillies; it is said to be of poor quality, and the people are emphatically unwashed: their dirty rags of bedding line the walls of their rooms.

Wages good, but habits very penurious : prejudices so strong that they will not go to hospital or stay there : upwards of 20 were sent by the police in a body yesterday morning, but only 7 could be induced to remain : fever and dysentery are their complaints : most were adults.

These people arrived here about a year ago and had been well until the last month or two, when fever broke out, which is gradually increasing : 4 died of fever out of 24 in the first week, in one place ; 2 men died out of 5 living in another room ; 1 out of 4 in the adjoining room ; 1 out of 3 elsewhere a few days ago, and some half-dozen others are now lying prostrate from the same ailment. One half of those taken to hospital at present show the blood-spirillum ; others are in the depressed condition : in all 14 cases of spirillum fever were admitted.

The fever is said to have appeared first amongst the residents who are now affected, but enquiry elicits the fact that individuals of the community have lately come in and are now tramping hitherwards from their native country, where much public distress prevails : until accommodation is found, these sleep beneath the jail walls close by, and some have fever on them now (verified on inspection) : they visit freely with the older residents and it seems probable have communicated the fever to them. All these people will go to work in the town even when sick, so great is their need and greed for money. From this statement it appears possible that famine-fever is being constantly imported into Bombay, and then spreads by contagion.

No other castes, Mussulmans or Hindoos, nor even the bhangies (local sweepers) who live alongside these Gujerathis, ever mix with them, and none are at present affected. Evidence against local malaria complete ; that in favour of bad hygiene not supported by the history of the outbreak, and the natural conclusion is that just mentioned.

The further history of this colony is as follows : they were bodily transported to another locality by order of the municipality, and on the approach of the rains, they took flight and returned to their native country to cultivate their fields.

2. House of Hussein Ali, whose case is fully detailed above (*vide* No. X.).—He was attacked with fever on May 28, 1878 : home in the Teli Mahulla, No. 21. Eight persons were living in the same room, out of whom 4 are known to have suffered : these were attacked 15 to 20 days before, and in order were Kurban, Kurmali, Junood and Hussein : all have been admitted into hospital on successive occasions, with spirillum fever. The house being small and narrow, there are no other rooms on the same floor : the people living above are Jews, with whom these Mussulmans have no intercourse and not even the community of a staircase ; the Jews are unaffected. All eight are of the same caste, mixed freely and live together on the lower floor (basement). The first case occurred in their room in a man who had been in Bombay 3 months.

The people were hawkers of tea, lemonade and soda-water : not strangers in Bombay ; in fair health and never had fever before.

NOTE.—An illustration of frequent occurrence, several persons in one room successively attacked ; no evidence of spreading, the reason being obviously construction of house and caste prejudices. The value of the illustration therefore depends upon the likelihood or possibility of there being in operation a strictly endemic cause capable of giving rise to relapsing fever in these men : of any such agency I am ignorant.

3. House of Karim Fakir, fever patient admitted July 1, 1878 (spirillum present). He was attacked one month after arrival from Azimgurh (Hindustan) at his first residence, No. 17 Ghass Mahulla. By personal enquiry I (through my native assistant) learn that two or three months ago fever was very prevalent in this house, and so much so that no single inmate escaped : ever since the fever has prevailed more or less. The following are the names in order of recent attacks—(1) Bhorun, (2) Bhiccan (died in hospital), (3) Kutwaroo, (4) Amir, (5) Bunnoo, (6) Bechoo, died after 10 days' fever, and 8 days afterwards the patient Karim contracted the same : 2 days after his illness began Bechoo's sister-in-law Voolphut became ill, then her brother Ismail, after that his nephew Kumjau, his mother Mariam : then a brother-in-law Karimbux, and on the 3rd day of his illness his wife Fatma and daughter Jainab. In the next room but one (No. 3 on the map appended to this Report, but not reproduced here) an old man named Mudari was attacked with fever 10 days ago : in a room behind two young lads also, who are now free. On the upper floor, second back-room,

Chitroon was attacked with fever 4 days ago. In room No. 1 on the plan, at present there are 2 women and 2 men with fever, and 3 others convalescent : in room No. 2 same upper floor is a case of fever.

The adjoining and opposite houses are free from sickness : patient had no fever on the road hither : occupation weaver : cause of emigration from his home want of means. All the above persons are of the same caste and all are weavers by trade : most come from the same place, and all freely communicated. Date July 1, 1878. Reporter an intelligent Marathi vernacular student.

NOTE.—There is no other memorandum regarding this particular instance, which was regarded by us as a fair example of fever spreading in a house.

4. House of Khudabux Sabrati and Lalmahomed Mugoora.—Admitted August 28 and September 1, 1878. Residence, No. 135 Camatipura 1st street, near the old Jews' graveyard : an upper-roomed house, 30 individuals mostly in families live above and nearly as many, mostly single, below. Fever began below in Kumroodan and 10 days after in Abdul Rahman living above, who was admitted into hospital, remained there one day and then took his discharge to die outside. In his room Fatma, woman, contracted the fever and died ; 5 days after her death her two sons became ill and both died : then Salim and his family living below were attacked, namely the wife Mariam, daughter Jumna and son Baboo, who all have left the place in alarm and gone into the country, place unknown. Immediately after them the first patient named above was seized with fever (bilious typhoid type), he resided below, came to hospital and died there. After him Ghulam Mahomed living on the upper story was attacked ; and then the second of the patients named above, who died of spirillum fever in hospital this morning (September 3) : after him Jumayee, living below, had fever which left him with profuse perspiration yesterday morning on the 7th day ; this man had an eruption of purple spots resembling those of typhus, as reported on a subsequent visit. On the 4th day of Jumayee's fever Baichun, woman, living in the above-named Mariam's room, was seized with fever ; and shortly after Kairoo, living below ; he left the house with Jumayee and went to live in Lower Duncan Road in order to get rid of the fever. Kairoo had fever on him at the time and was followed by my assistant, who persuaded him to let a drop of blood be taken from his finger ; this I examined and found to contain the spirillum. The poor man lay helpless in his lodging till the crisis of the fever, was then brought to hospital and died on the same day : autopsy not allowed : the symptoms were those of spirillum fever and not typhus.

My informant remarks with regard to the probable origin of the fever in this house, that he was told a man named Dilgan from Benares arrived here about six weeks ago with his family ; the son and daughter were attacked with fever on the road, one after the other, and the son died then : soon after their arrival in Bombay the daughter also died in this house. It was about this time the fever broke out in it, whose ravages are above described, but at this interval of time the people in charge could not say for certain whether Dilgan's family were the first attacked.

All people of the same race : occupation that of weavers : all residents, except the man Dilgan, who came to Bombay to seek work and food.

On visiting the spot I found the 'chawl' one storied ; ground floor raised a few feet, made of beaten earth and excavated with pits in which the weavers sit to work at the loom ; closely packed, dark and low-roofed : the upper space is divided by light framework into small compartments, which lodge married people, the others sleeping below where are also one or two partitioned rooms. Accommodation very limited, but have seen worse amongst the Marathas (Hindus) at Dongri. Many facilities for the spread of fever, and sanitary arrangements bad. Up to September 2nd, seventeen cases of fever had happened in this house, with six deaths.

I have alluded to another case, and find a note that on September 8 a man named Karim was admitted, delirious and collapsed, from the same house : my impression is there were even others known, but a frequent result of such wide infection is the scattering of the lodgers, who, as we have seen above, may carry the fever with them into other houses, just as patients have brought it into a general hospital, with the result of extension of the disease. Regarding the nature of the 'fever'

I have only to say that no doubt remains in my mind that it was of the relapsing type, and besides we have the hospital records which show that this spirillar form was co-existent with a typhus-variety, a coincidence elsewhere alluded to. A similar conjunction was observed in two cases admitted in the month of June, also of Mussulman weavers ; but I do not consider it necessary to pursue this subject here, nor need I add at present further illustration of the spread of fever in a house. The rest of my notes closely resemble the above, and I am not unaware that objections may be taken to all such instances, however closely pursued and long watched ; for it is simply impossible to eliminate every source of error in complex events of this kind. Yet if I could describe in a moderate space the numerous minutiae having but one common tendency in favour of contagion, which during my enquiries were seen and heard, I should not hesitate to insist wholly upon the examples above briefly summed up.

The above data are from the earlier J. J. Hospital series ; the following like memoranda belong to the G. T. Hospital collection, and they refer to the close of the epidemic in 1879.

1. From a house in Koliwada (fever-haunt) in March 1, August 1, November 1, and in December 6 cases of demonstrated specific fever were admitted : in all 9, of whom 5 females ; 2 residents, 6 immigrants from Kurmala, a Deccan village almost depopulated by famine-fever ; subsequently there were two other specific admissions, and at various intermediate periods entries of persons affected with fever of undefined but suspicious type. All persons intimate and commonly related : the details of this house epidemic are before me, and I note that 7 typical cases had a reputed origin between November 23 and December 26 : usually contagion seems the inevitable inference.

Earlier in the year 5 other Hindoos were admitted from a street or lane inhabited solely by the low-caste sweepers who suffered early, long and severely : these included a mother and children with fever not always demonstrable by the microscope, and contemporaneously with a child's relapse, a neighbouring patient became affected.

2. From a house (or two adjoining houses) in Surati mula (fever-quarter) 9 Mussulman weavers, connected by birth or work, were admitted in January and February 1880 : they included 2 fathers and sons, attacked on 12 and 15, and on January 21 and 22 respectively ; besides, were several admissions with fever at stages not determinable.

From a house not far off 6 other weavers were admitted in February ; they included a father and 2 sons (wife and daughter also coming with pyrexia of undetermined character) whose illnesses began as follows : January 22 and 29 and little son February 6, that is to say, at intervals of about a week, which is the ordinary incubation-period of spirillum fever, and I will add about that of the earlier apyretic intervals ; now, since infection may proceed any time during specific pyrexia and probably a day or two before (see the account of my Comparative Experiments), I conclude that an induced attack may appear either immediately after, or not till 7 days after, that in another person giving rise to it, and hence the possibility (at least) of this man and his young sons being infected successively from each other, beginning with the father, who necessarily had charge of the lads because of the mother's preceding illness : if in strictness more cannot be said, from want of experimental knowledge regarding the human species, I shall still regard these instances as in the highest degree suggestive.

The case of the 5 negro relatives living together and successively attacked has been already alluded to, and I repeat that of 53 proved examples of spirillum fever which I became well acquainted with in 1879-80, the 44 which came from outside contained only 4 seemingly

isolated instances : the 9 arising within hospital precincts offering peculiar evidence of contagion, considered under the next heading.

Note on the possible Development of Fever outside a recognised focus.—Judging from hearsay evidence, sickness was not very seldom conveyed from house to house by affected individuals, and such occurrence is comprehensible enough ; an illustration is the following :—

August 1878, J. F. lived in a house reported on above, and left because of much fever prevailing in it for a new residence fever-free ; here his daughter soon showed symptoms and immediately afterwards his wife and a few days after he was attacked himself : meanwhile the child had died, when he and his wife came to hospital, she having then a relapse and he a primary attack which is severe. When this family left their new home none of the 6 other residents had become affected, but J. F. lived somewhat apart from the rest.

Cases of this kind have been already alluded to, and upon enquiry I became assured that here was one method in which the epidemic was maintained.

That a hospital might become a source of disease outside is obvious upon the least reflection, yet it would be difficult in so large a town as Bombay to prove this. I have a note as follows :—October 1877. R. B., admitted for secondary syphilis, was afterwards transferred to the Ophthalmic Hospital, and there showed the symptoms of specific fever : he then returned for treatment, but supposing he had gone to his home instead, he would have carried infection thither. Several of the patients seized with fever in hospital insisted upon leaving before the possible or probable termination of their acquired attack, and some doubtless became ill at their homes : such *contretemps* was practically unavoidable.

C. HOSPITAL EXPERIENCE.

Whatever be the evils, surgical or medical, which arise from 'hospitalism,' it is not customary (even in India) to include malarious fever amongst the contingencies of contact with the sick ; and if, as a fact, it should be found that officers, ward-attendants and patients in succession have distinctly acquired in hospital a serious febrile ailment, the presumption would be that such acquisition is not a malarious remittent. Add that the occurrence is a novelty, happens at all seasons of the year, is limited to certain medical wards in the building and is strictly contemporaneous with the influx into these wards only, of poverty and famine-stricken subjects themselves presenting identically the same febrile disease ; say, further, that the fever is not regarded as a 'remittent' (but quite the contrary) by those medical men who have had most to deal with it, and the presumption of a new, contagious disease will become greatly strengthened. Yet such is the essence of hospital experience at Bombay, during the years 1877, 1878, and 1879. Certainly, the origin and spread of pronounced and highly characteristic 'fever' within the walls of the J. J. Hospital, was an event unknown during the previous 12 or 13 years. In 1864-5 an occurrence of a similar kind took place, which I have referred to elsewhere (*vide* page 26) ; but at no subsequent date until about the commencement of 1877, could I find in official records, or tradition, any notice of the like.

I have now to narrate a series of incidents, which nearest of all my

data approach to conclusive evidence of contagion. They have been seen always when a number of fever-sick was collected, as in the Famine-camp, the J. J. Hospital and the G. T. Hospital, which were the only known public resorts of the ailing.

Camp Experience (*see page 21*).—As regards this series, it will be enough to state that amongst the healthy attendants at the camp of refuge erected on the outskirts of Bombay, 3, if not 4, instances are known of individuals becoming affected with the same fever as that prevailing amongst the immigrants themselves; what proportion these three bore to the other supervisors and servants I am unable to say, but the latter were not numerous, and it is quite possible some of them were affected unknown to me.

Regarding patients, I find that of 47 cases under treatment for 'fever,' no fewer than 16, or quite one-third, were attacked in the camp at an interval of 6 to 11 days after their entering it; and in every instance, the statement is expressly made that they were previously well. It is true that in about one-half these 16 there is an account of some other member of the family being affected, but in others it is not so; and as the time at which the fever became developed is similar in all, I cannot avoid the conclusion that infection took place at the time when the healthy joined the closely-packed numbers occupying the huts of matting provided for their accommodation. It was well known that those sick with fever could not be at once isolated, for they had often to be sought out, being unwilling to leave their families and resort to the contiguous huts which served as hospital fever-wards. The temperature-charts and recorded symptoms of all these cases are either decisive or strongly presumptive of the new disease being famine-fever; and the blood-spirillum was detected in some of them, as well as in their neighbours suffering in the same way and coming direct from dwellings in the town: further, soon after the camp was broken up, I was twice enabled to trace specific fever in its late inmates.

Illustration of the conjoined camp experience is as follows:—

1. J. A., æt. 20, hospital apprentice, was one of two apprentices detached for duty at the camp and joined on April 9; ten days afterwards he was attacked with high fever, and taken to hospital. The pyrexia was irregularly remittent, max. t. $105^{\circ}4$; typhus symptoms early supervened, and he died on the 8th morning of invasion. I did not see this patient, but an experienced medical officer who visited him, informed me that the case resembled one of true typhus.

2. V. L., æt. 22, a Vernacular student (one of two on the same duty), joined the fever-camp on April 10, and was himself attacked on the 23rd. The invasion was sudden, max. t. $105^{\circ}2$, form ascending, remittent and continued, duration 7 days, fall moderate but prompt; a sharp rebound followed, and at its decline four days afterwards, he was exhausted and still delirious, the attack having been a severe one: convalescence, I heard, was very slow.

Supposing infection to have taken place at the camp, the longest periods of incubation would be 10 and 13 days respectively; probably it was shorter.

3. D. L., æt. 30, was Brahman cook at the camp which he joined March 30, and he remained well till April 11, when he was seized with fever: max. t. $105^{\circ}4$, form of pyrexia continuous descending, duration 6 days, fall rather gradual but considerable (t. $96^{\circ}2$), reaction gradual and when last seen (April 24) there had not been a relapse. This man I saw, and believed to have 'camp' (here 'famine') fever.

4. V. B., æt. 7, previously unaffected, had been in the camp 7 days before he was attacked: brought to hospital-shed two days after the fever came on. Max. t. seen

105°·6, form remittent; fall prompt and considerable (t. 96°): a relapse took place 7 days afterwards, lasting 4 days (max. t. 103°·8), and the last entry is that of a sudden fall to 95°·8.

5, 6, 7. The Tookia family. B. T., a girl of 8, came to the camp March 30, being quite well, and she remained so till the morning of April 9. The same evening the t. rose to 104°·9: on the 5th day the fever suddenly left, and the t. then became normal. The blood-spirillum was seen during the pyrexia.

Y. T., her brother of 10, arrived here on the same day, being also well and remaining so till the forenoon of April 8, when strong fever set in which lasted till the evening of the 13th: no relapse seen.

M. T., a younger brother of 6, arriving the same day, remained well until April 7, when fever came on which lasted 6 days, suddenly left and was not seen to return.

These 3 sick children of one family lying side by side in the rough hospital they were brought to from their adjoining dwelling, conveyed to me a vivid impression of the reality of contagion in camp, and I thought how desirable prompt isolation was in this heterogeneous assemblage.

Hospital Data.—In both Institutions there occurred examples of contagion amongst patients (1), amongst servants (2) and the medical staff (3), which in this order will be alluded to below.

1. J. J. HOSPITAL.

Here there are reckoned to be 24 wards, containing 436 beds; but, omitting the mixed Parsee ward, only 10 of these with 184 beds are general medical wards: I may exclude also the 2 clinical wards (32 beds) set aside for selected cases, so that in only 8 wards with 152 beds could it be said that contagion was most likely to occur from the admission into them of the poorest and worst cases of fever, and from their often overcrowded state, which could not be avoided. Two, with 38 beds, were set apart for female patients.

Each ward is provided with an attendant (ward-boy or nurse) and a helper (hamall): two had three attendants, making in all 18 servants, or with the 5 sweepers (latrine-cleaners) on part duty here, 23. I should mention that half an additional ward was latterly appropriated to fever cases, and if the servants there be included the total rises to 26.

The clerical work was done almost wholly by young men belonging to the Vernacular classes, who took regular turns of duty for two or three months at a time; it is difficult to say how many were thus actually employed, but a written statement has been made for me mentioning 47 in the abstract, though as some were engaged more than once it seems likely that hardly more than two-thirds of this number came into close contact with the sick, during the year and a half (viz. from May 1877 to October 1878) prior to the breaking up of the Vernacular classes.

The eight wards in question were visited by 4 Native assistant-surgeons. The hospital establishment includes a large complement of cooks, washermen, barbers, tailors, foremen, &c., besides the stewards' department, office and messengers; and though none of these individuals have a particular connection with the medical wards, yet it happens that 2 amongst them were attacked with the specific fever.

During the period under review, it may be estimated that the total number of patients passing through these 8 (afterwards 9) wards, was about 3,000: the servants 26, the writers and medical officers 50. I may here add that of the sick about 1,400 admissions were cases of 'remittent' fever (so-called), out of which nearly 500 were proved to be

spirillum fever ; on deducting this last number, the total of patients exposed to infection becomes 2500.

a. Contagion amongst Patients in Hospital.

Subjects of fever brought in were placed amongst other medical cases, and only latterly, *i.e.* after April 1878, were those cases ascertained to be of specific character, removed to a separate ward : indeed, at no time could it be said that strict segregation was in force, and consequently the opportunities of contagion abounded. On the other hand, nearly all the medical wards are open on two sides to the prevailing winds, and though some are not well designed, there is good ventilation in others ; and generally there is a degree of aërial perfusion, which probably equals that of the average European hospital. Owing to pressure of applicants, however, the open verandahs had often to be occupied ; and as the cots were always placed at no greater distance than 4 feet apart, no surprise need be felt at the occasional occurrence of contagion, but rather that it was not more frequent. I have sometimes had good reason to believe that instances of infection were overlooked ; and considering the early date at which many patients were discharged, it must be allowed that sometimes fever acquired in hospital had not time to become developed there. But the circumstance which most of all has tended to shorten my list, is the difficulty experienced in determining the shortest period of time after admission which is compatible with the occurrence of contagion.

For example, a patient free from fever on admission shows its signs 7, 10 or 12 days subsequently ; and since the longer of these periods is known occasionally to intervene between invasion and relapse, it becomes a question if the attack seen in hospital be the first or second, or in other words, if contagion have or have not taken place immediately upon his entering the wards, as obviously might be the case. Having very early been led to infer that the incubation-period of spirillum fever essentially corresponds to the apyretic intervals which separate its periodic developments and are, in fact, their respective incubative stages, I consider that an instance like that now supposed may be differently interpreted. The following circumstances would, however, determine the diagnosis : when the patient is admitted just after the close of the invasion, there will be a low temperature and depression of the system, to be promptly followed by some mild febrile reaction, or with sweats, aching pains, diarrhœa, cough, swelling of the parotids, the spleen or liver still enlarged or tender, jaundice or night-delirium and, briefly, any of those symptoms belonging to the first interval which have been already described. There will be a history of smart fever just prior to admission, and probably of contact with fever cases outside, or of relatives likewise affected. Could, indeed, a trustworthy history be had, no reasonable grounds of doubt should exist in the majority of cases ; and similar remarks apply to admissions after the first relapse. But I know, from observation, that every one of the signs just mentioned may be absent after an attack of veritable spirillum fever ; also that paroxysms of intermittent fever at any interval may precede, and that the specific fever itself may assume such form that a dull or depressed patient becomes quite unable to furnish definite information. Some moderate pyrexia may be present on admission, which subsides in 3 or 4 days, leaving the patient tolerably well, and a week

after he may be suddenly seized with high fever attended with the spirillar infection ; and, in short, it is sometimes very difficult to be sure that there has not been an attack of fever prior to admission, which of course would negative the idea of contagion in hospital.

A brief and irregular, or intermitting attack, with high fever and considerable fall, would almost certainly indicate a relapse and not a primary attack. But when there is no history of possible infection or of a previous seizure, and no marked perturbation of the system on and after admission for 8, 10, or 12 days ; then I think we should be justified in regarding the new attack as due to contagion in hospital. The longer period is that fixed on in my own determinations of cases seemingly doubtful ; and as immediate infection was always possible, and prompt investigation sometimes impracticable, it is by no means unlikely that I have overlooked some examples of fever acquired amongst the sick.

The records of 30 cases of demonstrated spirillar contagion are before me, dating from all periods of the epidemic ; but rather most numerous towards its close.

In general, it may be stated that the acquired disease was identical with the introduced, as to form, duration and severity ; sex, age and mortality were also the same. Thus, as regards the subject ;—males 22, females 8 ; ages ranged from infancy to 60 years, mean age of males 33, of females 31 years ; general condition prior to infection bad in 5, indifferent 12 times and fair in 13 : mortality 10 or 33 per cent., which is very high ; death were males 7, females 3 ; the very young and the aged succumbed, but intermediate ages were not spared. The stay in hospital before attack varied from 12 to 77 days, the mean period being 27 days : the greatest average number being attacked between 2 and 3 weeks after admission, namely, 9 in 25 (infants at breast being here excluded), or upwards of one-third ; and 13 or one-half, at least, showed the disease during the first 3 weeks after admission. This fact is interesting, but whilst it points to more or less prompt infection, it cannot be made use of in estimating the precise incubation-period of spirillum fever ; for the conditions of exposure to specific influence were not always the same, and neither date nor source of infection could ever be exactly determined.

There is no fixed ratio between the ages of patients and this intervening time, as is shown by the adults (30-40 years of age) being attacked both earliest and latest : individuals between 20 and 30 were, however, mostly affected (4 in 6) during the third week. A definite connection between previous disease and length of pre-febrile period is not manifest ; subjects with ague were attacked both early and late, and so some of those affected with organic disease, and in either fair or reduced general health.

A definite relationship of previous disease to occurrence of infection does not appear, and even that of previous disease to mortality is not apparent, as the following enumeration will show—the stars indicate deaths :—Anæmia 1, malarious cachexia 1, debility after remittent fever 1, agues 8*, scrofula 1, secondary syphilis 1, lumbago 1*, mania 1*, hemiplegia 2*, bronchitis 2*, cardiac dropsy 1*, hepatitis 1*, diarrhœa and dysentery 5**, no disease (infants) 4*. Here, agues not un-

frequently preceded contagion, still they and remittents are the commonest forms of all disease seen in hospital.

Regarding the form of disease acquired in hospital, I find it even in this small series to be the same as usual—thus, half the cases had a relapse, the remainder being equally divided between abortives and undetermined : the time and mode of death were the same, and so also the more peculiar *post-mortem* appearances.

As to the probable mode of infection, I cannot specify any one in particular : contact was always possible, whilst mere contiguity of beds seemed to have no essential influence, for it is comparatively seldom noted that the attacked in hospital lay alongside the affected new-comers, though it was distinctly so in the latest instance known. Nor did it appear that caste (which here mostly determines the degree of contact) played any special part ; and as to contagion through the food, water, clothing, or excreta, no positive data were elicited. Viewing this complexity of conditions and the incidental occurrence only of contagion in hospital, I was led to suppose that either a personal predisposition must be present in some individuals, or (what is more likely) some intervening facts had been overlooked.

Illustrative Cases.—Infants. It seems likely that sick mothers commonly infect their sucklings, which they pertinaciously keep by them : there are three instances of it in the small collection here, and others are alluded to in the family series ; the event may lead to death of the child. Children may catch the fever from other children, as in the instance of an orphaned girl belonging to the list under notice ; but some children seemed to escape, at least during their stay in hospital.

Adults. My list is almost representative of both ordinary and complicated forms, and very mild as well as severe disease finds place in it. This feature serves to illustrate the close similarity of the spirillum fever under various conditions of origin. Indeed, a tolerably complete account of the affection might be compiled from the contagion-series alone ; and I regard this circumstance as a highly suggestive one.

1. D. B., æt. 40, had splenic enlargement and some slight febrile paroxysms at night when admitted on April 14, 1877, there was also occasional diarrhoea ; though soon reported as convalescent he remained in hospital until July 2, when he underwent a mild attack of fever ; max. temp. $104^{\circ}\cdot6$, form remittent descending, duration the greater part of 5 days, fall very moderate (98°), and the body-heat was normal for the remaining 25 days of his stay in hospital. It happens that the notes of the case were taken in the ordinary way, and beyond the statement of some tenderness over the spleen and a brownish tongue towards the close of the attack, there is nothing in the record to indicate its nature, nor is the t. chart quite characteristic ; yet the instance was none the less undoubted. Mild abortive attack.

2. P. G., æt. 50, admitted April 5, 1878, with valvular disease, had by the 4th May so far recovered that the morning entry runs 'feels well ;' the same evening it was 'feels very ill,' t. $103^{\circ}\cdot4$ with chills ; this was the initiation of a febrile attack, max. temp. only $103^{\circ}\cdot6$, form of pyrexia remittent, concave, duration 7 days, and termination death in the typhoid state. The fever was unusually low and the depression early and extreme. Spirillum fever in a bad subject : fatal.

3. V. A., æt. 35, admitted March 14, 1878, with scrofulous enlargement of the cervical glands and occasional feverishness at night, which soon subsided, was on April 13 suddenly seized with fever which lasted 5 days, attained a maximum of $105^{\circ}\cdot2$, assuming the form of convex remittent and ending with a prompt fall to 97° , with sweats and delirium : after a slight rebound the temperature remained normal till

April 27, when pyrexia returned, again lasted 5 days, reaching 105° and then falling abruptly to $94^{\circ}\cdot6$; delirium reappeared, but rallying was quick though rather unsteady during the 13 days longer he was seen. Relapsing form.

4. B. D., æt. 30, admitted August 8, 1877, with chronic dysentery which had much reduced his strength, was on August 24 attacked with fever of irregular intermittent form and undefined duration (7 or 12 (?) days), max. temp., in first paroxysm (105°) finally only 100° , but the general symptoms were out of all proportion more serious than indicated by the temperature, the man speedily falling into the typhoid state, and in any other fever known to me his case would have seemed hopeless; the spirillar development, however, has only a limited duration, and along with it the attendant pyrexia: this man slowly recovered and his Chart No. 5, PLATE IV. remains as a striking example of anomalous spirillar pyrexia; there was no relapse before his leaving convalescent on September 21.

5. A. B., æt. 35, admitted November 15, 1878, had only slight fever (called intermittent), some diarrhoea and cough until December 15, when pronounced tertian fever set in, which after 4 paroxysms ended in a quasi-continuous type, max. temp. 106° , lasting 9 days and terminating December 30 with a tendency to crisis; no complication detected, and no spirillum: a rebound followed, but the man soon became free from fever and remained so for 9 days, when there appeared (January 8) high, level, remitting pyrexia (t. $105^{\circ}\cdot4$), lasting 6 days and ending with a characteristic morning crisis: the same evening the body-heat had again risen to 105° and he died. The blood-spirillum was found only during the last attack, which on the chart is distinct enough from the previous one by its compact outline; and Mr. L. Arjoon and myself were of opinion that this second attack was due to infection from a patient ill in the next bed with spirillum fever, whose daughter soon after was affected and from whom also a second patient, lying in an opposite bed, doubtless acquired the typical relapsing attack he too shortly displayed in hospital. A. B.'s case is a remarkable one of successive intermittent, remittent and spirillar fever. See Chart 19, PLATE V.

6. A. R., æt. 40, admitted September 12, 1877, with intermittent fever and chronic bronchitis, had an aguish paroxysm the same night, but for the next 10 days he was quite free from febrile ailment: on the evening of the 23rd smart pyrexia (spirillar) set in, t. 104 , form remittent, level; duration 6 days; after a partial fall irregular and milder paroxysms commenced (non-spirillar) which lasted 10 days and accompanied the development of severe asthma. Then a fresh attack began of high, regular, persistent pyrexia ($105^{\circ}\cdot6$), from remittent ascending, duration 6 days (spirillar relapse); fall sudden and considerable ($96^{\circ}\cdot2$): a sharp rebound occurred, and thence normal t. for the 17 days longer he remained in hospital. Although the specific attack began so early as 12 days after his entrance, I concluded the case was one of prompt local infection upon the grounds before mentioned (ague preceding 10 or 14 days has been seen in other instances), and it is a good example of a co-existent complication which may well obscure the nature of the illness, until periodic exacerbation or sudden amelioration afford a clue: here the microscope at once revealed the character of the fever, and very ill as the patient was, he at once began to recover at the close of the relapse. Chart No. 12, PLATE V.

b. Infection of Hospital Servants.

Amongst the inmates of a ward there must happen between patient and attendant, a closer and more frequent contact than amongst the sick patients themselves; and this is doubtless one of the reasons, if not the chief, why a larger proportion of hospital servants come to exhibit the signs of febrile infection. There may have been other circumstances concurring to explain the great difference between the ratios of 1: 5 (attendants) and 1: 80 (patients), which I find to represent the comparative liability to infection of the two classes named; but what those conditions were, I did not perceive. All the hospital attendants who became secondarily affected being on duty, were in fair health; and as a large proportion of the patients attacked after admission

were not in a seriously impaired state, I infer the normal or quasi-normal condition is favourable rather than otherwise to the reception of the spirillar contagion. Apart from this consideration (which at first sight seems an improbable one), I am not aware of any prominent circumstance so valid as that of direct contact in determining infection.

The list before me of ascertained spirillum fever amongst hospital attendants contains the following instances:—the ward-boys* of 4 wards out of 6, both matrons** of the two female wards, and 2 helpers* of the most crowded wards for both sexes; 3* (or 4) out of 5 'sweepers' and some members of their families frequenting the wards; 1 'office hamal'* out of 4 who assist in attending to incoming sick: 1 stewards' servant out of 2 whose duties necessitate their often entering the wards; 1 cook out of 12, some of whom serve out the food directly to the patients; 1 gardener resident very near to the male dispensary: total 15, with 6 deaths (marked *). A second list I have shows every case of so-called 'remittent' fever amongst the hospital attendants during the period under notice, and it contains only eight other instances besides the above with one death: supposing, therefore, that these last were all non-spirillar and may represent the ordinary amount of incidental illness amongst servants (they rather exceed this), we perceive that marked febrile attacks were nearly trebled in 1877-78, and the mortality much more increased. The significance of this datum cannot be overlooked, and it receives confirmation from the other contagion-series. In the first enumeration I have omitted one suspicious case of a 'sweeper,' because the blood was not examined, and there are some others of the kind in which this special test was not applied; such instances were, however, of hardly doubtful character. All the known specific cases were treated in the medical wards; their families who lived in the hospital enclosure had free access to them, and in one instance a young lad became infected: that the fever did not further spread in this direction, is a fact illustrating the prophylactic effect of separate residence and only occasional contact.

Details.—Males 13 with 4 deaths, females 2, both dying: ages from 10 to 60 years, the mean about 30, and one-third at 21-25 years; 5 of the deaths were under 35 years. Mortality 40 per cent., or at a high ratio difficult to explain, seeing that it rather exceeds the rate amongst infected patients and very greatly that amongst the medical pupils also contemporarily attacked. The houses of the resident attendants are, however, remarkable for their ill-construction and insanitary condition.

The form of pyrexia acquired, and its general course and symptoms, entirely correspond with those of spirillar infection; so that absolutely no doubt is possible, as to the identity of the several illnesses. The nine surviving in type were 5 relapsing (one with two recurrences), and 4 abortive; the 6 deaths happened, as is the rule, in either first invasion (4) or in first interval (2): in general the attacks were severe, and there was one characteristic autopsy.

Illustrative Cases.

1. V. S., æt. 55, attendant in No. 11 ward, resident at the hospital for 7 years, was admitted October 6, 1877, with high pyrexia ($104^{\circ}\cdot2$) dating from the previous morning; form of subsequent fever remittent descending (the temperature being lower

than usual, but the symptoms soon assuming a typhoid character), duration of attack 9 days, fall considerable ($95^{\circ} \cdot 2$): prompt rebound and no recurrence for 16 days after. In his ward were several cases of spirillum fever, and, like his predecessor (who was also attacked and who died), he was in the habit of taking a daily siesta on any cot in the ward that might be empty for the time. Previous health and family history good. See CASE LVII., Chap. V.

2. L. R., æt. 17, hospital sweeper and living on the premises for the last 13 years, admitted February 2, 1878, near the close of the invasion-attack, had afterwards a regular first relapse and a prolonged (? duplicated) second recurrence: he recovered completely. He was on duty in No. 9 ward (clinical) where not many specific fever cases were taken, and used to sleep in the hospital verandah: his symptoms were pronounced, and that he had acquired an infective kind of fever, is apparent from the fact that his younger brother who at first waited on him, was also attacked, manifesting the invasion just after the close of his senior's first relapse. See CASE XXXI., Chap. IV.

3. J. P., æt. 30, matron in No. 10 ward, resident in the hospital for 4 months, was attacked October 13, 1878, with fever seemingly intermittent at first (t. $103^{\circ} \cdot 2$), then subsiding and reappearing in more prolonged character with still low t. ($101^{\circ} \cdot 2^{\circ}$), and finally on the 8th evening rising to 104° , when death took place. She had cerebral symptoms, a few petechial spots and a general typhus aspect. Almost simultaneously with her, a clerk engaged in the same ward became affected in a very similar way, and he, too, died: at this time there was a family (the Gopals above-mentioned) of children displaying the spirillum fever in No. 10, and in the bed next to one of theirs a young woman recovering from the same. In these last instances the attacks were of the usual severity, or even mild, though the father of the children (in another ward) suffered considerably: hence the opportunity of contagion was evident, and inferentially the proof of its actual occurrence. That an apparently mild form of infectious fever may communicate a severe form, is well known in the annals of every species of such fever. J. P. was in seemingly good health, and very active prior to her fatal attack: details of symptoms are furnished in CASE XIII., Chapter II.

c. Contagion—Medical Staff.

The clerical work in the wards is carried on by young men of the Subordinate Department who were either apprentices of pure or mixed European parentage, or Native students of the Vernacular classes. During the greater part of 1877 and 1878 they were distributed as follows:—the apprentices almost exclusively to wards 2, 5, 9; and the Vernaculars to wards 3, 4, 6, 8, 11 and 12 for males, and 10, 13 for females.

I have already stated that the clinical wards 2 and 9 were occupied by selected cases only, seldom including an example of the spirillum fever; whilst ward 5 (for Parsees) was not known to have a single case in it; it may be said, therefore, that in these three sections of the hospital the probability of contagion was less apparent than in the remaining medical quarters; and, as a matter of fact, during my tenure of office not one of the apprentices was ever attacked, nor, I should add, were any of the College students attending the clinical classes. This circumstance is a very significant one; and should it be said that the exemption in question arose from the clinical and Parsee wards being better ventilated than most of the others, or from the apprentices and English-speaking students being of superior physique and hence less liable to infection, I would remark that neither of these conditions was universal, for wards 6 and 8, which furnished several instances of contagion amongst the subordinate staff, are as open as Nos. 9 and 5; and as to the protective influence of good physique, that is a point quite open to debate. Further, during my brief supercession by the permanent first physician, I find that an exceptional case did occur amongst the apprentices, which,

as virtually proving the rule of close contact aiding contagion, is worthy of particular mention here :—

In wards 8, 11 and 12 one student apprentice is occasionally on duty, and A. P. was thus employed in No. 8 during November 1877, when it contained several cases of the spirillum fever : on the 22nd he was attacked with persistent pyrexia ($103^{\circ}\cdot4$) which on the 6th day promptly subsided, leaving him apyretic for 8 days, when a recurrence took place (max. t. 105°) lasting 5 days and ending with a sudden and low fall (t. 95°) : then a perineal abscess formed and after it was opened, smart but brief fever again set in (Dec. 17 and 18 or 7 days after the last critical fall), which would correspond to a second relapse : he was discharged convalescent seven days afterwards. The first of these relapses as displayed in the charts before me is quite characteristic of the spirillum fever (the invasion not being all seen) ; but the blood was examined only in the second relapse and with a negative result, as is sometimes the case in even typical instances under ordinary scrutiny. From the notes written on the case I infer it was regarded as being probably one of contagion, and such would be my own strong impression : admitting the accuracy of this view (and there is nothing against it) I see that here was really no exception to the common event of infection, when exposure has been adequate.

The case of apprentice J. A., one of two on detached duty in the famine-camp, who died from fever coming on 10 days after he joined, has been above alluded to ; and here, too, the risk of contagion was more considerable than usual.

In ordinary years it is very seldom that these lads become ill with serious fever, and not often with milder forms of irregular intermittents ; as the records of the hospital show.

I now pass to instances occurring in the Vernacular classes, the junior members of which attended for instruction in No. 12 ward (clinical), whilst the seniors as ward-clerks and diet-writers were in general employ, and came more habitually in contact with fever patients. The last-named alone manifested infection, and after excluding from my list the case of V. L. (*vide* Camp-series) I find that not fewer than 10 of them were attacked in hospital with the spirillum fever ; the symptoms in all being highly characteristic, and the diagnosis always being verified by the microscope.

In a prefatory remark the number of clerks on the roster for duty was stated at 47, but it is certain that the turn of many came more than once during the period under review ; how frequently this happened could not be precisely ascertained, and hence I am unable to furnish the correct proportion of attacked to non-attacked : 1 in 4 would be a rather low estimate, but if it be adopted, the evidence of contagion becomes considerable. Its occurrence indeed is so manifest in this series, that I might insist upon such testimony as in itself conclusive.

As to the comparative experience of previous years, I will show in the summary of this section that casual sickness from fever in the Vernacular students was infrequent and almost exclusively limited to aguish attacks ; such attacks amongst these lads happened occasionally during 1877 and 1878, and their clinical discrimination from the typical relapsing types, now first seen, soon became easy.

Some particulars of the above-named 10 cases are the following :—General condition good or fair, all the affected being on active daily duty in the wards : age 20–25, mean 22 years : a common residence outside hospital, considered healthy ; diet ordinary, and means sufficient : work not excessive, though some lads read hard : one had a repetition of the attack (showing the influence of idiosyncrasy?) : one died.

Disease :—5 cases from June to September 1877, 5 from March to October 1878 : most occurred in June 1877 (3) and April 1878 (3) : form of fever relapsing in all the first 9 cases and 1 had two relapses : once the attack was repeated : the symptoms (often very pronounced), duration of pyrexia and of apyretic intervals in close and invariable correspondence with experience amongst the pauper patients : even the last case which ended fatally during the invasion and had a typhus-aspect, was practically identical with some of the worst cases seen in patients also towards the end of the epidemic. True abortive spirillum fever did not occur in this series (which is noteworthy) ; but once the relapse was mild and obscurely indicated.

Illustrative Cases.

1. M. R., æt. 22, Vernacular student, was clinical clerk in No. 12 when he was attacked with fever on June 10, 1877 : max. t. $104^{\circ}6$ on the last morning (*perturbatio critica*) ; form level, remittent, duration 7 days ; fall upwards of 8° in 4 hours : a sharp rebound followed, and on 15-16 day a mild relapse ($100^{\circ}2$) : thenceforward convalescence. In September 1877 having returned to duty, he was again attacked with spirillum fever : max. t. seen at close $103^{\circ}6$, duration 7 days, fall pronounced, a delayed rebound on the 11-12 day (? relapse) after which a slight rise on 21 day and convalescence. Here although the infection was not the same, his symptoms were nearly alike, and having noted this event in other repeated attacks, I infer that the individual constitution probably determines 'varieties' in the fever. This young man's blood showed the spirillum at initiation of the second attack, when he was still going about and engaged in collecting specimens of blood for one of my regular afternoon inspections.

2. N. P., æt. 20, Vernacular student, was a writer (*i.e.* copied the prescriptions) in No. 3 ward (where fever prevailed), when on April 4, 1878, he was attacked himself : max. t. prior to crisis $105^{\circ}4$, duration of attack 8 days, fall moderate : he was apyretic for part of 6 days, when the first relapse set in ; it lasted 6 days (max. t. 105°), fall pronounced ; he again remained well for 8 days, when a second relapse came on, lasting part of 5 days (max. t. 104°) ; and on rallying, he left for change of air.

3. B. S., æt. 22, Native medical pupil, was a writer in Nos. 6 and 10 wards, when in October 1878 he became subject to mild evening pyrexias, which were regarded as aguish and did not increase ; there was also diarrhoea (chest sound). On October 13 severe fever suddenly set in, at first remittent then continuous (max. t. $104^{\circ}4$), and on the 7th day he died with typhus-like symptoms. The matron of No. 10 ward was attacked similarly and simultaneously, also dying of the fever : there were spirillar cases in this ward, and no other person was affected. *Post-mortem* appearances in both instances confirmative.

The record of contagion at the larger hospital may be concluded with a reference to the case of Dr. A. M. K., one of the assistant-surgeons in charge of fever wards and diligent in blood-examinations, who being seized with high fever in June 1877, suffered for 7 days, and again after 9 days apyrexia, for 4 days more (relapse). The blood was not scrutinised, but all the main symptoms corresponded with those of relapsing fever. I visited once Dr. A. M. K., and like his friend Mr. S. A. and himself, had little doubt that the fever was specific and had been acquired in hospital. None other of 6 medical officers on duty in the medical wards became infected ; nor, I may add, did any of the operators at the numerous autopsies made of famine fever cases. This last-named experience contrasts strongly with that of 1879 at the smaller hospital, where although the *post-mortem* examinations were very much fewer (*e.g.* as 10 to 100), yet several operators became infected.

GOCULDAS TEJPAL HOSPITAL.

1877. During my brief tenure of office in parts of April and May, and of November and December, contagion in the wards was distinctly manifested : as regards the remainder of this and the following year, I am unable to write from direct knowledge. The building is new and excellently constructed : in site and ventilation it offers a favourable contrast to the larger native hospital, the cubic space per bed amounting to about 1,800 c. f. or more than double the allowance elsewhere, and overcrowding was not so common. Hence, probably, the less frequent occurrence of intra-mural contagion which has been observed. The accommodation for medical cases amounted to 72 beds, and of these 40 were allotted to male fever-cases collected together : in attendance on the latter were 1 medical subordinate and 6 ward servants, besides 2 sweepers. With the exception of a very suspicious (though unverified) instance amongst the latter, all these individuals escaped infection ; but, as if to show there was no real immunity, three well-marked specific attacks appeared in other persons seemingly less exposed. The earliest instance arose thus :—Owing to increasing numbers a few fever-cases were transferred to a surgical ward on the first floor in charge of a subordinate named R. W., who thereon was promptly seized with fever : the second instance occurred in one of two ‘peons’ or messengers accustomed to assist in the out-patient department ; and the third happened to the hospital barber.

Amidst the 50 famine-fever cases detected in April and May, no instance of communication amongst patients was known ; nor in November amongst 15 other cases. The proportions of infected were 1 : 25 patients, and 1 : 15 of establishment. After escaping for eight months both here and at the larger hospital, I was myself attacked, in December, in consequence of a dissection-wound.

Illustrative Cases.

1. R. W., æt. 24, while on duty under the circumstances named above, was on April 2 seized with high fever (max. t. seen at close 105°) lasting seven days and ending very abruptly ; he then remained apyretic, and the nature of his illness not being recognised, he was discharged to duty on the 8th day after crisis. The same evening the fever returned (first relapse), lasting 5 days (max. t. $105^{\circ}4$) and quickly subsiding (min. t. $95^{\circ}2$) : after an interval of 10 days’ freedom, a second recurrence of smart though briefer pyrexia came on (t. 103°), and shortly afterwards he went on leave. The blood-spirillum was detected in both relapses.

2. T. K., æt. 20, hospital barber, previously well and on duty, was in October seized with high fever (max. t. seen $105^{\circ}2$) which lasted 6 or 7 days and suddenly subsided : he remained free for 6 days, when a relapse occurred lasting part of 5 days (max. t. $105^{\circ}6$) and terminating with a fall of 10° in the course of less than twenty-four-hours ; after slowly rallying he had no further return of fever. The spirillum was seen during the relapse.

1879. In the thirteen months prior to March, 1880 inclusive, 53 cases of demonstrated relapsing fever were seen, and 9 of these arose within the hospital precincts, viz. in March, 1879, two of a male ward assistant and a female patient, in June the two male sweepers of fever wards, and in July the assistant apothecary : in January, 1880, two medical subordinates (one of whom died), and in the following month a fourth subordinate and myself, the officer in charge.

In this series there was only one example of specific fever communi-

cated from patient to patient, in the well-ventilated wards ; it was the following :—

H. S., æt. 60, a destitute and paraplegic female, alone attacked of twelve others in a ward where an infected family was admitted, one member of which underwent an attack of specific fever just before she was suddenly seized. Her seizure included a characteristic relapse, and she barely escaped with her life.

Total admissions amongst women of spirillum fever 7, of communicated fever 1. There were three examples of male servants attached to the fever ward (20 beds, and total of 41 famine-fever patients), becoming infected in hospital : proportion 1 to 14 patients, and 3 out of 8 employés. They were as follows :—

1. N. P., æt. 28, one of three ward-boys, was an extra hand newly taken on duty, and 11 days after joining became suddenly affected with pronounced spirillum fever, which followed a typical relapsing course. The exact date and source of primary infection could not be learned : the two more habituated ward-boys he worked with escaped.

2. J. P., æt. 22, assistant-sweeper, had for a month been sleeping in the verandah of the same fever-ward, when he was suddenly seized with high fever and passed through a characteristic relapsing attack of much severity.

3. J. J., æt. 25, the remaining sweeper habitually on duty, was attacked three days after his assistant, with fever which displayed a typical course without distinct relapse. As in all cases of this series, the blood-spirillum was frequently seen during fever.

Analysis of Hospital Data.—Of all the particular evidence of contagion above adduced, that furnished by hospitals is most precise and best suited for analysis.

1. *Conditions of Infection.*—Total instances recorded 71 (omitting 2 not verified microscopically and 6 others overlooked, of patients seized in the wards). Of these, only 11 females. Mean age that of young adults. Season of the year had no apparent influence. At hospitals, the question of a local malarious agency must be excluded ; and the varying date and amount of acquired disease display a relationship rather to the changing numbers encountered, of sick persons likely to convey disease. Thus, at the J. J. Hospital between May, 1877, and May, 1878, whilst the mean monthly admissions of spirillum fever were 29, and that of infections 3·4, the maximum monthly admissions were in 1877, May 40 and August 50, and concurrent herewith the maximum number of infections (after allowing for delay due to incubation-period) were in June (7), July (6), and August (6) : so again, in 1878, April 50 admissions and 4 infections. It was further noticeable, that contagious events were commonest in those wards containing most fever patients : thus, the total means being 4·3 infections to 31·8 fever patients, in ward No. 11 the numbers were 8 and 64, in No. 12 ward they were 7 and 41, and in No. 13 (female ward) 8 and 42 ; these three wards are situated to the leeward of current winds, and this circumstance by lessening freedom of ventilation may also have been influential in favouring contagion, as would appear from the instance of ward No. 3, which although placed to windward is very ill-ventilated ; here there occurred no fewer than 7 infections upon 29 fever-admissions. Apparent discrepancies of both time and place of contagion were not wanting, and it appeared that some fever cases were more infective than others, also that possibly a certain immunity was at length acquired by individuals exposed ineffectually to infection ; but after much consideration, I was still unable to account for some anomalies of experience.

There is, besides, the significant fact—alone conclusive of the operation of fever-contagion, that all these instances of communicated disease happened among patients in the medical wards only, and only in attendants, clerks and medical officers employed in those wards: no surgical patient, servant, dresser or officer ever falling sick of the fever, though sojourning under the common roof. Even more, this rule was, at the G. T. Hospital, confirmed by the exceptional instance of a surgical clerk being attacked when to avoid overcrowding his ward had to lodge a few fever cases; and also by two other exceptional instances of surgical assistants becoming infected, only after assisting at the autopsies of fever patients.

2. *Prevalence of Hospital-contagion at Bombay.*—Admissions for spirillum fever being 500 in a total of 3,000 medical cases admitted during the given epoch, their proportion was 1 in 6. Of the 2,500 ordinary cases, 1 in 46 became infected from some or other of the fever-cases. Of the specific fever-cases, 1 in 8.5 gave rise to an infection of either patient or member of the hospital establishment. These data are derived from the J. J. Hospital statistics, under analysis; at the smaller and better constructed hospital, all the ratios were much less, and this is important because it is certain that some fever-cases at the G. T. Hospital were highly infective (see below, Inoculation Series).

Of the J. J. Hospital establishment, 1 in 7.2 was infected: viz. of ward-attendants only, 1 in 5; and of Native students about 1 in 4. One Native doctor in 4 was also attacked. The few European officers and the half-caste apprentices were much less implicated, being commonly much less exposed to risk. In sum, at this hospital during 1877-8, it was demonstrated that 500 spirillum fever admissions gave rise to 57 infections (and almost certainly there were more); the ratio being 1:8.77.¹ With such a strong tendency to spread in the comparatively open wards of a hospital, this fever must have been capable of wide extension in densely crowded tenements of the town; and for evidence that it did so spread, see some preceding paragraphs of the present chapter.

3. *Character of the Illnesses acquired by Contagion.*—The clinical records are still extant, and without exception I find the symptoms to have been identical with those of ordinary spirillum or relapsing fever (and of it alone), as displayed by the patients themselves. In a total of 54 survivors the type was probably the abortive form 17 times (about 30 per cent.), in 32 cases (60 p. c.) there occurred a single relapse, and 5 times (about 9 p. c.), two relapses. Some variations were noted here, in separate groups; and it is noteworthy that amongst infected patients and servants with a death-rate of 30-40 per cent. the acquired fever was as often single as relapsing; or, in other words, tended to resemble a typhus-attack: whilst among 13 Native students with only one death,

¹ The proportion of attacked in hospital to total admissions for relapsing fever in the London Fever Hospital, during 14½ years, according to Dr. Murchison (*l.c.* 1st ed.), was 1:440; at the Allerheiligen Hospital, Breslau, epidemic of 1868, it was 1:33; and during the epidemic of 1872-3, it was 1:23 (Dr. Litten, *l.c.*). Such is the remarkable testimony of progressive experience. Doubtless the late epidemic in Western India was unusually severe, yet I must add that perhaps unusual pains were taken by means of constant blood-testing to avoid, so far as practicable, the oversight of casual infections.

the relapsing type always obtained, exclusive of the casualty. All 18 (or 19) deaths took place during the first or invasion-attack, which is the commonest epoch of most deaths ; and the inclusive death-rate was over 26 per cent., which is far in excess of the common mean. Hence proof of the unusual severity of the spirillum infection in the tropical climate of Bombay.

As a final contribution to the evidence of the contagiousness of spirillum fever, my latest experience at the G. T. Hospital is subjoined apart.

Inoculation-series.¹

From being tantamount to deliberate pathological experiment, the following 6 instances of infection merit especial attention. They are all examples of disease acquired by *post-mortem* inoculation, and the dates of successive events being accurately known, they become serviceable in estimating the incubation-period under this condition :—

1. H. V. C., æt. 47, infected December 9, 1877, first fevered 12th–13th, interval of incubation 3–4 days or 72–96 hours ; a severe attack with one relapse. Had scratched finger at an autopsy made $3\frac{1}{2}$ hours after death, of a female patient dying in the critical fall of first relapse : spirillum not seen in her blood at this time, though previously found.

2. C. E., æt. 32, 1879, infected July 18th, 7 A.M., first fevered on 21st, 9 P.M., interval therefore $3\frac{1}{2}$ days, or 86 hours ; a severe attack with two relapses. Had scratched finger at an autopsy made $5\frac{3}{4}$ hours after death of a female patient, dying on 2nd day after close of first relapse : spirillum not seen in her blood at this time, though previously found.

3. R. S., æt. 26, 1880, infected on January 6th, 4 P.M., first fevered on 13th (precise hour uncertain), interval of incubation 7 days (or about 168 hours) ; a severe attack with two relapses. Had assisted (no wound known) at an autopsy made $1\frac{1}{2}$ hours after decease of a male patient, dying on 6th day of primary attack ; many spirilla in the patient's blood at this time.

4. A. S., æt. 38, same place and date of infection, first fevered at a similar hour, therefore incubation interval the same ; a pronounced single attack ending fatally with rebound of temperature immediately after the first crisis. Had assisted at the last-named autopsy, and had accidentally cut the left thumb.

5. R. S., æt. 18, 1880, infected February 16th, 11 A.M., first fevered on 23rd at 3 P.M., incubation interval 7 days 4 hours (172 hours) ; a marked single attack with no relapse. Had assisted (no wound) at an autopsy made 4 hours after decease of a patient, dying on 15th day (?) of fever, and then displaying many blood-spirilla.

6. H. V. C., again infected on same date and occasion as No. 5 above ; first fevered on 23rd at $3\frac{1}{2}$ P.M., interval therefore identical ; a marked single attack also. Had scratched the finger.

For convenient reference, the principal data of this important series are here tabularly arranged.

¹ When these instances occurred at the G. T. Hospital, Bombay, I was not aware that any like them were on record. I have since learnt that similar experience has been recently met with in Europe : *e.g.*, Dr. B. Lachmann of Giessen ('Deutsch. Arch. f. Klin. Med.' v. 27, 1880), mentions the case of Professor Perls, who became infected at the autopsy of a patient dying at acme of fever, whose corpse was examined 14 hours after death, when numerous dead spirilla were present in the blood. After 7 days Prof. Perls became feverish and the usual symptoms followed, except that pyrexia was of irregular type and the body-pains few ; a slight relapse ensued. The blood-spirillum was not seen till the 4th morning of invasion, and hence until then diagnosis was uncertain : such a feature attended my own last infection (see page 69). Some stress has been laid on the circumstance that the spirilla seemed to be 'dead' in the above-named patient's blood ; in my opinion, a motionless state of the organism is by no means a sign of its death. In the Berlin 'Med. Wochenschrift,' December 27, 1880, allusion is made in an Official Report to two other examples of infection at autopsy of a relapsing fever patient, a medical man and an attendant becoming ill after $7\frac{1}{2}$ and $8\frac{1}{2}$ days respectively.

TABLE XVII.—STATEMENT OF ACCIDENTAL INOCULATIONS AT AUTOPSIES, G. T. HOSPITAL.

No.	Wound.	Incubation Period.	Attack.	Severity.	Infecting Material and Conditions.
1	Yes.	72-96 hours.	Relapsing : 1 relapse.	Severe.	No spirillum in blood of corpse, but possibly germs ; see fig. E.c. PLATE 2. Blood two hours before death—plasma clear, fibrillation indistinct and partial, many white cells, large granule cells, and much free protoplasm swarming with active granules; red discs mostly dispersed. Many free, active granules, some in chains. No spirillum. Death a few hours after the acme of first relapse; last temp. $102^{\circ}6$, p. 144, woman then unconscious. Cause of death cerebral hæmorrhage. Palesplenic infarcts.
2	Yes.	86 hours.	Relapsing : 2 relapses.	Severe.	No spirillum in blood just before death, many white cells, plasma clouded. Decease at rebound 58 hours after crisis of first (?) relapse : copious epistaxis (many spirilla) at acme of relapse, and with the rebound femoral thrombus. Spleen very large.
3	No.	168 hours.	Relapsing : 2 relapses.	Marked.	Many spirilla in blood just before death, mostly of small size, (?) immature forms, many large nucleated cells. Death at end of invasion, last temp. $99^{\circ}2$, p. 140, probably lysis defervescence. <i>Typhus biliosus</i> of pronounced type. Splenic infarcts (red).
4	Yes.	168 hours.	Single	Fatal.	Infecting materials and conditions as in No. 3. N.B.—Death of No. 4 took place immediately after acme of fever. Previously many spirilla in his blood. A plethoric subject : cause of death 'fever : ' decease sudden : no jaundice. Autopsy not permitted.
5	No.	172 hours.	Abortive.	Marked.	Many spirilla in blood just before death, many immature forms. Death at end of invasion (?), last temp. $99^{\circ}2$, p. 106, probably lysis defervescence. Cause of death—copious gastric hæmorrhage : no vomiting. Splenic infarcts (red).
6	Yes.	172 hours.	Abortive.	Marked.	Infecting materials and conditions as in No. 5.

Remarks on the above Table.—Validity of these observations.—It is needful to state that all the individuals named excepting Nos. 2 and 3 (who had charge of surgical wards), were engaged in the fever wards of the hospital. It might therefore be said that they were habitually exposed to infection through ordinary channels; and this was certainly the case. It is, however, highly improbable that they were so infected: for, in the first place, they had been similarly exposed to contagion during several months previous to present illness, without ever suffering therefrom; and on the dates concerned, the likelihood of contagion was not increased by any unusual crowding of fever-patients. Further, the whole sequence of phenomena clearly pointing to actual infection about the dates of autopsy, it is in the highest degree improbable that infection took place in the wards at precisely the same date as the autopsies in question; and if the chances are few in favour of such coincidence in the earlier single instances, they become reduced to a minimum as regards the last two pairs of coincidences—it being practically inconceivable that upon two separate occasions, a double concurrence of infection should have happened. Lastly, 2 of the individuals concerned had not been habitually exposed to ward-contagion, and yet were attacked like the rest. I must, therefore, assume that all the tabulated instances are truly interpreted as being examples of incidental infection at autopsy.

Subjects.—All these men were adults in good health, and without evident predisposition to disease. European 1 (twice infected), Indo-Briton 1, Jew 1, Hindus 2.

Wound.—Four met with cuts or scratches, whilst examining the corpses; two were not known to have abrasions on the hands, though it is possible such might have been overlooked. Upon comparing the last two pairs of cases, it becomes evident that the presence or absence of a cut had no material influence on results.

Incubation-period.—The absolute identity of this period in the last two pairs of cases is conclusive, not only of identical infection, but also of an identical incubation-period succeeding a common infection: and this is a valuable datum. As to the first two instances of the Table, I note that in both the incubation-stage was materially and almost equably shortened: that of No. 1 being somewhat uncertain, because infection not being suspected the beginning of illness was disregarded by the subject (myself). In both cases, there is the remarkable fact that infection followed the inoculation of non-spirillar blood; and so far as I know, this datum is a unique one. Being intimately acquainted with all the circumstances, I feel assured there is no fallacy here; and, hence, that it is possible for the human subject to become infected at the post-critical period of spirillum fever. There seems to be no other explanation of this fact, than the supposition that the blood at this time may contain germs of the spirilla; and the data given with No. 1 amount nearly to a demonstration that such germs were really seen. According to my notes, the incubation-period of relapsing fever is commonly 7 days; and under certain conditions named, may be reduced to $3\frac{1}{2}$ days.

The attack.—Of 5 illnesses in survivors 3 were relapsing, 2 abortive; the single casualty took place at close of invasion-attack. This experience accords with the results of infection through ordinary channels;

and confirms the clinical distinctions made in the present work. The relapses also differed in themselves ; both second recurrences were pronounced ; the single recurrent event of No. 1 was brief, though sharp. I note that the blood of the patient suffering from *typhus biliosus*, did not induce jaundice in Nos. 3 and 4 ; and though once causing a fatal infection, yet once it led to a typical relapsing illness only. Full notes of all 6 cases are before me, which show them to have been in every respect identical with ordinary illnesses ; in 5 the presence of the blood-spirillum was demonstrated at each febrile event, and in the exception (No. 2), the sufferer (who went away) was well qualified to furnish memoranda conclusively proving the nature of his illness. The casualty was of a kind not rarely seen in hospital, and very instructive : see Chart 18, Plate V.

Infective Material and Conditions.—The details in this column speak for themselves. One case dates in 1877, when the epidemic had passed its height, and the rest at later periods, viz. 1879 and 1880, when public sickness had subsided ; still, I know the disease was the same throughout. There must have been some peculiarity about these four patients, whose corpses proved infective ; for in several other casualties at the G. T. Hospital, the autopsies made by the same individuals as those indicated in the Table (personally I always took part) were not attended with like calamitous results. More remarkable still is the fact, that at the J. J. Hospital, where 100 (or ten times as many) autopsies of fever-patients were performed (I was there, too, very rarely absent), no instance occurred of accidental infection. This diverse experience seems unaccountable, except on some such supposition as I have just offered.

Respecting the infecting material, so far as estimated by the microscope, I note that when the spirilla were present, a great many imperfectly formed filaments were to be seen in the blood. Whether these were in process of development or of decay, may be matter of opinion ; my own impression was that of active spore-production at the close of visible infection and of fever, and since from the above data it appears spores alone may infect, the greater their number the more likelihood of infection.

As presumed collateral evidence of the reality and infectivity of 'spores,' I may add that (contrary to observations made at Bombay) Drs. Albrecht and Spitz have both found spirilla in the blood of aborted fetuses, contemporaneously with maternal infection. As hitherto mature organisms have not been seen outside the blood-vessels, this datum bears the interpretation now suggested.

SUMMARY OF CHAPTER ON CONTAGION.

For complete demonstration of the identity of the Bombay fever with the Relapsing fever of Europe, there remained only to consider the Etiology of the disease, and especially to enquire whether or not Contagion were here concerned. I have now shown what were the Predisposing Causes of the local fever, and their similarity in detail to those of European forms will be sufficiently obvious. Respecting the influence of Contagion, it has been stated, first, that certain town-residents not themselves famine-subjects, but living in certain localities where such

subjects congregated, suffered early and severely : the necessary inference being through means of infection.

Next, 17 more or less detailed examples are given of spread of the spirillum fever amongst members of a family, who became affected only at successive intervals of a few days, extending over a period varying from 1 to 3 or 4 weeks : seldom did any member escape, and rarely did the disease spread beyond family-limits.

Next, I have quoted at length six instances of spread in a house or domicile, inhabited by associated individuals or by families. Here, too, the same successive order of infection, and the same limitation within very definite bounds, were to be noted.

In both these series, it was often possible to trace back the importation of fever to a certain date and a certain individual, whence its spread was clearly referable.

Then follows an account of some new hospital-experience ; many sick patients, ward-attendants and members of the Medical Staff acquiring the fever, after being in contact with the sick. A certain order of events was noticeable, even here ; and, particularly, must I insist upon the absolute limitation of hospital-infections to the medical side of each of the institutions concerned, where alone fever-patients were admitted. These events happened at all seasons of the year.

Lastly, I have narrated a series of inoculation-infections, which though connected with the recently dead subject, are none the less demonstrative of the contagiousness of spirillum fever.

Appreciation of Evidence.—Whilst each series of data is complete in itself, all are coherent ; and their conjoint testimony points, in my opinion, to the presence of an active contagium not possessing the diffusibility attributed to the so-called 'miasms.' In the absence of 'typhus' proper, malarious agency becomes the only other influence likely to have been present at Bombay ; yet had malaria been in such intense operation as the conditions would require, assuredly its effects would not have been limited to groups of individuals confined in a room (often an upper room), nor would they then have been slowly manifested in one individual after another. Moreover, malaria is a peculiarly seasonal production, and it does not give rise to a fever clinically like the spirillar.

Incubation-period of Relapsing Fever.—As commonly employed, this term applies only to the latent stage preceding the first or invasion-attack of fever. For accurate estimate of its duration, the date of effective infection should be precisely known, as well as that of earliest febrile manifestation ; but this is not always procurable in ordinary clinical practice.

From a large though not equally well-defined series of cases, Dr. Murchison reckoned the average duration at 5 to 9 days : the extremes ranging from *nil* (symptoms at once supervening) to 16 days. Dr. Litton, from a few data, estimated it at 3–9 days : Dr. Spitz at 7–8 days. At Bombay, many cases amongst strangers were seen, where the illness dated about a week after arrival in the town and consequent exposure to infection.

More precise data are furnished by experiment : thus, Dr. Motschut-

kowsky having inoculated fresh febrile blood on healthy men (number and dates of experiments not accessible), ascertained the incubation-period to be not less than 5 nor more than 8 days. Accidental inoculation at autopsies gave a period of about $3\frac{1}{2}$ to 7 days (*v.* Table XVII., page 404); and, in sum, I should judge the average period to be not more than a week. As regards extreme ranges of first incubation-period, the most precise information I possess is derived from comparative experiments, inference from which is doubtless applicable to man. Thus, whilst the mean duration in the monkey was about 90 hours, the average was rather less: the mean varied somewhat, according to severity of ensuing fever, being rather shorter in the severer cases; the extreme range was 12 to 186 hours, or possibly longer; and though I am not disposed to rely upon inordinate exceptions, yet at present it must be allowed that the primary febrile incubation-stage is by no means a fixed datum. The composite character of this latent period, as determined by the microscope, is alluded to at page 354.

Regarding the second and succeeding incubation-periods, which are commonly termed 'apyretic intervals,' their duration is stated in Table III., p. 129; briefly, it may be said that the first interval (*i.e.* the incubative-stage of second febrile attack) is in the mean longer than the primary period by a day or so (duration 7-8 days), and the succeeding intervals also tend to lengthen by 2, 3, or 5 (?) days successively; all the ranges here being considerable. This extension of the non-febrile intervals concurs simultaneously with an abbreviation of the febrile stages. See the following Chapter, page 414.

General Conclusions.—According to my observations, the laws of contagion in relapsing fever may be summarised as follows:—1. This disease spreads solely through means of actual contact with the sick. 2. A single individual may become the source of a new outbreak of fever. 3. Amongst a group of associated individuals, the disease does not appear simultaneously in all, but separately and successively at intervals more or less brief. 4. The disease is communicable during its successive febrile manifestations, and also for a short time both before and after the earliest of these. 5. A prompt reinfection is possible, no immunity being conferred by a first attack. When particular examples of propagation are considered, it is well to recollect that the manifest results of spirillar infection are highly varied; all febrile manifestations after the first (which itself may be no real exception), being liable to oversight. The application of my lately acquired data will explain many seeming exceptions and contrarieties in the operation of contagion, and the number of discrepancies yet unsolved may be expected to lessen with further additions to exact clinical knowledge.

Properties of the Contagion.—Though there be no abstract reason why the spirillar contagion should not be portable from place to place, or so rank that several persons should be simultaneously infected from the same source; yet there was very little evidence of these diffusible properties, in even a concentrated state of the poison. I never heard of an unaffected person conveying active infection to another individual. Nothing was learnt of the specific gravity of the poison, or of the influence of temperature on its diffusion. A tendency to adhere

to clothing and bedding, whilst retaining due activity, was conspicuous by its absence; for on particular enquiry I found the washermen of the hospitals never were affected by fever, and only once did it seem that a habit of sleeping on empty ward-cots might have led to the illness of a servant. Besides, my experiments on man and the monkey showed that blood infective when fresh, when dried lost its power of infection, although the spirillar filaments remained quite distinct.¹ Hence, at Bombay (as elsewhere) the rôle of fomites was not clearly indicated. Diffusion of the contagium through water was not ascertained, nor its convection by currents of air. Comparative essays have shown that the blood at first and second relapse of man, is at least as infective as at invasion-attack (see Appendix A, No. I.); and supposing that the spirillum is here essentially concerned, it is possible on analogical grounds, that its matured germs are capable of a prolonged existence as 'lasting-spores.' Future experiment must determine this point, and also whether or not infectivity diminishes on artificial culture of the organism.

Mode of Contagion.—Cutaneous transpiration and the breath of the sick are the probable channels of infection, the contagium passing inwards and outwards with equal facility. The critical sweat does not seem peculiarly infective. As the spirillum is absent from contents of the thoracic duct when abounding in the blood, the entry of its germs with the food appears unlikely. Men and women habitually handling the urinary and faecal excreta of fever patients, were not especially liable to fever. Spread of the disease by 'infection,' that is, through means of the more indirect channels, was neither indicated nor required upon theoretical grounds; actual contact with the sick being invariably possible, and commonly inevitable. The less comprehensible side of experience is the comparative rarity of illness, after repeated contacts at all stages of spirillum fever; for even the close and prolonged association of a suckling infant with its parent, may not eventuate in its illness until after the mother's relapse. Briefly, whilst the fact of this disease being communicable by contact is undoubted, little else is known except that its propagation is most certain where people are huddled together, and much less frequent in the absence of overcrowding. The records of family and ward infections show this. It is quite noteworthy that illnesses resulting from direct inoculation with contaminated blood, precisely resemble those acquired through ordinary channels; as regards both time of advent and general character. Hæmorrhagic effusions (e.g. of epistaxis) may contain many spirilla, which are also present in the *menses*.

Anomalies of Infection and their Explanation.—To account for seeming vagaries in the transmission of spirillum fever, under conditions

¹ Dr. Motschutkowsky (*L.c.*) found that blood kept at 60° F. in a capillary tube (the spirillum still active), remained infective: when diluted with 0·1 per cent. solution of Quinæ Hydrochl. it was effective, though the organism became quiescent; the addition of alcohol rendered blood inoperative. Respecting infection through *fomes*, it is proper to add that several observers from Cormack (1842) to Clark and Parry (U.S.A. 1869), have supposed that the clothing of fever-patients is a medium of contamination. Not all early original writers, however, mention this mode of transmission; and, so far as I know, no late observer acquainted with the blood-spirillum has adduced valid evidence of infection through fomites.

outwardly similar, there is supposed to exist a varying susceptibility in the individuals exposed to infection, which becomes revealed only upon trial. So far as appears, ordinary indisposition or disease does not either predispose or protect the individual. The alternative supposition is that the spirillar poison itself is practically effective only at particular periods of fever, such epochs being probably very brief, even if repeated: they must also be rare, for the great majority of exposed persons wholly escape illness.¹ Either of these suppositions is consistent with known data: and it is conceivable that contagion becomes effective, only where there happens the conjunction of active germs and a susceptible recipient. The phenomena of inoculation-experiments dispose me to think that a personal predisposition is the least influential of the above-named conditions. There is historical testimony that different epidemics of relapsing fever display different degrees of infectivity; and there are good reasons for supposing that during the same epidemic, different cases of the disease vary in their infective power. Whether or not contagion is commonly most evident at the beginning, height or decline of an epidemic, does not appear; certainly at Bombay, the later cases proved not the least infective.

There is no clear evidence that long and unscathed contact with the sick, confers positive immunity against infection. Nor am I acquainted with instances of infection '*à petite dose*,' such as have been assumed for the other continued fevers of man; but the possibility of this phenomenon must be allowed. In the spirillum fever, the immunity conferred by an attack, if real, must be comparatively very brief.

¹ See the first Appendix, page 431 (near the top), for intimation of increscant infective properties of the blood in relapsing fever. If the relapses of the spirillar infection represent its maturer conditions, infectivity may be greatest in them. Typhus fever, also, is said to be most contagious towards the close of illness (*v.* Dr. Murchison, *l. c.* p. 93). My comparative experiments further indicate a possible augmentation of infectivity, by transmission of the spirillar virus through the bodies of certain individuals.

CHAPTER III.

NATURE OF THE DISEASE.

UNDER this heading I propose to discuss briefly the spirillum fever as a whole ; explaining, with as little speculation as possible, the essential relations of the disease (A) ; its more characteristic symptoms (B) ; and, lastly, the conditions of its appearance as an epidemic (C).

A. In the current nomenclature, Relapsing fever is placed amongst general diseases or those affecting the whole body ; coming after enteric fever, and preceding the malarious. Officially, it is included in the Zymotic Class, under the Order of Miasmatic diseases.

Respecting these assigned nosological relations, it is permissible to remark, that only with considerable limitations can spirillum fever be regarded either as a disease of the whole body, or as being due to a miasmatic influence.

In a more precise sense, the 'fever' belongs to the group of acute infectious diseases arising from a specific blood-poison : its acute character being obvious from the febrile manifestations, its infectious properties from its constant reproduction in fresh subjects, and its specific nature from its always preserving the same features. The fever is distinguished clinically by its tendency to recur spontaneously a certain number of times, at definite brief intervals and in diminishing degree ; but it is by no means the only fever which 'relapses.'

As regard the poisons which are here the essential cause of illness, they are chiefly known by their effects whilst present in the circulation ; yet they have a separate existence, possess physical qualities and may be transported from place to place without losing their virulence. When isolated from the body they are termed 'contagia.' As the result of experiment, it is known that contagia exist as solid particles ; nothing quite definite has been learnt of their chemical properties, but by aid of the microscope minute organisms (termed *bacteria*¹) become visible in liquids proved to be infectious, and the question has arisen whether or not these organisms *per se* represent the specific virus. Some observers answer in the affirmative, whilst others regard the bacteria as simply associated with the virus ; at present the data are somewhat uncertain, but proof seems to be accumulating in favour of the affirmative reply. Meanwhile, the

¹ More precisely 'pathogenic' bacteria, from their power of producing disease in the body. Their aspect differs but little from that of organisms met with outside the body, in various free organic media. There are no bacteria in healthy living blood, and the healthy tissues display a positive resistance to the entry of most foreign organisms. Once implanted in the body, such growths may be regarded as truly parasitic. They may flourish in the tissues as well as in the blood, and not be present at first in the circulation.

presence of these organisms furnishes a ready means of recognising the specific poison. It seems that every infectious disease has its own organism, which presents unaltered characters so long as it remains visible; and it has been ascertained that for a time more than one organism may co-exist with another in the blood. This is an important clinical fact; for as the pathogenic bacteria are connected with definite symptoms, some anomalous variations of specific diseases may hence be referred to an admixture of poisons. By experiment, it is known that when two specific poisons are present together, one soon predominates and eventually excludes the other. (Dr. R. Koch, 'Wundinfectionskrankheiten.' Leipzig, 1878.)

The organism which belongs to relapsing fever is the spirillum (or spirochæte) above described; and its equable distribution in the circulation and ready detection in the blood, renders the study of relapsing (or spirillum) fever of unusual interest, as being likely to assist the investigation of other acute infections with less accessible blood-phenomena. There is to consider first, whether or not the spirillum is wholly peculiar to relapsing fever; and next, what are its relations to the disease.

1. *Range of the Spirillum.*—I have examined microscopically the blood of many patients suffering from various febrile and non-febrile diseases, with the express purpose of ascertaining if in them the blood-spirillum is ever present. There were special reasons for making this enquiry, and it was done with care. The result has been a firm conviction that under common modes of research, the organism is to be found only in a certain disease possessing the attributes of so-called relapsing fever. It has been detected when symptoms were at first of doubtful clinical import, but then the previous history, subsequent course of illness or *post-mortem* revelations invariably confirmed the diagnosis indicated by its presence; and it was never found except under such affirmative conditions. In some instances of practically undoubted relapsing fever, the organism was not seen; but only when the completer methods of examination were not applied, or when specific pyrexia was not present: and, briefly, my experience has been in entire accordance with that of most observers in Europe.

The cases scrutinised occurred chiefly during the late epidemic, and at the J. J. Hospital; they amounted to 393, with 91 deaths—there were Ague 70 cases; Remittent fever 101, Pneumonia 72, Dysentery 28, Bronchitis 13, Meningitis 5, Cerebro-spinal Meningitis 1, Hepatitis 3, Enteritis 3, Nephritis acuta 2; to the best of my belief, none of these affections were mere 'complications' of relapsing fever, and they could only be regarded as substantive diseases. There were 2 instances of fatal enteric fever, and 6 of a typhus-like character; besides numerous examples of hectic fever in various forms, and chronic rheumatism, syphilis, scurvy and the like, with slighter febrile and non-febrile affections examined as tests. At the G. T. Hospital I omitted no opportunity of testing in this way the diagnosis of fevers; and I never met with contradictory testimony.

2. *Relationship of the Spirillum to the Disease.*—The term disease including all the phenomena ensuing upon infection, there is to notice a

prior non-febrile state and a final febrile manifestation. Respecting the apyretic condition, few or no definite symptoms have yet been recognised; yet it is certain that morbid changes are present, for there is ocular evidence of blood-contamination at this stage of disease. Ignorance of the elementary aspect of the spirillum may account for its non-detection from the first brief interval after effective inoculation, but it has been learnt that so early as 16 hours afterwards the organism becomes detectible in the blood of monkeys (Appendix A, No. 1), and always it is present for a more or less prolonged period of time before pyrexia sets in. During 'fever' the organism is invariably present in augmenting amount, and finally pyrexia and the visible blood-contamination abruptly come to end. Not to interrupt description, I note that without any interference from outside and in spite of therapeutic effort, the entire series of phenomena is again reproduced, and this for once or twice more. Throughout, the visible blood-contamination is first in order of time; and it must be conceded that manifest 'disease' follows such contamination as represented by the spirillum.¹ Regular relapsing fever was never seen without spirillar infection: and though the infection may fall short of producing such clearly recurrent fever, yet it always tends to induce it. The question whether or not the spirillum alone and *per se* produces the fever, cannot be answered until the organism be completely isolated, and its pathogenic power tested then as well as after cultivation in other media than the blood. At present no pathologist has succeeded in doing this; but in some comparative instances (*e.g.* the organisms of anthrax and some kinds of septicæmia), such testing has been accomplished, with the result of demonstrating these parasitic growths to be the *veræ causæ* of disease.

There remains to note that 'fever' consists of two parts, viz. of 'pyrexia' or preternatural heat of the body, *plus* 'functional or organic derangements' ensuing. Though clinically the most important phenomenon, yet from a pathological point of view, fever is but the culmination of infection; it varies more than the visible blood-state, and it may be so reduced in prominence (*e.g.* in certain comparative inoculations and at the relapses of man), as to appear quite subordinate. As to the functional and structural derangements attendant upon high and sustained pyrexia, their relationship to the spirillum seems to be mainly a collateral one. None of these derangements are absolutely peculiar to relapsing fever; though their relative frequency, sequence and combinations are more or less characteristic of the spirillar infection.

Recurrency of Spirillum Fever.—There is always present and commonly manifested, a tendency of the disease (as estimated by 'fever') to recur at definite periods; the successive recurrences becoming briefer and separated by longer intervals. *Vide* Table III., p. 129. Recollect-

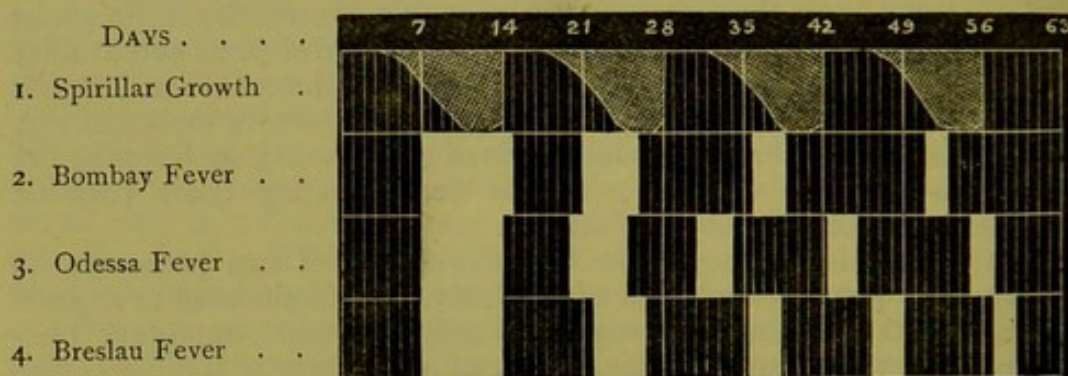
¹ In so far as spirillum fever ensues upon lesion of the blood, it is 'symptomatic' of such lesion; and so in a similar sense are all the acute infections. In purely inflammatory diseases the order of events is different—evidence of blood-poisoning supervening on local lesion; yet at Bombay it was maintained that the blood-spirillum simply attends upon various febrile affections, *e.g.* the pneumonic; and, elsewhere, it has been argued that the 'soil (*i.e.* the blood) becomes suitable for development of the organism only during the febrile process.' As matter of exact and repeated observation, the succession of phenomena is precisely the reverse; fever always following visible blood-infection. That pneumonia or even another 'fever' can co-exist with the spirillar, is likely enough; but their true relationship need not be confounded.

ing the circumstance (which may be termed a biological fact), that each apyretic interval is strictly the incubation-period of the following febrile event—the two forming a complete cycle, it will be found that these inclusive phenomena of a complete relapsing attack present a certain order, which cannot be regarded as accidental.

Though abnormally placed in the blood, the spirillum as a living plant must be supposed to retain something of that periodical order of growth which belongs to the vegetable kingdom ; and since it first appears, and then reappears, solely in connection with the febrile stages of disease, the inference becomes inevitable, that the periodic recurrence of fever is intimately associated with corresponding growth-stages of the parasitic organism. From this point of view, the tolerably regular succession of relapses becomes comprehensible ; and since in the blood is not the natural habitat of the spirillum (as is shown by its more or less rapid decay), it would be sufficient to establish its association with the morbid phenomena, if a moderately exact conformity in time be made apparent.

The following scheme shows the periods to be compared : I have added to those of Bombay some clinical data from other towns in

CLINICAL AND BIOLOGICAL SCHEME OF RELAPSING FEVER.



The light bars are spirillar manifestations and febrile attacks.

Europe. The upper series of pale bars (1) represents successive stages of spirillar manifestation in the blood, that first occurring being repeated with hypothetically undiminished vigour ; the white bars below represent successive febrile attacks, according to their actual mean duration at Bombay (2), Odessa (3) and Breslau (4). The blank spaces are all incubation-periods.¹ According to this diagram the spirillar development in the blood, as estimated by the febrile attacks, tends to re-appear at progressively earlier intervals lasting also for a briefer time at each successive manifestation ; this order of phenomena indicating the manner in which infection subsides. According to Bombay experience, however, the

¹ The same primary incubation-period of 7 days is assumed for all fevers. The succeeding periods, febrile and non-febrile, for Bombay are contained in Table III. ; those for Odessa are as follows, mean duration in 148 cases—of febrile attacks in order, 6½, 5½, 3½, 2½, and 1½ days ; of the apyretic intervals in order, 5½, 6½, 9, 10½ days. (Dr. Motschutkowsky, 'Deutsch. Archiv. f. Klin. Med.' Nov. 1881.) The numbers for Breslau (about 400 cases) mean duration in same order are—6·6, 4·9, 3·1, 3·1, 2·3 days for febrile attacks ; for apyretic intervals, 8, 9, 6·3, 8·3 days (Dr. Litten, *loc. cit.*) There is rather the most appearance of regularity in the Bombay series, which were microscopically identified ; but a certain variation may obtain, without it being necessary to discard the data or the hypothesis founded upon them.

later febrile attacks fall within the supposed typical spirillar periods ; but such is not quite the case with the European disease, in which the 3rd, 4th, and 5th attacks, whilst still preserving a common declining and deferred character, are found to take place distinctly earlier than in the Eastern fever. Experience at Odessa and Breslau is enough alike to show that here is no mere accident. Divergent clinical experience is little marked at the first attack, but becomes more and more apparent with the relapse ; it would be easy to speculate on its likely causes.

That fever should sometimes not recur is another contingency open to speculation, as to the possibility of the implanted parasitic infection itself then being slight or peculiar, or the state of the host being unfavourable to its ordinary reproduction. That the organism may duly re-appear in the blood, with but little febrile disturbance ensuing, I have already demonstrated ; perhaps this event is commoner than yet recognised. (See p. 165.)

The nature of late relapses without visible blood-infection, is open to varied interpretation. From their paroxysmal character, they have been compared to superadded ague ; yet the fact of their preserving a certain periodicity, points to their essential connection with the preceding spirillar manifestations. As residual phenomena, their uniformity in time is commonly much less clear than at earlier attacks. It is not unreasonable to suppose that as the spirillar manifestations become weaker, they also tend to be less regular ; and, besides, after repeated auto-inoculations the system itself will re-act differently. There is no evidence that it becomes indifferent to the spirillar infection. Individual constitutions varying, varieties in febrile attacks become explicable on this ground ; as well as from original difference of degree and quality of infection.

Lastly, true recurrency is a feature not peculiar to the spirillar infection, being also met with commonly in malarious fever, frequently in typhoid and sometimes in anthrax.¹ It pertains, as well, to hectic (or septic) fever ; but, on the other hand, is not a recognised character of typhus. With respect to the malarious and typhoid infections, I note that they too are occasionally attended with 'secondary fever,' which may simulate a true relapse ; and there are some other specific fevers displaying such consecutive pyrexia, *e.g.* variola and yellow fever. From these analogous instances, my observations on the spirillar disease seem to derive both confirmation and extension.

B. EXPLANATION OF THE CHIEF SYMPTOMS.

These are either (*a*) common to most specific infections, or (*b*) more or less peculiar to the spirillar infection.

¹ 'From the intermittent character of anthrax, which is frequently observed, and the paroxysmal nature of the symptoms, it is certainly allowable to assume a corresponding appearance of the bacterium *en masse*, and a corresponding disappearance of the same. In this way, the coincidence of the bacterium with the attacks, would take place analogously to the appearance of the *Spirillum Obermeieri*.'—Dr. Bollinger ('Ziemss. Cyclop.' Americ. Transl., vol. iii., 1875).

To the list named in the text may be added a fever attributed to contaminated milk (Dr. Ewart, Proc. Roy. Soc. Lond. No. 215, 1881) : also trichiniasis, dengue and some epidemic pneumonias (so-called). A similar tendency is exhibited by some chronic infections, as the leprous and elephantoid (or filarious).

(a) Well characterised as is this disease by its attendant pathogenic organism, still its general symptoms and lesions do not differ essentially from those of other acute infections ; the great majority of clinical phenomena, from first to last, being fundamentally alike in man, and hence it would appear that the system reacts similarly against each of the ordinary specific poisons. 'Fever' is the most prominent sign of such reaction ; how it is produced is not precisely known, but of the conditions attending its production and course something may be learnt from study of the spirillar infection. The origin and character of ordinary complications and sequelæ of fever, are also better known empirically than understood ; but here, too, my enquiries are not wholly insignificant, if it be allowed that a certain bond of relationship obtains amongst the groups of acute infections.

(b) Pre-febrile State.—The practical absence of morbid signs during the earlier part of incubation-periods (taking the first one as type of the rest), is coincident with absence of visible blood-contamination ; and it may be said that *premonitia* tend to appear, with the advent of visible infection (see page 69). The gradual development of the parasite in the blood, should give rise to a progressive systemic disturbance ; and doubtless this is so, although I am unable to prove it in detail. From the data given at page 123, it did not seem there was pre-febrile augmentation of the daily urea, even on the two days before relapse, when some spirilla were already present in the blood (Chart 2, page 358) ;¹ at this time, the temperature and pulse are not materially changed.

Fever.—The onset and maintenance of pyrexia is consentaneous with increased production of the blood parasite. Partly from observation of the spirillar disease and partly from some analogous ague-phenomena, I am led to connect the pyrexia with increasing growth and spore-production in the blood. The initiation of spirillar pyrexia resembling a nerve-explosion, it probably ensues after an irritation accumulated to a certain point, corresponding with some stage or degree of bacteria-growth. The maintenance of fever is strictly commensurate with sustained parasitic manifestation (apparently due to successive broods), degrees of each being probably connected at their root ; and the abrupt end of fever is unquestionably related to the cessation of parasitic growth. The variations of specific pyrexia seem best explicable by variations in luxuriance of the spirillar infection ; and some exceptions are not inconsistent with this view, if allowance be made for incidental states of the system.

It is found that the excretion of urea is promptly and largely increased during the fever (v. p. 123), and for some time afterwards the amount continues to be considerable ; the maximum daily quantity was on the second day after crisis. The chlorides in the urine diminish towards the acme ; albumen in small amounts is then apt to appear.

As to Relapses, there is, on the whole, sufficient evidence of their increasing brevity being connected with a diminishing parasitic production. Variable phenomena are, however, common at recurrent attacks ;

¹ Dr. S. Ringer found that in ague an increased discharge of urine distinctly precedes the onset of pyrexia ('Med. Chir. Trans.,' London, v. 42), I have not been able to refer to other analyses of the urine in relapsing fever.

and this might be anticipated, since the human frame is not either the natural habitat of the spirillum or an inert substratum for its growth.

The peculiar dusky, haggard *facies*, often pallor and lividity of the skin, and unequally distributed temperature of the surface (see page 71), which are noted during advanced specific pyrexia, seem referable to a charging of the blood with foreign organisms and a consequent impeded circulation. There is contemporary evidence, also, of more or less acute congestion of lungs, brain and liver, and of renal blood-pressure in the increased flow of urine: the spleen seems to be especially implicated. Moreover, the large consumption of oxygen and of albumen by the vast numbers of parasitic organisms in the blood (see p. 350), must contribute to the morbid signs, as well as to the emaciation, excessive thirst, and perhaps the craving for food occasionally expressed.

In severe attacks the symptoms are urgent, and complications threaten; then, too, visible blood-changes may be pronounced or peculiar, and this conjunction was often enough noted (in even cursory microscopic examinations) to warrant its being regarded as more than accidental. A narrow interval here seems to separate the severer from the complicated or fatal cases, and this contingency is explicable by the varying luxuriance or quality of specific blood-growth. The tendency of the spirillum to cluster into meshes or become aggregated into compact masses, which might act as 'emboli' in the smaller vessels of brain, lung, skin, serous and mucous membranes, has been distinctly noticed during life.¹

Hurried breathing and rapid, weak action of the heart, with the small, feeble, yet not dicrotic pulse, are probably connected with peculiar irritative and physical states of the blood, in addition to a metabolic

¹ As evidence of similar views, also based upon observation, I am able to quote some remarks of Dr. L. Heydenreich ('Klin. u. Mikrosk. Untersuch. ü. d. parasit. d. Rückfallstypus,' Berlin, 1877), who mentions two instances seen at St. Petersburg, in which there was reason to suppose the spirillar clusters had formed within the blood: both were cases of petechial typhus biliosus with pronounced symptoms, the peripheral circulation being depressed and the limbs cold, whilst the trunk was warm; a cyanotic aspect was present in one. As repose favours such clustering outside the body, it is noted that in places where the circulation is languid, e.g. the spleen, bone-marrow, nasal cavities, skin, &c., the possibility of thrombi, infarcts, abscesses, hæmorrhages, petechiæ, &c., becomes explicable. Such hæmorrhages from plugging of the vessels, would more readily take place when the lining membrane of the vessels is affected, and degenerated endothelium becomes detached and free in the blood-current.

The brain with its pia-mater may be mentioned in this connection. —H. V. C.

Dr. Heydenreich previously alludes to other blood-changes, as follows:—The appearance of all these structures (*i.e.* augmented white-cells, granule-cells, endothelium and free granules) in great quantity within the blood, at the end of the febrile attacks and especially just after the crisis, is a fact of special interest. Their advent is commonly abrupt, and similar abrupt, though scantier, outpourings may be noticed during the course of specific pyrexia. The largest proportion of these abnormal ingredients consists of degenerated protoplasmic bodies and white cells. Such structures are not absolutely peculiar to relapsing fever, being found in the blood of patients suffering from inflammatory and other infectious diseases; but there is no disease known in which such a remarkable and abrupt influx of them takes place, at a definite time. In other diseases these bodies are seen more rarely, in smaller quantity and at separate periods of illness. From microscopic examinations of the spleen-pulp, of splenic, arterial and venous bloods, and from observations on the contractility of the spleen, it may be said with much probability that, at least, the greater part of these abnormal elements are derived from the spleen; out of which they are, as it were, squeezed into the blood, by contraction of the spleen towards the end of febrile attacks. The recent observations of Dr. Roy on the rhythmical contractions of the spleen, may here apply in an obvious manner (*Lancet*, 11 Feb. 1882).

deterioration of its qualities. It is remarkable there were but few signs of deranged renal function, although the kidneys may be much implicated; uræmia was never declared during this brief form of fever. Even the equally implicated liver seemed to suffer but temporarily, whilst the local signs might be very marked. So with the muscular system; and, in general, parenchymatous inflammation or cloudy swelling was a change which, if occurring at all amongst survivors, did not *per se* entail serious or persistent injury. Commonly, therefore, enlargement and tenderness of the spleen and liver, and tenderness of the epigastrium may be referred to temporary difficulties of the circulation: so probably, tenderness and enfeeblement of the voluntary muscles, with pains in the locomotive apparatus generally.

Acme and critical perturbation.—The remarkable congeries of symptoms seen at acme (*vide* p. 72), cannot be dissociated from the contemporary state of blood and spleen (see pp. 114 and 338); both series of phenomena alike supervening abruptly, and lasting for only a brief period; and, further, degrees of each seeming to correspond. Even in ordinary cases, the troubles of circulation and respiration noted at acme, may be explained by visible changes of the blood-state; the stress passing away without entailing risk to life. In severe and fatal cases, events are intensified; and with the more urgent symptoms, there concurs a true 'secondary' contamination of the blood through the excessive influx of spirillar relics and morbid spleen-products. It is to such secondary or quasi-septic contamination, that I should attribute many hæmorrhages and local complications; for obviously the clusters of leucocytes, large granule-cells, granular protoplasmic masses and endothelial cells could not traverse the capillaries without sometimes leading to blood-stasis. Further, as some of these structures are in a state of fatty degeneration, there is here evidence of a profound metabolic change which cannot but be injurious. According to my clinical and autopsic enquiries, there is at this epoch often a distinct conjunction of irregular pyrexia, typhoid symptoms and accidents, with an irregular spirillar manifestation of abundant free granules and quasi-immature filaments, and with hyperplasia and fatty degeneration (infarction) of the spleen-pulp. As to a depurative action on the blood of liver and lungs, such must necessarily be defective; and in fact these organs themselves invariably suffer: nor are the kidneys spared. Since the aspect of the blood, under ordinary inspections, is not quite uniform, it becomes necessary to recognise various kinds as well as degrees of cell blood-poisoning; but what are the several effects of the morbid elements named, cannot as yet be stated, nor how far with each variety a noxious chemical influence is combined. It is likely that a true septic element (either autochthonic or extrinsic) is combined with the spirillar infection, in the worst cases: see below. This inter-relation of unusual morbid phenomena may be manifested prior to acme-date, or as soon as the spirilla have become numerous, the fever pronounced, and the spleen enlarged; and after the crisis, splenic contraction with resulting blood-contamination, may continue or increase: clinical (besides anatomical) evidence to this effect being furnished by the occasional early appearance of severer fatal symptoms, and in post-critical secondary fever and pneumonia.

The other phenomena of crisis indicate a marked impression on the nervous system, which commonly is but temporary; and if lasting so long as two or three days, does not entail more than the delirium of inanition or some other functional derangement due to depression of the circulation.

Lysis.—Gradual decline of specific pyrexia was always serious, from its being accompanied by prostration and the signs of secondary blood-poisoning. The spirillum may persist to the end; but since at the same time there is evidence of cell blood-contamination, I came to view 'lysis' as being essentially a kind of premature and prolonged 'acme.' Complications are very apt now to arise, or to occur afterwards; and the typhoid state supervening, death by exhaustion may ensue in the form of gradual collapse. Though some patients recovered, it was always evident the specific attack had been unusually severe in these lytic cases.

Bilious typhus.—This variety of spirillum fever was essentially an irregular and usually a lytic form, accompanied by deep jaundice. In my experience, the cases resembled a spirillar infection early attended with other blood-poisoning, which could generally be traced to the spleen; and hence the view that the 'secondary' blood-contamination was the cause of the severe symptoms generally present. These include a decided tendency to the typhoid state, such as is manifested also in lytic attacks; the pyrexia seldom being pronounced. Jaundice seemed no more than an incident of the general bad state; there being seen cases precisely similar, except in the absence of this symptom. And, besides, deep jaundice may be present without entailing excessive risk.

Accidents and Complications of Specific Pyrexia.—Local congestions, hæmorrhages and inflammations were referable generally to the state of the blood. The first-named supervened late in the febrile attack, and, to judge from post-mortem appearances, were probably due to localised obstructions of the circulation leading to repletion or rupture of the smaller vessels.¹ Such obstruction, when occurring prior to acme, might arise from spirillar aggregations; but emboli of this kind were not seen, even in minute examination of skin-spots: and this is no matter for surprise, since such delicate filaments are exceedingly difficult to find in uninjured vessels, and would be destroyed in a hæmorrhagic effusion.

¹ That emboli and hæmorrhages supervene upon plugging of blood-vessels by parasitic blood-growths, is a datum now sufficiently recognised in Pathology; but I may allude to some information respecting Ague, gained at Rome in 1881. Dr. Marchiafava ('R. Accad. d. Lync.' 1880-1) has lately recorded two instances of pernicious ague ending fatally from diffuse, punctiform cerebral hæmorrhage; this occurred at the acme of fever. On microscopic examination, each hæmorrhagic spot was found to present in its centre a small vessel distended with white cells enclosing minute rounded granules of uniform aspect, and with free accumulations of such granules, which were regarded as the micro-organisms of malarious infection. In another case there was gastric hæmorrhage, and the liver was marked with pale conical infarcts, composed of lobules the capillaries of which were so distended with white cells and small accumulations of bright granules as to compress the neighbouring portal vessels. A connecting link here is the fact that in the blood itself such granules and even bacillary filaments (see Z, Plate II.) have been found abundantly at the onset of pronounced ague, and then only: the resemblance, as regards periods of fever, corresponding, so far as I could learn, with advent and disappearance of the spirillum. According to this interpretation, the date of risk is the time when the blood-organism resolves into spores, which in severe illnesses abound and persist for a time.

I have known cerebral hæmorrhage to occur when the blood contained large, sluggish and adhering spirilla, which at the time suggested the possibility of their blocking smaller vessels; and as it is known the unsupported cerebral capillaries are liable to acute fatty degeneration (see page 253), the occurrence of this accident and the like becomes explicable. Commonly, vascular infarctions are later in occurrence, and presumably due to large-cell contamination of the blood.

Of local inflammations, pneumonia was much the commonest and most serious: it supervened at or shortly after the crisis, and while doubtless made liable by prior febrile congestion, yet when of the lobular and disseminated form might be referred to blood-poisoning of the consecutive kind. Its occurrence was independent of season. It has been known to supervene some weeks after crisis, being then probably sometimes a rekindling of latent disease. But that after the close of spirillar infection, the blood for a time may remain contaminated, was shown by the occurrence of hæmorrhagic skin-spots and of parotitis on days following the crisis of invasion and (seldom) the relapse. Sometimes it seems that such contamination becomes increased about the date of relapse, *e.g.* by parotitis then coming on; and at such period, the splenic disturbance again takes place.

Secondary fever, occurring as a 'rebound' after crisis, was seldom accompanied by fresh local lesion; and then, as well as later, did not seem due to reaction of the nervous system. Commonly not serious, it sometimes ended fatally, and as then there were the marks of blood-contamination in diffused lesions, I could not but refer the pyrexia to such blood-state.¹ A rekindling of hepatic or splenic parenchymatous inflammation was sometimes indicated, or, at least, attended secondary fever; and I was led to suppose that such tissue-lesion must entail febrile disturbance: all the cases recovered, and the local signs being but little marked, it was evident the inflammation was not of the common acute character. Upon review, I regard this reactive phenomenon as commonly a result of the excessive blood-contamination which is liable to ensue at close of specific fever.

That the ordinary sequælar events are but incidents, is shown by their occasional entire absence; they chiefly pertain to the nervous system, and indicate its considerable implication during acute spirillar infection. The severe aching pains are not traceable to muscular degeneration, and such degeneration occurs in other fevers without the pains; if the bones of the joints ever suffered from semi-necrotic changes at Bombay, the signs of this were not apparent. Sequælar emaciation and debility are comprehensible on ordinary principles; they were not seen in the better nourished subjects.

¹ Reference is here made particularly to diffuse inflammation of the mucous membrane of the alimentary canal. See page 215 for examples of Enteritis in conjunction with, and as a sequel of, the spirillar infection. To be compared with the Gastritis and Enteritis found in monkeys dying after inoculation with human saliva, and the Enteritis in other animals dying after septic infection (Dr. Sanderson in 'Rep. Med. Off. Priv. Co. and Loc. Gov. Board, new ser., No. 6, 1875. Appendix, p. 63).

That some forms of consecutive fever may be of a septic nature, is evident from the concurrence of pyæmia with the spirillar infection—*e.g.* with pneumonic complication (*v.* p. 202 and CASE LXX.) Dr. Lüdimoff (*l. c.*) in a fatal case of bilious typhus with the spleen infarcted, ruptured and containing small abscesses, found micrococcus-colonies in connection with the abscesses and in the splenic vessels.

In conclusion, whilst most symptoms of spirillum fever resemble those of other fevers, some are peculiar, and a liability exists to special complications. Many of these latter phenomena are directly explicable by the visible blood-changes alone, or chiefly ; and this may be said without overlooking chemical and dynamic agencies of a less obvious kind. The distinction here made of a 'primary' and 'secondary' blood-contamination, being founded upon actual observation, is probably of general application in the acute infections ; and the foundations of an amended pathology of 'fevers' are hereby indicated.

C. SPIRILLUM FEVER AS AN EPIDEMIC.

Origin of the Disease.—At Bombay, no precise information was procurable ; but as the 'new' fever seemed to appear simultaneously with famine in the country districts of the Presidency, I may briefly allude to the possibilities of an 'independent origin' of the disease. This expression necessarily refers to the origin of the specific blood-poison : and if such poison consist of unorganised material, speculation concerning its casual production cannot at present proceed in any definite direction. Supposing, however, that the blood-spirillum in some way represent the blood-poison, it becomes conceivable that in certain impaired states of the frame (the result of starvation and bad hygiene) it might originate by a spontaneous variation of the spiro-bacterium of the saliva (*e.g.*), which acquiring it may be pathogenic properties, is adapted to enter and live within the circulation. Or the organism might not be autochthonic, but derived extrinsically ; *e.g.* from the spiro-bacterium of impure water, which in a similar impaired state of the body finds an effective entrance, otherwise debarred to it under healthy states. Possibly, germs of the spirillum are commonly present, but remain inert from absence of a *nidus* in the normal blood ; meeting in it when becoming impoverished, a suitable soil for some stage or degree of their development. Such germs produced during previous illnesses might long lie dormant in the earth or a building, *e.g.* as 'lasting-spores,' until a return of the conditions adapted to their growth ; and thus would be accounted for, the seemingly independent origin of some fresh epidemics.

A purely spontaneous origin of the infection being absolutely inadmissible, it might be said that could any of the above suppositions be substantiated, a modified spontaneity of relapsing fever would become comprehensible : at present I know of only a few analogous facts tending even remotely to their support, and of more negating their admissibility.

The clinical difficulties often met with here being the absence of a history of contagion, latency of the disease for months or years and its reappearance with return of certain conditions, I am of opinion that on the hypothesis of an independent origin even more serious objections would be raised. For with present information and analogy as a guide, it is more likely than not that 'spontaneity' or 'independence' is only apparent ; and the occasional inability to trace a history or the mid-course of infection, should not entail the idea of renewed creation of a disease so permanent in its characters and so highly communicable as relapsing fever.

Conditions of Spread.—Relapsing fever not extending by means of infection or miasmatic agency, and specially affecting pauper communities, the conditions which regulate its propagation may be summarised as follows :—

1. Presence of the Disease at Matured Stage.—It is to be noted that as contagiousness extends over prolonged and interrupted periods, the whole length of illness of a suspected subject should be known, before it is decided whether or not contagion has been conveyed by his means. In associated bands of individuals, the disease may be kept up for weeks or months with no very obvious or long disqualifying sickness. Comparative experiment has proved that the blood of a patient during a very brief second relapse (illness at 30th day), may be markedly infective. See pp. 11 and 29.

2. Facilities of Contact.—These are pre-eminently numerous and considerable amongst poverty-stricken and vagrant communities. In famine-areas they abound, and constantly in the low quarters of towns; equally amongst pilgrims travelling by land or sea, caravans, coolies on board ship, and convoys of prisoners under transfer.

3. Mal-hygiene.—The noxious influence of foul soil and air, of exposure to inclemencies of weather, and of bad or insufficient food, will be obvious. Possibly one or other of these insanitary influences especially predisposes to relapsing fever, but from observation at Bombay I am unable to say which. The degraded, yet not starved aspect of tramps and vagabonds, who seem to be peculiarly liable to this disease, is suggestive of combined personal influences being here operative. Similar conditions predispose to typhus fever, but this malady disqualifies for wandering, and spreads rather in areas of concentrated individuals.

Epidemics of Relapsing Fever.—The historical connection of Relapsing fever (*morbus pauperum*) with Famine, points to a frequent common limitation of these phenomena as regards date and locality of occurrence, and concurrent severity, which is so remarkable as at first sight to warrant the idea of a strictly casual relationship. No opportunity, however, has latterly occurred permitting a thorough scrutiny of all the conditions and events here concerned; and therefore judgment must still rest on personal conviction. In what manner individual destitution facilitates the advent of infection, has not yet been accurately ascertained; but so far as I perceive, this can only be done by way of predisposing influence, and that some essential factor may be sometimes wanting, is shown by many examples of wide-spread starvation without the hunger-pest.

Former experience was concisely expressed as follows, in the Report of the Health Commissioners of Ireland on the Epidemics of 1846-50 (Parl. Paper, 1852):—‘The potato crop failed considerably in the autumn of 1845, and epidemic fever was anticipated, for experience had shown that scarcity of food in Ireland, if of any considerable duration, had been invariably followed by an epidemic of fever; therefore preparations were made. . . .’ ‘Like all former visitations, this epidemic assumed a contagious character, and the mortality was great amongst the poorer classes.’ It is generally assumed that the ‘fever’ here meant was in great part relapsing fever.

The negative experience at Madras forms a striking contrast, and hereby an illustration of the fundamental non-connection of fever with famine: thus, Mr. W. R. Cornish, in his valuable Report on the Sanitary and Medical Aspects of the Famine of 1876-77 (Madras, 1878), writes of the emaciated wandering peasantry flocking by

thousands into the camps of relief: and Dr. A. Porter (*loc. cit.*, p. 37), who made 450 autopsies, states that 'the majority of the bodies were mere skeletons with legs tucked up on the abdomen, the walls of which almost touched the spine.' And yet 'there was no fever at all resembling the relapsing typhus of the Irish famine.'

In Western India it may with reason be urged that had there been no famine, there would have been no epidemic; and this instance might be quoted as evidence of the essential relationship of the two events. But, strictly, too little is known of the previous state of the country, and of possible communication with other areas where fever prevailed, to permit of a valid conclusion being formed; and similar remarks apply to the few instances of late occurring in Europe (*e.g.* Breslau 1868, and Finland 1867-68), when fever has appeared to be closely associated with unusual degrees of poverty. That famine-conditions of all degrees are highly favourable to the production of an epidemic seems undoubted, but they are not essential.

A second general condition under which epidemics have arisen, is when, in the absence of local want, a temporary surplus of population has occurred; the result being both excessive overcrowding and a comparative scarcity of food. Here etiological influences become more complex, and a varying estimate may be formed as to the effect of the two agencies named. Epidemics thus arising are commonly severe.

Illustrations are afforded by Scotland in 1840, where 'owing to the construction of railways, which, it is said, attracted numbers of Irish labourers and caused the inhabitants of the small villages and towns along the lines, to flock into the large towns and swell their pauper populations, and to other causes, the misery and want of the poor year by year increased;' the result being the severe epidemic of 1843. (Dr. Murchison.)

At St. Petersburg, 1864-5, the beginning of sickness was consentaneous with an unusual influx of labourers who flocked to the city during the winter of 1864 (an excess of 43,000 is named), whence arose excessive overcrowding—*e.g.* as of 60 men in a single room with closed doors and windows, and the air so foul that a candle would not burn in it. Food was scarce and of inferior quality; there were great climatic variations; the water-supply was bad, and much intemperance prevailed: yet the people were cleanly in their persons, taking a bath of steam at least once a week. The troops were affected, and to a slight extent English and German workpeople. The epidemic was very severe, typhus co-existing. (Dr. G. Whitley, Eighth Rep. Med. Off. Priv. Co. 1865). In his description of the fever, Dr. Zuelzer mentions that the 'new disease' came on some months earlier at Odessa. Moscow, also, has been mentioned in this connection; and hence there arises a reasonable doubt if the fever strictly arose *de novo* in St. Petersburg, and had not been introduced prior to, or along with, the great influx of labourers.

In all countries a surplus rural population is of necessity compelled to migrate, and commonly the movement is towards the larger towns. When remunerative labour abounds, the attraction increases; and thus at Bombay, in 1863-4 (see page 20) in consequence of fresh wealth large public works and cotton-manufactories were set afoot, and ample employment promised for all; hence the great influx from outside. At St. Petersburg, also, a special cause was in operation, there as at the Eastern city, it is the custom for many country labourers to repair during winter to the capital in search of employment, and owing to a recent 'ukase' liberating the serfs, a much larger number of them went to St. P. to obtain work: hence the unusual stress. (Dr. J. Millar.) As regards both these remarkable instances (and I might add that of Scotland alluded to above), it seems to me there are grounds for suspecting that the inevitable overcrowding and comparative scarcity of food, were not the sole causes of the epidemic fever which ensued; but that the immigrants brought this disease with them.

Should famine arise from failure of the crops, the necessity for

migration becomes intensified ; and if fever have already appeared, the emigrants will carry it with them to the towns, where it will spread: then the epidemic conditions are further multiplied. This is a summary of experience at Bombay in 1876-7-8, for not only did there occur a large exodus towards the presidency, but a contagious fever was brought with it, which, as I have shown, soon extended to the town residents themselves. Epidemics of this kind will be severer than usual, from the combination of untoward circumstances ; and this seems to have been the case in Great Britain during 1847-8, in connection with immigration from Ireland. A precise parallel to experience in Western India, I have not met with in European records.

Lastly, the origin of an epidemic in towns has usually not been attended by either increased scarcity of food or augmented overcrowding ; and yet the disease, though often mild, has not seldom been of pronounced type. Thus, at Breslau and Berlin, cases of the bilious typhus type were fully recognised ; and this circumstance is noteworthy. In probably all epidemics of this kind, the sickness is solely of imported origin ; and often it can be traced to a single individual, who is a stranger and wanderer coming from an infected province. The distance to which relapsing fever can be conveyed by one or more affected persons, and the number of possible *foci* of fresh disease established by them in their journeyings, is matter of mere calculation. I have mentioned illustrations at p. 9 *et seq.*, and others will be found in the records of many epidemics, *e.g.* at London, 1868-69, Glasgow (Dr. Tennent), Edinburgh (Dr. C. Muirhead), Liverpool (Dr. J. de Zouche) during 1870 ; and in 1873, at Bradford (Dr. A. Rabagliati). On the Continent of Europe there have occurred numerous similar instances of town-epidemics totally unconnected with famine or unusual overcrowding, and always traceable to incidentally imported disease. Records I have looked at date from Prague 1867-68 (Drs. Pribram and Robitschek) and Griefswald 1868-73 (Dr. Haenisch), down to Giessen, Magdeburg and Heidelberg 1879-80 (Drs. Gegensohn, Aufrecht and Friedreich) ; and in all, whilst a varying importance is attributed to collateral influences, there is a striking consensus of testimony as to the purely extrinsic origin of the fever.

I note the frequency of such casual epidemics in capital towns, and in maritime countries at the seaports. According to Dr. Bryden, India has furnished many examples in her central jails, whither bands of infected prisoners under customary transfer have brought the fever.

As obviously, sporadic cases of relapsing fever do not always give rise to epidemics, there must be favouring conditions well worthy of consideration. It is also desirable to ascertain the reason why typhus does or does not attend epidemics of this sort ; experience varying—thus at Breslau in 1872-3, with 32 deaths in 448 hospital admissions (death-rate 7.2 p. c.), there was no typhus either with or after the epidemic ; whilst in 1879, with only 15 deaths in 325 admissions (death-rate 4.6 p. c.), there was much typhus and also many cases of cerebro-spinal meningitis. On both these occasions, the beginning of relapsing fever was traced to a single affected individual and that a stranger.

The following additional remarks on the beginning, duration, severity and variations of recent epidemics will serve to illustrate the late experience at Bombay.

Commencement.—There is every reason to suppose the 'new' fever began gradually, the earliest recognised cases being sporadic : see page 25 and col. D. of the Chart, p. 30. At the G. T. Hospital fever admissions rose considerably at the close of 1876, some weeks before public attention became aroused ; the conditions here were, however, highly complicated, and I therefore quote some simpler data clearly indicating how the fever now begins :—

The severe and persistent epidemic at St. Petersburg made its appearance in the summer of 1864, and at first sporadically; its increase being gradual. (Dr. Herrmann.) The mild outbreak in London, 1868, also began by scattered cases, the first of all in a woman of Irish birth, resident and not very destitute : the disease was supposed to be imported from Germany; it could not be said to have become epidemic until after at least a year had elapsed. (Dr. Murchison). At Prague, a case was seen after the middle of 1865, and two severe cases in the spring of 1867, which were probably the first publicly noticed in Germany and mid-Europe. The mild outbreak in Philadelphia, 1869-70, commenced by isolated cases (the earliest being those of women, not impoverished and not all neighbours) ; those sleeping with the sick became infected. (Dr. Parry.)

At Griefswald, 1868, the first case was that of a sweep tramping from E. Pomerania, and 4½ months intervened before the next case was seen ; thence an unbroken series for 12 months, all being regarded as importations, excepting the instances of two ward-boys. At the end of 1871, there being no instance known in the meantime, a journeyman saddler introduced fever again, and a second epidemic arose.

At Breslau, 1872-73, the disease was introduced by a woman without employment and of no fixed night-lodging, who was ill on arrival in the town, and who infected another woman in the same asylum ; for a month no other cases there. Then two other wanderers not in apparent connection with the first : the spread was very gradual, and it was effected solely through contagion amongst the inmates of single tenements. (Dr. Litten, in full detail.) Again at Breslau, 1879, the first case was that of a tramp, who could not give a definite history of his illness. The same localities in the town were most implicated as in earlier epidemics. (Dr. Spitz.) At one of the farthest points westward to which the disease has yet extended, viz. Heidelberg, in S. Germany, it is stated the first case seen (Dec. 1879) was that of a strolling musician, in whose party fever prevailed. (Dr. N. Friedreich.)

Duration.—Late epidemics in England and Germany have lasted from a few months to 1 or 2 years, seldom so long as 3 years ; the periods varying not always in proportion to the population of the towns affected. The essential conditions of varying duration have yet to be elicited. A remarkable exception is the instance of St. Petersburg, where for 17 years relapsing fever has persisted, and still flourishes : some other towns in Russia also present the same experience, *e.g.* in Odessa and easterly-situated Kasan, where this fever has been known for 15 years. When of brief duration, outbreaks have, moreover, not seldom recurred ; and on the Continent, at almost regular intervals of a few years since 1868. It would appear that the persistence of the disease in Russia and the adjoining western provinces tends to maintain a constant flow of infection westward : not dissimilar to the several importations into Britain from Ireland, in former years. Epidemics come to an end through either a gradual exhaustion of the contagion, or a failure of subjects susceptible to infection : of the first not much evidence appears, but regarding the last, it seems possible that constant contact with the infection may (as in ague and typhoid fever) confer a kind of insusceptibility.

The Course of epidemics has been commonly that of gradual in-

crease, with a brief and sustained acme, followed by a gradual decline ; both onset and end may be tolerably abrupt, but no rule seems to have obtained. From first to last, town-epidemics are constructed (so to speak) of minor successive extensions from house to house, or quarter to quarter ; fresh *foci*, sometimes apparently disconnected, arising at intervals. According to the pauper population, is the prevalence attained ; but curious limitations are noted, and the circumstances of persistence or decline have yet to be compared. I have already stated how the disease was kept up at Bombay (page 18).

Its comparative severity has differed considerably, the death-rate varying from 1 per cent. (or less) to 9 or 10 per cent. (or more) : and this not always with evident reason except that of varying intensity of infection, which is a subject worthy of special notice. Whether such variation attends corresponding difference in the visible amount of spirillar blood-contamination, is not yet known. By general assent, epidemics differ in fatality according to the frequency of the severe bilious type of fever ; for as this becomes more frequent, so is the outbreak severe. *Typhus biliosus* is not seen in some mild epidemics, but it was early evident in the severe outbreaks at St. Petersburg and at Bombay. On the other hand, it first supervened only at the end of 15 years, at Kasan.

A difference has also been noted as regards virulence, or infectivity of the disease ; both during independent epidemics, and in those recurring in the same locality. When thus recurring, the later outbreaks are apt to reappear in the same quarter and streets of the town as previously ; the successive epidemics may or may not increase in intensity.

In sum, the present gradual spread by contagion of relapsing fever across mid-Europe, illustrates, as if by experiment, the natural progress of this disease as an epidemic ; and former outbreaks in Britain, which differed only in their greater severity, must have proceeded in similar fashion.

It may be said that epidemics of relapsing fever by a natural classification fall into two chief groups, as follows :—

1. Those arising under local famine-conditions.

Examples : Ireland 1817-19, and 1846-7 ; Silesia 1847 ; and the Bombay Presidency 1876-77.¹ Disease—severe.

2. Those due to imported disease, which, originally of small amount, increases by propagation amongst the resident pauper population.

Examples.—The majority of epidemics in Great Britain, and nearly the whole of those occurring on the continent of Europe since 1863 ; also in N. America, Réunion, Mauritius. Disease—mild or severe, according to varying local circumstances.

¹ Doubtless other parts of India might be named, and on either side Persia and China. In the late Dr. J. L. Bryden's Report (*l. c.*), great epidemic dates for N. India are given as 1816-20, 1836-38 and 1859-65 ; besides another. Were the identifications of relapsing fever quite satisfactory, these instances might be quoted as large illustrations of the origin and course of epidemics of that disease ; but at present only a few better proved cases are known, and sometimes where demonstration might have been anticipated none is available. Thus, a recent instance of relapsing and contagious fever in the jail at Umballa, gave negative microscopic results ('Rep. San. Commiss. with the Gov. of India,' 1879). The early experience of Dr. Obermeier at Berlin, is a part of his valuable discovery which is intuitively recalled to mind on occasions like this. *Vide* p. 333.

The city of Bombay offered the peculiarity of an epidemic consisting in great part of disease actually transferred by immigrants, as well as of a component derived from town-residents placed in contact with the new-comers.

The data thus summarised, indicate the prevalent etiology of epidemics ; and a consideration of the history of relapsing fever in those countries where it is oftenest said to arise *de novo*, induces me to suppose that the disease may there be constantly present. The actual endemicity of relapsing fever is, in my opinion, strongly indicated by present experience in eastern and mid-Europe, where the disease has only lately been known. Thus, it is now apparent that if not present at one spot, it may be present in another ; and hence the so-called epidemics of large towns are only temporary and recurring exacerbations of a widely diffused malady. On this interpretation, the seeming latency or absence of relapsing fever which has been regarded as an argument for the occasional independent origin of the disease, becomes readily explicable ; such absence being not real, but relative to certain localities only. The frequency of recurring epidemics in certain towns (*e.g.* Breslau, Berlin) maybe due to facilities of communication, or to comparative proximity to *foci* of the disease. In some Russian towns where relapsing fever seems already to be practically endemic, it exacerbates at intervals just as in other localities, where evidence of endemicity is not recognisable ; and experience at Bombay has shown such movements to arise from fresh accretion of susceptible subjects.

Explanation of the concentration and tenacity of the disease in certain areas (*e.g.* Ireland, Russia, India, and Egypt: *v.* page 29), might, I think, be found in the social conditions and relations of their pauper population ; but this topic has yet to be studied in detail.

The first of these is the fact that the British Empire is a vast and diverse entity, covering a large area of the world. It is not a single country, but a collection of many different countries and territories, each with its own history, culture, and people.

The second fact is that the British Empire has been a major force in world history for over a century. It has played a significant role in the development of the world, both in terms of its economy and its culture. It has been a major power in the world, and its influence has been felt in many different parts of the world.

The third fact is that the British Empire has been a major force in the development of the world. It has played a significant role in the development of the world, both in terms of its economy and its culture. It has been a major power in the world, and its influence has been felt in many different parts of the world.

The fourth fact is that the British Empire has been a major force in the development of the world. It has played a significant role in the development of the world, both in terms of its economy and its culture. It has been a major power in the world, and its influence has been felt in many different parts of the world.

The fifth fact is that the British Empire has been a major force in the development of the world. It has played a significant role in the development of the world, both in terms of its economy and its culture. It has been a major power in the world, and its influence has been felt in many different parts of the world.

The sixth fact is that the British Empire has been a major force in the development of the world. It has played a significant role in the development of the world, both in terms of its economy and its culture. It has been a major power in the world, and its influence has been felt in many different parts of the world.

The seventh fact is that the British Empire has been a major force in the development of the world. It has played a significant role in the development of the world, both in terms of its economy and its culture. It has been a major power in the world, and its influence has been felt in many different parts of the world.

The eighth fact is that the British Empire has been a major force in the development of the world. It has played a significant role in the development of the world, both in terms of its economy and its culture. It has been a major power in the world, and its influence has been felt in many different parts of the world.

The ninth fact is that the British Empire has been a major force in the development of the world. It has played a significant role in the development of the world, both in terms of its economy and its culture. It has been a major power in the world, and its influence has been felt in many different parts of the world.

The tenth fact is that the British Empire has been a major force in the development of the world. It has played a significant role in the development of the world, both in terms of its economy and its culture. It has been a major power in the world, and its influence has been felt in many different parts of the world.

APPENDIX A.

NO. I.—ARTIFICIAL PRODUCTION OF SPIRILLUM FEVER IN THE MONKEY.

ATTEMPTS to reproduce this infection in pigeons, rabbits and dogs have failed, but in an animal approaching nearer to man they readily succeed; and for convenience sake, my experiments were limited to the common small monkey of W. India, known as the *Macacus radiatus*. The specimens brought to Bombay from the neighbouring hills, had an average weight of 3 lbs. avoird., were seemingly young and in vigorous health; those employed were kept apart in a large room and regularly fed. The material used for inoculation was the blood of fever-patients, either entire or defibrinated, 3 to 20 minims of which were injected with the ordinary hypodermic syringe, beneath the skin at the inner side of the thigh. The axillary temperature of the animals was then taken in the usual manner, every 2 or 3 hours day and night, and at the same time one or more preparations of the blood were made: whenever practicable the fresh blood was also scrutinised, and the daily records were carefully preserved.

Experiments were begun in February 1879, and continued for several months: a detailed account of them is published in the 'Transactions of the Royal Medical and Chirurgical Society of London,' vol. lxiii. 1880, and the following is a summary of the original records. Similar observations confirming the fact of the communicability of spirillar infection to the *Quadrumanus*, were early repeated in Germany by Dr. R. Koch (then of Wollstein), as intimated in the 'Deutsche Medicinische Wochenschrift,' No. 25, dated June 21, 1879. Berlin.

Enumeration of Data.—The total number of essays was 46, of which 36 were made with fresh blood, 7 with dried blood, and 3 with human saliva. The first series of 19 inoculations with fresh spirillar blood, includes all the instances in which specific infection and fever ensued: of these 14 were made with human blood, viz. 4 at invasion-attack of relapsing fever, 8 at first relapse, 1 at second relapse and 1 at first apyretic interval (ninth day): 5 were made with blood of the monkey, viz. 3 at febrile stage and 2 at incubation-stage of fever. The second series consists of 8 experiments, none of which were followed by fever; they include 5 inoculations of human blood, viz. 1 at decline of invasion and 4 at first relapse; and 3 inoculations of the monkey's blood, viz. 2 at incubation-period and 1 at incipient pyrexia. All the 9 essays made with non-spirillar blood failed to produce infection, the material used being taken at incubation-period, crisis and secondary fever of man or animal, and also during the first apyretic interval of man. All the 7 inoculations with desiccated blood (6 taken during fever and 1 at crisis) also failed: this material had been kept for intervals varying from 5 days to 8 months.

The 3 injections of fluids from the mouth (which contained the spirillum of saliva along with other bacteria and organic particles) did not produce spirillar infection, but rather a kind of septicæmia, which proved fatal when the saliva of patients ill with fever was used. In some of the experiments more than one animal was operated on at a time.

Commentary.—Of inoculations made with fresh blood containing spirilla there were 22 of human material with 6 failures, and 11 of monkey's blood with 3 failures: total 33 with 9 failures, leaving near 73 per cent. of successful results, or a proportion large enough to prove that the spirillum disease is readily communicated from man to the lower animal, and from one animal to another. My hospital experience had already shown that it may be easily communicated from man to man during life, and from the recently dead subject to the living.

Negative results. At present I am not able to assign a reason why some experiments failed; but assuming no faulty manipulation or unseen accident, it would appear that the following conditions are unfavourable to success—a period prior to onset of fever, or the very beginning of pyrexia, at the acme of attack with high temperatures or at the very end; and if there be present some other blood-contamination, as pyæmia or septicæmia. From 7 experiments with blood taken at incubation-period, it seems that infection may or may not occur at this non-febrile stage; and this contingency is explicable from the spirillum being always rare, and therefore either very scanty or absent in the material inoculated.

After injection of non-spirillar blood there was no apparent disturbance of the system, and this seems to show that in the absence of the parasite, infection does not occur in the lower animal; yet as regards man, there are good grounds for believing that it may sometimes take place immediately after febrile crisis (*vide* Chapter on Contagion, p. 403).

The negative results of inoculation with dried spirillar blood, are noteworthy with reference to the supposed conveyance of relapsing fever by clothing or other *fomes*; and as regards the contrast here with the long persistence of virulence in the blood of splenic fever.¹

I should mention that I frequently met with appearances of the blood in these non-productive essays, which seemed to indicate a partial or abortive development of the spirillum; but not knowing how to interpret them satisfactorily, had to record a negative decision, the impression still remaining that a very slight or transient blood-contamination may have taken place.

Lastly, I remark that this occasional absence of manifest infection after inoculation of specific material, quite corresponds with ordinary clinical experience, according to which only a certain proportion of subjects exposed to the contagion of relapsing fever do really acquire characteristic disease.

Positive results. Pyrexial phenomena:—The normal mean temperature of the monkey is 101·35 F. or about 3° above that of man; the daily cycle is very nearly the same in range and course, and it was not less indicated during continuous high fever: allowance had sometimes to be made for incidental variations of temperature these small animals were necessarily subject to. General features: the febrile attack in the monkey was almost always a single one, or without relapse; an apparently spontaneous recurrence of fever being noted only twice (or at most 1 in 8 illnesses): it dis-

¹ Some human blood proved to be infective in the fresh state, was dried over sulphuric acid and bottled for 10 days; 2 grains of it were then rubbed up in 10 drops of distilled water, and of this coffee-like mixture 2 drops were injected beneath the skin of the arm of two young negroes, who probably had had spirillum fever a month before: a monkey was also inoculated in the same way. Results negative. This was the only experiment practised on the human subject, at Bombay.

played much more variety of form and duration than the corresponding primary attack in man, being comparable in this respect rather with the 'relapses' so-called in him, which have been shown to be highly diverse in character. The patients furnishing blood for inoculation, displayed relapsing fever of the ordinary kind; yet in the monkey it was not possible to foretell accurately the severity of illness, and it did not seem to me that this unanticipated variability was referable to differences in the amount of spirillar blood injected. A mild or severe attack in the human subject, does not necessarily entail the same in the lower animal; but as inoculation from the sick monkey always produced a severe infection, it appears that the human virus becomes intensified in its passage through the monkey; and a somewhat parallel augmentation of infective properties was perceptible in inoculations from man at succeeding pyrexial stages; thus infection at invasion-attack resulted in the mildest kind of fever, whilst at first relapse it induced marked illness, and the seemingly little contaminated blood of a transitory second relapse was also highly effective: this is a subject well worthy of closer investigation than I could accord to it.

Incubation-period. My experiments showed conclusively, that immediately prior to the onset of fever there always occurs a more or less prolonged period of visible blood-contamination; and hence that the interval between infection and fever is divisible into two parts, viz. an earlier and usually longer non-spirillar stage, and a final stage of spirillar manifestation, during which the body-heat so far from being augmented is often rather depressed. The duration of these quasi-normal periods, varied from 10 to 50 hours or more, and I was unable to account for the difference in particular instances: in 13 selected examples the mean duration of the entire incubation-period was about 90 hours, its range from 30 to 126 hours, and the average rather over the mean, or nearer 4 than 3 days: generally it was longest in the mildest febrile attacks, and shorter when severe illness followed. Though still inexplicable, these results accord with the ordinary clinical experience of relapsing fever.

Specific pyrexia. The onset of fever was as abrupt as in the naturally acquired attack; its duration was briefer than that of the corresponding first event in man, never exceeding 86 hours, in the mean only 43 hours and at the shortest only 6 hours; this wide range of variation also being a character pertaining to the 'relapses' of the human prototype, and not to the invasion-attack: and hence the lower animal may be said to exhibit at this stage all the known degrees of spirillum fever. Proportionately to its bulk, it suffers most; and so, I may add, does the human infant.

The form of fever presented is, as in man, paroxysmal at briefer attacks, remittent or continued in the severe: multiple daily exacerbations and remissions were seldom indicated, and the normal daily cycle was never effaced during even high fever. The maximum intensity of pyrexia was about equal to that of man, although the normal starting-point is so much more elevated.

Termination by crisis always happened, profuse sweating and sub-normal temperatures not, however, being noted. Nor were acmal and epicritical phenomena remarked in the monkey. All the severe attacks were succeeded by a sharp rebound or secondary fever, during which 3 animals of 16 died, and a fourth would probably have died at this time.

No fixed relationship was observed between incubation-periods and ensuing fever. Neither immunity from, nor predisposition to, a second infection, seemed to follow the first attack; the symptoms in monkeys again inoculated two or three weeks after a prior experiment, being in nowise peculiar.

Secondary fever. This striking reactive or residual phenomenon assumes, as in man, the form of either a brief or a severe attack; and now

the blood is equally free from visible spirillar contamination. Rebounds set in quickly and abruptly, present all the recognised forms of pyrexia, and may be even more intense than the preceding specific illness. The circumstance of secondary fever being as prominent in the *feræ* as in man, seems to show that this event belongs essentially to the specific infection; in monkeys ordinary local inflammations, such as pneumonia, were not seen as causes of sequelar pyrexia.

Relapses. The only two known instances occurred at 6 and 12 days after the primary attack, and both were mild: they were preceded by a stage of non-febrile spirillar infection, and were clearly repetitions of the specific fever. Their rarity and highly irregular date of onset, gave rise to the notion that the relapses might really have been instances of fresh infection by contagion from other sick animals: yet were it proved that the comparative febrile attack is always a single one, this fact would be no valid argument against its identity with the so-called 'relapsing fever' of man, because at Bombay as many as one-fourth of all human illnesses were apparently not followed by a recurrence, and still they presented no divergencies from the common type of primary attacks.

The aspect of the sick monkeys was indicative of oppression rather than of prostration, the animals sitting up with folded arms and head so drooping that there were visible only the radiating hairs of the scalp (whence the name of the species); the visage was pallid and shrunken, eyes dull and mostly closed, the cutaneous muscle was sometimes tremulous, appetite failed, even fresh fruit or water being hardly touched; a low moaning was common and on hearing this, even at a distance, I could tell that an experiment had succeeded: with the crisis relief was prompt, the appetite might become ravenous and convalescence quickly ensued. Renewed shiverings and depression ushered in secondary fever; rapid breathing, prostration, semi-consciousness and lowering of temperature followed in the casualties.

The Post-mortem Appearances in Spirillum Fever. Twelve autopsies were made of nine animals killed and three dying, at various dates after infection. During the incubation-period (three instances) commonly no striking change was noticed; but once deep congestion of the mucous membrane of the stomach, with minute petechiæ, about the middle of the viscus, and liver and spleen congested. In five deaths during fever, vascularity of the stomach about the middle, with small petechiæ; and vascularity of the duodenum and lower end of ileum, with congestion of liver and spleen; in one animal bled to death the liver was almost translucent, and the spleen small, flabby and pale, the impression hence arising that the solid parts were but little altered. Twice, however, there was some pulmonary apoplexy; the brain, heart and kidney were never notably changed in aspect. In one death at critical fall, the viscera showed nothing peculiar. In the three animals dying during secondary fever, the liver was congested and once enlarged, the spleen large and congested, kidneys healthy looking, mucous membrane of stomach once unchanged, and twice inflamed about the middle; in two dying on the third day of rebound, the small intestines were inflamed throughout, beginning abruptly at the pylorus and ending at the ileo-cæcal valve, there being some hæmorrhagic spots also: and in one dying a little later, the same intestinal mucous membrane was very vascular, the walls of the gut generally being very thin, and the large intestine showing only some vascularity in the rectum: petechiæ were seen twice on the lungs and on the heart in these three cases; the brain was pallid only. At no time was the presence indicated of changes commonly found in human autopsies—viz. cerebral meningitis, pneumonia, marked parenchymatous inflammation, fatty degeneration or infarcts of liver, spleen and kidney; and hence these results would rank as contingencies belonging to the subject himself, rather

than to the infection. Minute examination of the tissues has, however, still to be made.

After death from poisoning by saliva (two cases), there were noted inflammation of the stomach and extravasation of blood into the arachnoidal sac.

Lastly, no local irritation followed the injections; and on careful examination of the tissues at site of injection in animals that were killed, there were no morbid changes to be seen.

In PLATE VIII. are shown the three-hour temperature-charts of mild and severe attacks of spirillum fever artificially induced; and also the same charts reduced to the ordinary clinical bi-daily form, for purpose of comparison with the human data. The state of the blood is described in Sect. III. Ch. I. p. 333.

NO. II.—SOME CULTURE-EXPERIMENTS.

Although under similar artificial conditions the *Spirochæte Obermeieri* does not grow nearly so freely as some *bacilli* (e.g. of anthrox), yet it presents certain appearances which are not without interest. Thus, in April 1879, Dr. Rob. Koch (then of Wollstein) informed me that when cultivated the *spirochæte* become 'transformed into long, undulating and densely inter-lacing filaments'; and upon trial at Bombay, I met with the results briefly described below.

Small quantities of liquids were enclosed in shallow cells, and exposed to a continuous moist heat of 95° – 110° F. (35° – 43° C.); microscopic examinations were made at short intervals, and with the higher powers. The medium commonly used was *humor aqueus* of man, monkey, sheep, or oftenest the dog: to a drop of this being added a minute speck of the blood or serum to be tested. Frequently the blood-serum alone was cultivated. Materials taken from man and monkey served equally well.

Of the essays with material known to contain the spirillum, all 7 made with entire blood taken at febrile stages of disease or at specific incubation-period, gave results invariably negative as regards the spirillum; and therefore it appeared that entire blood is unsuitable for experiment. I attributed this unsuitability to the presence of the red blood-discs, which themselves undergo striking changes afterwards alluded to.

Nine essays made with non-spirillar blood-materials taken at critical defervescence, and at date of expected relapse. No signs of a fresh development of the organism were to be seen.

The remaining series of 32 experiments made with infected serum, either alone or after dilution with aqueous humour, commonly furnished positive results. The changes noted in the spirillum were: 1. General enlargement. 2. Extension and the formation of open networks. 3. Dense aggregation and blending. All these changes may coexist in the same specimen, but one will predominate; they supervene in a few hours, and are earliest apparent at the margins of the cell. Warmth favours them, and a temperature above fever-heat (e.g. 110° F. or 43° C.) does not prevent them. As to the influence of air—in collections of serum kept open at 76° F. for several days, the spirilla may retain their activity for 3 to 5 days, yet their numbers do not increase and the clusters formed are small; if then enclosed in cells and warmed, the specimens sometimes showed signs of growth, but it seemed that exposure to air did not favour the tendency to change.

I noted that whilst dense aggregation of the filaments was preceded by their active clustering, general enlargement and ramification were preceded by a quiescent state of the filaments. Such quiescence, therefore, does not indicate death of the spirillum; and I have occasionally seen in the fresh

blood of patients suffering severely, a sluggish, enlarged and adhering aspect of the organisms, like to that presented at early stage of cultivation. Dense aggregation of the spirilla is also to be noted in some cases of severe illness; and if this likewise be a preliminary to growth, it follows that some of the fatal and more serious results of spirillar infection are referable to premature growth of the parasite in the blood, increased liability to obstructions of the circulation thence resulting.

Changes subsequent to the above-mentioned, are varied: often the filaments soon become dotted with bright or opalescent spots, and clusters of bright granules may form at their free ends. Multiplication by segmentation, and by growth from bright granules, either attached or free, may be indicated. Some of the leucocytes in the serum seemed to give rise to new spiral filaments (Fig. H. Plate I.); and occasionally there later appeared the intervention of vesicles, and granular masses.¹ In specimens kept for one or two months under different temperature-conditions, the spirillar masses and networks may persist nearly unchanged; and I have sometimes observed at about weekly intervals the signs of renewed growth, as if a fresh outgrowth of filaments was produced: such intervals corresponding to those of relapses in the living subject. The reproduction of free and active spirilla was not witnessed; but I saw in free granular matter, quiescent fresh filaments at various stages of growth. Fig. I. Plate I.

Selected illustrative experiments are the following:—

1. *Hypertrophy of the Spirillum*.—In some degree this change is invariable; perhaps the most striking instance occurred when additional nutriment was supplied. Thus, serum taken at 3rd day, 1st relapse of man, was mixed with healthy serum in the proportions of $\frac{1}{4}$ and $\frac{3}{4}$; specimens warmed. In a few minutes the spirilla had drifted into proximity at the margin of the cell, becoming quiescent and enlarged: in less than an hour, their number was greatly augmented and all were elongated, thickened, rigid in aspect, partially unfolded, and more or less bent. Generally translucent, they presented midway or at one end, one or more knots; and here some were dividing or branching, the smaller segment before becoming free being connected by a short band too delicate to be visible with the $\frac{1}{16}$ -in. immersion-lens. Free granules few; some short, bent, dotted filaments free and comparable to incipient spirilla, but not actively moving. Subsequent changes watched for 10 days, not considerable; but medium then clouded and the parts too pallid to be distinct. In one specimen the spirilla were at last found together, and imbedded in a colourless membrane; some of them having attained an enormous size. Plate I. fig. G.

2. *Elongation of the spiral filaments and apparent ramification*, both vertical and lateral; the result being a complex network formed of primary close meshes, and secondary open meshes. This was the commonest result of culture.

Example.—A drachm of blood was drawn from an infected monkey, at stage of incipient fever; it contained many active organisms, and in one hour had set. The serum first exuding displayed some extended, rotating spirilla; but so few, that I thought the experiment would fail. A minute quantity (perhaps the $\frac{1}{12}$ of a drop) was added to fresh aqueous humour of the dog; and at 9 P.M. placed in the warm chamber at 105° F. (40°·5 C.). Two hours later, the spirilla appeared very numerous and quiescent, none being active and isolated as before; but slender networks were being formed by interlacing of the filaments, sometimes the aspect was that of radiation from a common centre. Very long and delicate fibrils were seen crossing between the meshes, or passing over the whole field, gently waving, became lost to vision from their extreme fineness. The spiral contour was everywhere preserved. Some shorter filaments were noticed, either new or detached from the networks (?). After 16 hours, the structures had not extended; but now were dotted, in parts, with bright granules of varying size and seated in the substance of the filaments: once a small vesicle was seen arising from the tip of a terminal branch; the field remained clear. Another specimen kept at 84° F. (29° C.) showed

¹ Once there appeared in connection with the spirillar network, a tubular and vesicular growth; and at other times large spiral fibres more or less resembling ordinary spirilla, but quiescent. As some doubt existed as to their direct origin, these bodies are not named in the text.

after 6 hours the spirilla in different conditions, some being isolated and moving languidly, others quiescent and enlarged, and some forming small open clusters; growth being much less prompt and luxuriant than in the warmed preparation. For illustrations see L, Plate I.

3, *Dense aggregation and blending of the Spirilla into granular masses.* This aspect is common in preparations not exposed to warmth: the compact congeries vary much in size, are sub-angular or ovoid in shape, and finally assume the form of thin floating discs, two or more of which are usually connected by means of outlying filaments: the contour at first undefined, then becomes more distinct. A tendency to such aggregation is noticeable in the denser angles of spirillar networks, and transitions are seen between meshing and blending in most specimens under culture.

Example.—The blood of a woman on 3rd day of 1st Relapse, furnished in 7 hours a little turbid serum containing many spirilla: in 20 hours a warmed specimen of this serum showed luxuriant and delicate networks and small granular masses, all quiescent. A cool specimen (t. 80° F., 27°·4 C.) displayed also an apparent increase of the organisms, many being isolated, others in small clusters, and most collected into dense aggregations: these when large are formed partly of red blood-discs swathed in spirilla, and are visible to the eye as brown specks: all the freer filaments still move, the isolated sluggishly and sideways; and when aggregated the outliers move actively, especially when in contact with the red discs, which are then drawn in and added to the masses already formed. Movement early ceases in the centre of these collections, where the spirilla completely blend into a finely granular substance soon presenting somewhat the aspect of a micrococcus-colony, but without the same uniformity of structure. Some movements persisted on the 3rd day, with enlargement of the isolated spirilla: on the 4th, the granular masses becoming detached, floated upwards; 5th day, they became segmented and began to deliquesce, the outlying spiral filaments sometimes remaining distinct and being dotted with bright specks. Sixth day—the masses had become almost diffuent, and in their place appeared many active granules, as well as fresh spiral meshes, thick and clear: some smaller granular masses, seemingly detached, have also spirilla attached to them: the indication being that of renewed growth and development on this date, which would correspond to a relapse of fever. On the 8th day the medium at the circumference had become clouded with granules, and clear vesicles appeared in connection with the new growths: in the centre of the cell, the fluid was clear: subsequent changes resembled those of decay. Fig. K, Plate I.

Memorandum on the cultivation of entire Blood.—In 11 experiments made by adding a speck of blood to a drop of aqueous humour, and exposing the cell to a heat of about 100° F., there was seen a great production of free granules and, generally, the formation of a ramifying structure starting from the blood-discs. Thus, in less than an hour the discs pullulate clear vesicles, or part with their hæmoglobin; in 2 hours coarse granules of deep orange hue are found at the clot, and at a distance smaller granules arranged in meshes and becoming gradually more slender and ending in a very slender tremulous network. In 18-20 hours pale vesicles may appear, or dark granular collections: sometimes from the bottom of the cell sproutings arise which reach and spread out under the cover-glass. Detached fibrillar networks may be noted, not unlike those of the spirillum, but without a spiral twist. On the 3rd day these changes may still extend, or the whole remain unaltered. Some variations were noted, but all occur in non-spirillar and even normal blood. There is evidently a great increase of the hæmoglobin and the networks are not due solely to fibrillation. Fig. Y, Plate II.

APPENDIX B.

CONCURRENT FEVERS AT BOMBAY.

THESE are Remittent fever and Typhoid. During the late epidemic, however, the Typhus class was not unrepresented; and this is an interesting fact, which serves to connect the late local experience with that of some European cities.

1. **Cerebro-spinal Meningitis.**—The following case with characteristic lesion, was admitted into the larger Native hospital in July 1878, whilst spirillum fever was still prevalent, though declining.

M., 35, Hindoo resident, labourer, living in an infected locality; somewhat emaciated, feverish (t. 102°, p. 120), and only semi-conscious; there is tenderness on pressure over the back and loins; chest sounds normal, abdomen relaxed, hepatic and splenic tenderness with slight splenic enlargement; no jaundice; tongue coated and moist, state of the bowels unknown, bladder not distended. No eruption was seen, nor were spasms observed; history not available; no spirillum in the blood. On third day the temperature declined 3°, without sweats, dry cough and incipient consolidation of left pulmonic base going before (t. 99°·8, p. 128), and the patient became slightly conscious: immediately afterwards, however, the pyrexia increased (t. 101°–102°), insensibility returned and the man died on the fifth day of admission, never having displayed active delirium or convulsions, or other signs inviting special attention to his case: the treatment consisted of ice-bags, enemata and stimulants. The blood had been again examined with negative results, and whatever the nature of the fever, death seemed immediately due to pneumonia. Post-mortem examination 12 hours afterwards:—rigor mortis present, blood dark and semi-coagulated: brain—great congestion of the meninges, no hæmorrhagic effusion; in the arachnoidal sac some turbid serum, and over the convexity of both hemispheres a thin layer of thick, greenish-yellow pus, which was located between the pia-mater and arachnoid, and, following the vessels, had accumulated in the fissures and sulci of the cerebrum; at base of the brain this matter was especially abundant in the inter-peduncular spaces, there enveloping the cranial nerves; it had also passed round the middle cerebellar peduncles, and collecting above the valve of Vieussens had encroached on the upper surface of the central cerebellar lobe. The ventricles of the brain contained much turbid serum; the brain-substances softened and wet, but little congested. Spinal cord:—hardly changed in the cervical region, but about six inches from the *foramen magnum* the sub-arachnoid space began to be occupied with pus resembling that found on the encephalon; this effusion could be traced nearly as far as the lumbar enlargement, was limited almost wholly to the posterior surface of the cord, being not quite uniformly disposed here, and it appeared to be continuous by streaks alongside the vessels of the pia-mater, with the cerebral collection above: the spinal membranes were congested, but neither arachnoid nor pia-mater much thickened, substance of the cord somewhat softened and pale. Chest:—left lung consolidated, especially its lower lobe, right lung healthy: clots in the heart, substance and valves healthy looking. Abdomen:—liver of normal tint, weight 2 lbs. 6 ozs., disseminated throughout the organ were some small pale patches (fatty), bile in the gall-bladder: spleen wrinkled, normal in aspect, weight 8 ozs.; both kidneys somewhat congested, capsules and substance seemingly healthy: intestinal mucous membrane unchanged. The brief temperature-chart is No. 35, Plate VII.

The notes are before me of three surviving instances of this affection; all were lads,

mean age 15 ; the form of fever was remittent and not in itself peculiar. Two of the cases, at least, showed the common features of a semi-continuous pyrexia (max. t. 105°) ending with pronounced intermittent paroxysms, and one displayed a relapse ; in both the pulse was rapid. In the other case the pulse was very slow (65-80 per minute) rising to 90 with convalescence, and the temperature was lower ($104^{\circ}2$) though similarly prolonged, declining and once exacerbating. In all opisthotonos and other characteristic symptoms were marked ; pink spots, petechiæ, herpes, were noted twice ; once albumen in the urine, and once excess of phosphates. These lads were immigrants or residents, and belonged to the same class as that furnishing instances of relapsing fever : all were admitted within the epidemic epoch.

I am not aware that this affection had been previously recognised in Bombay. It was, with typhus, an early attendant on the epidemic at St. Petersburg, 1864-5 ; and also at Breslau in 1879, again with ordinary typhus.

Meningitis of the brain, with its consequence of local paralysis, was more or less clearly indicated in several cases of so-called 'remittent' fever. Two of these were fatal, and in one autopsy there was intense vascularity of the cerebral vessels and sinuses, with an universally diffused opaque deposit under the arachnoid (spinal cord not examined) : pneumonia and pulmonary apoplexy, with thrombi on the r. side of the heart ; jaundice ; liver and spleen much enlarged. Two other autopsies of patients admitted in a moribund state, without previous history, displayed also diffused puriform inflammation of the pia-mater of the brain : there was pneumonia beginning or confirmed, and similar liver and spleen implication : the cord remaining unseen, it is not possible to state if it were implicated with the brain ; but all these instances probably belonged to one series, since suppurative meningitis is not known to occur in ordinary malarious fevers.

2. Typhus Exanthematicus.—This disease has not hitherto been recognised at Bombay, and its occurrence of late still remains uncertain. Had typhus been at all frequent either during or after the epidemic of relapsing fever, its presence could hardly have been overlooked ; but I met with no single typical instance, and no evidence of a comparable fever spreading in a family or house to the exclusion of other febrile disease.

On the other hand the conditions needful for the production of typhus certainly prevailed in the town, and the disease might readily have been introduced from N. India, where its presence seems to be assured. Besides, there were seen many serious cases of 'low' fever with cardiac debility and sometimes distinctly ineffaceable spots, which were not attended by local lesion and did not yield to quinine. If these were remittents, their type was peculiarly typhus-like ; and in the absence of a pathognomonic sign of typhus (which the eruption at Bombay did not constitute), diagnosis must be somewhat doubtful. The co-existence of jaundice does not, in the East, exclude typhus (Dr. Griesinger).

Another condition most of all perplexing, was the occasional likeness to typhus of the fever consecutive to spirillar infection, which I have termed 'secondary' fever (page 171) ; and as matter of fact, in Europe the two have been considered as alike. The following case is probably one of this kind, the patient not being admitted until after the lapse of 10 days' illness, which might have been spirillar ; but if not, the resemblance to typhus, though not precise, is yet considerable. I possess the details of many serious and fatal cases of fever of uncertain character, some of which might be typhus ; the possibility, however, always existing of their being either remittents or fever-consecutive to the spirillar.

Muss., 25, living in a fever-haunt, servant, 10 days ill ; fever reported at first remitting, then continuous ; e. t. $104^{\circ}4$ p. 98 soft. He is reduced in flesh, countenance dull, eyes injected and sallow, gums discoloured and bleed, tongue coated and dryish, skin supple, much thirst, has appetite : no stool for three days ; has headache and general pains, slight

cough (chest resonant), precordial dullness normal, systole feeble; abdomen soft, no hypochondriac uneasiness. 11 day—m. t. $102^{\circ}4$, p. 96 small, soft, regular, some perspiration in the night; e. t. $104^{\circ}2$, p. 100 fuller, soft, eyes burn, skin moist, and a sense of itching in the chest; one stool. 12 day—m. t. $103^{\circ}2$, p. 102, small, soft, skin dry, thirst, headache, pains, one stool, appetite sharp, he slept a little, but cough troublesome; e. t. $105^{\circ}2$, p. 102, tongue dry, much thirst. 13 day—m. t. 102° , p. 96, skin dry, is giddy, headache less, he slept a little; e. t. $105^{\circ}2$, p. 102, skin dry, no appetite. 14 day—m. t. $104^{\circ}4$, p. 100, skin dry now, slight sweats at night, no headache, but pains in large joints, cough less; e. t. $105^{\circ}2$, p. 102, some headache, bowels not moved. 15 day—m. t. $104^{\circ}4$, p. 108, skin dry, did not sleep well and seems rather deaf (no quinine given); e. t. $106^{\circ}2$, p. 108, much thirst. 16 day—m. t. $104^{\circ}2$, p. 112, thirst, giddiness, headache, pains, constipation, slight sweats at 10 P.M., abdomen somewhat distended, no iliac gurgling; some dullness and tenderness in both hypochondria, no cough, no sleep; e. t. $105^{\circ}6$, p. 108, tremors of lips, eyes very red, staring, he is restless, excitable, easily startled and does not understand what is said to him, pupils slightly dilated, takes food. Up to this date I was unable to surmise the nature of the fever, which seemed to be almost simple pyrexia. 17 day—m. t. $104^{\circ}6$, p. 110, skin dry, lips parched, delirium in the night, no diarrhoea, a doubtful pink spot, less excitement (sedatives given); e. t. $104^{\circ}6$, p. 120, skin dry, no apparent complication. 18 day—m. t. $103^{\circ}6$, p. 102, no stool, sordes appearing, sudamina on the neck, and two pink spots at left costal margin, contiguous, effaceable; e. t. $104^{\circ}8$, p. 112, typhoid state near, dullness of aspect, tremors, urine passed in bed. 19 day—m. t. $103^{\circ}4$, p. 115, no stool after enema, sleep disturbed, slight hypochondriac tenderness on both sides, abdomen retracted; pupils rather dilated, sluggish, conjunctiva injected but moist, floccitatio is present and the supine posture, no spots on the back, those seen yesterday rather fainter, effaceable; heart's systole feeble, but first sound audible; e. t. $103^{\circ}8$, p. 115, skin dry, delirium persists. 20 day—m. t. $102^{\circ}4$, p. 118, sordes, delirium, yet he turns on side and raises the legs, pupils not contracted; the red spots have now disappeared; e. t. $103^{\circ}4$, p. 115, no stool. 21 day—m. t. $102^{\circ}4$, p. 118, no improvement, eyes injected but bright, pupils somewhat dilated, skin dry, no stool, occasional cough, no expectoration seen; e. t. $102^{\circ}6$, p. 110. 22 day—m. t. $100^{\circ}4$, p. 98, skin moist with decline of temperature since 3 A.M., features shrunken, delirium and the typhoid state persist, and greater weakness, no stool, no abdominal uneasiness; e. t. 101° , p. 96, feeble. 23 day—m. t. $99^{\circ}8$, p. 112, skin dry, face pinched, pupils rather contracted; e. t. $98^{\circ}8$, p. 108, small. 24 day—m. t. $98^{\circ}4$, p. 100, skin moist and he is less haggard and delirious, turning on his side, but does not rally; e. t. 98° , p. 90, takes food but indifferently. 25 day—m. t. $98^{\circ}4$, p. 100? very feeble and irritable, abdomen much retracted, pupils large, weakness extreme, less delirium, lies on the right side; e. t. $98^{\circ}2$, p. 102, skin moist. 26 day—m. t. 100° , p. 112, evacuations passed in bed, pupils rather contracted, he is only semi-conscious; e. t. $100^{\circ}4$, p. 110. 27 day—m. t. $99^{\circ}4$, p. 112, strength failing, and death at 11 A.M. Autopsy the same evening—body stiff, muscles red and dark, blood semi-fluid; brain much congested, serous effusion everywhere, puncta vasculosa on section, no alteration of consistence or special lesion; lungs pale and rather collapsed in front, congested behind, weights $17\frac{1}{2}$ (r) and $13\frac{1}{2}$ ozs.; heart weight $5\frac{1}{2}$ ozs., normal aspect, l. side empty, r. ventricle contained a pale clot; liver congested only, weight 3 lbs. 7 ozs.; spleen large, rather soft but otherwise unchanged, wt. $8\frac{1}{2}$ ozs.; kidneys appeared normal, wts. $3\frac{1}{2}$ (r) and 3 ozs.; intestines contracted, duodenum contains bright yellow mucus, the jejunum dark brown, and ileum some dark pultaceous matter, P. glands unchanged; caecum filled with yellow consistent faeces, rectum contained faeces and whitish fluid; the stomach pale, with starry congestion along lesser curvature. Chart of the case in PLATE VI. No. 28. The blood was examined almost daily, and the chief alteration seen was scantiness or absence of fibrillation.

3. **Typhoid or Enteric Fever** (*typhus abdominalis*).—In 74 autopsies of Native patients dying from 'remittent fever' during 1877-8, intestinal lesions like those of Typhoid were found four times, or in the ratio of 5.4 per cent. These instances are summarised below, with the addition of another seen in 1876. Small as is the above proportion, it is probably larger than could be established by the clinical symptoms noted during life; for often the signs of enteric lesion are too obscure to engage special attention. Aid to diagnosis from the presence of a peculiar eruption could not be relied upon at Bombay, where identical 'pink spots' were found in cases proved not to be enteric—viz., in both relapsing and malarious fevers. The recurrent attacks of typhoid have a particular interest, in connection with a similar feature of some remittents, see below: this subject has yet to be worked out, in India.

As to the etiology of typhoid amongst the Natives of India, the difficulty

upon current doctrines would be to account for the rarity of this affection, seeing that urban and rural sanitation is invariably defective. Perhaps, when special enquiries are instituted, enteric fever may be shown largely to prevail; but, hitherto, neither epidemic nor endemic exacerbations of 'country fever' have been traced to it alone.

As there seems a risk of this important subject drifting into a state of confusion, for want of preliminary definitions; it should first be determined whether or not the enteric fever of Europe is always one and the same disease, and next how far the true typhoid is capable of being modified in the tropics. Ulceration of Peyer's glands in the ileum may not pertain to a single infection alone, and diagnosis must ultimately rest upon the presence or absence of specific blood and tissue lesion. At present, the best proof of Typhoid fever is demonstration of a localised enteric lesion, through both characteristic symptoms noted during life and post-mortem appearances such as those described in the casualties below.

Surviving cases diagnosed as Typhoid.—1. M., 18, Indo-European, admitted Nov. 1878, recently arrived from the Mofussil, where he had a prolonged attack of fever and diarrhoea, the present illness being probably a relapse: now 4 days ill, e. t. 104°·6, p. 120. There ensued persistent but remitting and declining pyrexia for 13 days longer, a scanty eruption of pink spots on the 15th day; then comparative apyrexia for 12 days, and finally another relapse of 22 days, during which high fever (remittent in form), no fresh spots, diarrhoea and slight pneumonia near the end; convalescence uninterrupted. There was a flushed aspect; moderate nervous symptoms, quick pulse; no typhoid state. The pyrexia was doubtless modified by treatment (quinine), though comparable with that of enteric relapses, it might also have been of the typho-malarial form described below. Chart 36, Plate VII.—2. M., 14, parentage English, country-born, delicate, seen July 1878; ill 4 days with fever and diarrhoea; the pyrexia was sustained, on 7th day free sweating, but persistence of fever (t. 103°·4), with inflammation of the r. parotid and pulmonary congestion, which lasted 5 days, the t. rising to the end (104°·6, p. 114): then critical decline with sweats, prompt rebound to 105°·2 and the aspect of failing powers; these symptoms however subsided and rallying proceeded, the t. for 9 days longer presenting the character of a declining remittent: a few pink spots were seen on his admission, but no fresh crops. Though regarded as a case of typhoid, I am able to say it also resembled the modified remittent of Bombay. Both the above patients were under my care.—3. M., 21, European, country-born, seen July 1878, 5 days ill with continuous fever and diarrhoea, which persisted for 6 days, and after culmination and a remission, for 8 days longer with declining course and other remissions: there were successive crops of pink spots from 6th to 10th days: no urgent symptoms. The chart is most like that a modified remittent.—4. M., 17, Mussulman, seen July 1878, resident; a history of prolonged illness (3-4 weeks) from fever and diarrhoea; in hospital 16 days, leaving before cessation of fever; during this time the symptoms were not characteristic of typhoid proper, eruption not mentioned, cause of pyrexia much like that shown in the chart No. 14, Plate V.

Nothing definite could be learnt of the probable cause of sickness in the above instances; the three last indigenous cases occurred after rainfall, and I doubt not similar but less carefully watched cases might have been found in other medical wards of the hospital; these all being under special observation. Excepting the first one, the symptoms were not clearly indicative of simple typhoid: it will be noticed the subjects were all young town-residents, and either of European blood or Mahomedan.

Casualties.—1. M., Portuguese, 22, resident in the Fort (a crowded locality); ill 6 days with fever, no cough or diarrhoea, spots not looked for; the abdomen was rather distended and tender on admission: e. t. 104°·2, p. 120 small and firm, there was evident peritonitis, most acute on r. side; next day, t. 104°, p. 150, resp. 40, dyspnoea, no stupor, the local signs led me to suspect ruptured abscess of the liver; death ensued early the following morning. Autopsy—general acute peritonitis, sero-purulent effusion and much lymph exudation, all the viscera healthy-looking except the ileum, at the lower end of which Peyer's patches were found to be intensely inflamed and mostly ulcerated, some solitary glands being similarly altered; at about a foot from the ileo-cæcal valve was a distinct perforation, which in site corresponded with greatest peritoneal inflammation; in some near ulcers the serous coat alone remained intact. No tubercle seen: date Dec. 1876.

2. M., Mussulman, 30, railway fireman, 8 days ill, fever high, pulse quick, pulmonic congestion, splenic enlargement, tongue furred but moist, no diarrhoea; some delirium at night, and presently the typhoid state threatened; for 9 days the pyrexia was persistent and nearly level at 102° - 4° ; it then began to intermit in part; after 6 days death on estimated 22 day of illness; towards the end there was some diarrhoea, which did not increase. Autopsy—brain congested, heart healthy, lungs pale and collapsed, a few spots of firm tissue disseminated throughout, no recent tubercle seen; the bronchial glands black; liver large but normal, spleen large and firm, kidneys congested; the peritoneum widely and intensely inflamed, soft lymph and purulent effusion being present towards the pelvis. Intestines highly vascular, mostly contracted: ileum—its lower 18 inches presented 5 large ulcers 2-3 in. square, or even transverse, jagged, raised, purple-edged, deep, and at one place 6 inches above the i.-c. valve, perforation had taken place; here, too, was a focus of peritoneal inflammation: mucous membrane around tumid, soft, thickened; several smaller ulcers seen deep and of irregular form: contents of gut a bright yellow, scanty, tenacious material. The corresponding mesenteric glands enlarged, highly inflamed, and the larger suppurating; no signs of tubercle noticed. Large intestine—mucous membrane seemed healthy; contents the same sticky, yellow matter. Chart appended. Plate VI. No. 29: it resembles one of remittent fever. Date Sept. 1877.

3. F., Portuguese nurse, 17, resident, ill 15 days with persistent fever and diarrhoea from the first, spleen not felt; some night delirium, tongue white, dryish, complexion dusky and aspect oppressed: for 11 days the fever was high (103° - 5°), level, sustained; pulse extremely rapid; no eruption seen on admission, some fulness and tenderness of r. iliac region, 4 stools in the first night, thin, yellow, slightly feculent and passed without uneasiness; pulmonic congestion followed: with persistence of fever the diarrhoea did not increase; lumbrici were passed; the tongue became dry and the abdominal signs persisted, not increasing, deafness, eruption not noted (? looked for), the stools varied in aspect, some being liquid, lumpy, yellow; exhaustion came on and death on estimated 25 day of illness. Autopsy—at lower end of ileum several large and small ulcers seated in the Peyerian glands: within a foot of the i.-c. valve were 3 large, ovoid, defined, raised sores, with pink edges and irregular surface, in a state of granulation: the remainder, about 9 in number, were smaller and more superficial; some of the pink patches were simply enlarged and free from ulceration: the solitary glands here and in the neighbouring large intestine, were frequently enlarged, red, and ulcerated. I noted that all the ulcers appeared to be granulating from their margins, the sloughs being thrown off: a few small ulcers were seen as high as 5 feet above the valve. The lower part of the rectum showed patches of circumscribed redness: elsewhere the intestines were healthy. The mesenteric glands in connection with the above were still enlarged. Other abdominal organs congested only. Date May 1878.

4. M., Mussulman, 20, resident, admitted with fever and cough of two months' duration (so stated), tongue florid, abdomen tense and tender, bowels irregular; no dullness of the chest. The pyrexia was high (102° - 4°), level and sustained for the 7 days he lived; pulse quick, especially towards the end; on the first day one semi-consistent stool, never any diarrhoea, but the abdomen continued tender all over; the evacuations were sometimes noted as scanty, offensive, yellow, containing mucus and some feculence; vomiting was a late symptom (possibly due to medicine): the typhoid state speedily supervened, some indistinct pink spots were noted, no iliac gurgling the day before death. Autopsy—no lung disease: spleen large (9 ozs.): ileum—beginning four feet from the i.-c. valve a series of ulcers of varying size, longitudinally directed, edges raised, surface deeply excavated, or still covered with a yellow slough; all evidently located in Peyer's glands, the intermediate mucous membrane not being inflamed; the iliac surface of the valve itself is occupied by an ulcer; the corresponding mesenteric glands enlarged (not tubercular): no peritonitis: mucous membrane of large intestine simply vascular. Oct. 1878.

5. M., Hindoo, 18, admitted at height of epidemic, June 1877, a famine immigrant, 4 days only in Bombay; 12 days ill with fever and constipation; no eruption seen; max. p. 105° - 4° , the typhoid state present; fever remitted for 5 days, when death ensued. Both solitary and agminate glands of Peyer at end of ileum were sloughing (the exuvia being of yellow colour) and ulcerating: liver 2 lb. 14 ozs. mottled; spleen $5\frac{1}{2}$ ozs., healthy; patches of pulmonary apoplexy in both lungs. The blood examination in my hands furnished negative results, but as there is a note on the case of the spirillum being seen, this instance came to be regarded as one proving the presence of the spirillum in typhoid fever: at the most it could show only such conjunction of specific and enteric fever, as has been noted at St. Petersburg. So long as seen, the course of pyrexia offered nothing distinctive, being modified by treatment.

This last case indicates the possibility of typhoid arising outside Bombay. In the town excellent water brought by iron pipes from a distant source,

was in universal use ; a few wells, however, still being resorted to. So far as learnt on frequent inquiry, febrile sickness seemed very rarely attributable here to foul drinking water.

4. Malarious Fevers.—For the purpose of comparison and as illustration of common experience at Bombay, I append the notes and clinical charts of five cases ; these include two of the intermittent, and three of the remittent type. The best examples of malarious fever occur amongst individuals recently arrived in the town, from unhealthy districts of the interior.

Intermittents.—Well-marked ague is rare in Bombay : ‘pernicious’ ague and the ‘accès jaune’ or pronounced ‘bilious’ forms, are never seen to arise here. Nor in the interior, is the so-called ‘jungle-fever’ nearly so frequent as has been supposed. Very little definite information respecting typical ague has been acquired ; and nothing certain regarding that residual pyrexia of intermittents, to which Dr. Wunderlich long since drew attention. My notes on the aspect of the blood in malarious fevers are not yet arranged ; the appearances described by Dr. A. Laveran (*Lancet*, Nov. 12, 1881), I have seen, without placing the same interpretation on them ; and in PLATE II., fig. Z is copied from a Memoir of Drs. Cuboni and Marchiafava, of Rome : see also foot-note at page 419 : the true significance of such quasi-organic forms is not yet settled.

CASE 1. Mild Ague.—M., 30, Goanese cook, went with his master to the Vihar jungles (near to Bombay) in July 1877 (rainy season), and there caught fever ; having to return a fortnight later, and coming straight to hospital. The attacks had then become irregular, and for two days after admission no fever occurred ; a series of five daily paroxysms then took place, with pronounced stages, duration from about 6 A.M. to 4 P.M., extremes of temperature unknown ; no considerable liver or spleen implication, or headache, or thirst ; latterly pains in the calves and subsequently rheumatoid pains in shoulders, back, neck, the attacks seemed almost purely pyrexial. The blood taken during rigor, showed clouded plasma and irregular distribution of red discs, also numerous quiescent, yellowish particles. Chart No. 31, Plate VII.

Fatal Ague. CASE 2.—M., 25, arrived from the Persian Gulf three days before admission (Nov. 1878) with fever of uncertain duration, and was much reduced. The pyrexia subsided for a day or two, and then returned with diarrhoea ; again briefly declined, and finally rose with the typhoid state and fatal exhaustion after 28 days : the paroxysms were much pronounced. The blood on examination during life was found to coagulate slowly, contain few leucocytes, but much free protoplasm in small masses ; no moving particles seen. At autopsy—some cerebral congestion and effusion of serum, clots in r. side of heart, lungs collapsed and not congested ; the liver dark, smooth, soft ; the spleen enormous (46 ozs. weight, or nine times the normal), dark, soft, capsule thin, no infarcts seen ; kidneys mottled as if fatty ; some vascularity without erosion of mucous membrane of colon, no other change noted in intestinal canal (observer, Mr. S. A.) The Chart (No. 30, Plate VI.) shows one-day paroxysms of quotidian or tertian form and sometimes tending to blend ; the spontaneous subsidence and recurrence of pyrexia are noteworthy. There had been slight jaundice ; eruption not mentioned ; treatment antiperiodic and supporting.

Remittents.—The bilious, congestive, and insidious types of fever formerly distinguished, are now rarely seen in the larger towns ; in the more malarious country districts they may possibly still persist. At Bombay, remittents tend to a sub-continuous and persistent type which is little amenable to quinine ; and the influence of season on their production is by no means invariable.

The milder attacks might sometimes be termed ‘*febriculæ*’ and ‘simple continued fever’ ; but their distinction from incidental pyrexia (e.g. due to heat and climatic or dietetic irregularities) and mild enteric fever, would often be difficult—perhaps the effect of quinine furnishes the best guide here. Ordinary attacks of 7 to 21 days’ duration, present a certain regularity of construction which permits of their being regarded as ‘compound’ ague-forms ; thus, the commencement and end are distinctly paroxysmal, whilst their mid-course presents febrile exacerbations prolonged over 2, 3, or even 4 days, and separated by distinct remissions. I find the maximum temperature

of 104° – 5° F., to attend rather oftenest the initial and subsidiary paroxysm, blending of the exacerbations frequently leading to some abatement of temperature. Severe and fatal remittents are apt to be of irregular form and degree. Complications of local congestion and inflammation always threaten in these prolonged paroxysmal fevers, and in some form are commonly present. Varieties of remittents lately seen, were the ataxic and the relapsing. The first-named occurred chiefly amongst Mussulman weavers, and presented a typhus-like prostration, with eruption of red spots and cerebral, pneumonic or abdominal complications; some jaundice was usual, and the pulse was quick as well as feeble.

The relapsing variety of remittents was the more frequent, analogy here pointing to typhoid rather than spirillum fever; and this seems to be the kind which has been termed 'typho-malarial' fever. It is a distinct variety, attended with a sub-continuous type of pyrexia and nervous complications, and liable to recur once, twice, or oftener. The primary attack is not peculiarly severe, the succeeding apyretic interval lasts 1-7 days and the recurrent attack 5-20 days; subsequent events are more irregular. Pyrexia is not pronounced, but its paroxysmal character is marked; severe headache, chills and vomiting frequent; fulness and tenderness of the liver, spleen and mid-abdomen supervene, diarrhoea is frequent, but usually of dysenteric character; scanty eruptions of pink spots often occur, and pulmonic complications. Cases were commonest in the young, females, and the less robust subjects. Emaciation, debility and protracted convalescence were the rule: an early fatal termination was rare, and as enteric lesion has not yet been found in connection with such cases, I am unable to affirm that a true typhoid element is here present.

In PLATE VII., Chart 34 belongs to an instance of this kind, and I have placed it next to one (No. 36) of a case of supposed enteric fever for purpose of comparison; the resemblance being considerable. The two other examples of remittents detailed below were more distinctly malarious in character.

Mild Remittent Fever. CASE 3.—M., 25, ward-servant at J. J. Ho., in good health, admitted Dec. '77; fever came on during forenoon with chills, subsiding at 2 A.M. with sweats; it returned with chills on the second day, declining with sweats, and again exacerbating at night, when delirium temporarily supervened: there was a decline with some perspiration on the following morning; headache severe. On the third day, febrile exacerbation in the afternoon, subsiding with sweats at night; and again on the sixth day, tenth and eleventh, the paroxysms being neither regular nor equal. General pains and sleeplessness continued; constipation of the bowels: there was slight epistaxis on the eighth night (t. declining), and some signs of lung consolidation (r. side) on tenth day of remission: pyrexia and cough then increased and night delirium returned. Quinine injected hypodermically did not now check the fever; on the fourteenth day an intermission, and a more pronounced one on the seventeenth, when the remittent type seemed to end, and final ague-paroxysms appeared. The blood was occasionally examined for the spirillum with negative results; skin eruption not noticed: the case shows that even moderate fever when persistent may be attended with local complication, and most remittents, however simple in aspect, are liable to be thus complicated. Chart No. 32, Plate VII.

Fatal malarious Remittent. CASE 4.—M., 23, admitted Nov. 1878; said to have been 8 days ill; for 5 days longer fever though not high (104° max.) or sustained, was of continuous type, then occurred an intermission, followed by return of somewhat irregular pyrexia and after 6 days another decline to 98° : again a rise with remittent form and occasional high paroxysms for 10 days; and a final exacerbation of high fever (max. 105° ·4) lasting 6 days; with decline and death 32 days after admission. The typhoid state gradually came on, with various complications, viz. delirium on 12th day, some jaundice at the first reaction on 16th day, with dysenteric, bloody stools and some hepatic tenderness, an eruption of pink spots, pains in the shoulders; lastly on 33rd day rigors, renewed pyrexia, impaired resonance on l. side of chest, with cough, more persistent delirium, rapid and feeble pulse, and death by exhaustion. The pupils were not changed, eyes remained bright, gums not discoloured, and in general the true typhus state was not developed; whilst on the other hand the depression was great, the aspect apathetic, there was a copious eruption of pink spots not effaceable when first seen

on estimated 18th day of disease, yet not altogether petechial, for they had disappeared five days later. The final exacerbation lasting 6 days, may have contemporary with the sub-peritoneal hæmorrhage found after death at decline of temperature. The case was watched with interest as somewhat unusual at the J. J. Hospital, in so far that with this persistent pyrexia there was no evident organic lesion, such as commonly arises in course of prolonged remittents: even the suspected pulmonic dullness on l. side of chest may have been illusory, and the idea of acute tuberculosis had occurred to Mr. S. A. and myself. Treatment by feeding and stimulants, and attention to special symptoms.

Autopsy 5 hours after death; blood dark but sets, muscles dark red, firm: brain pallid, wet, soft; heart substance pale; lungs pale, tough, dry, lower lobes congested (hypostatic): liver enlarged (3 lbs. 6 ozs.) pale, smooth, bloodless, greyish; gall-bladder empty: spleen much enlarged (14 ozs.), dark, pulp soft, capsule smooth; no infarcts seen. Kidnies rather large, 5 ozs. (R) and 4½ (L), smooth, mottled, capsule non-adherent, section pale. Mucous membrane of colon unchanged, so of small intestine, except at places in jejunum and ileum where vascular patches were seen: Peyerian glands unaltered; mucous membrane of rectum corrugated: intestinal walls generally thin. In the sub-peritoneal tissue on under surface of diaphragm, at left side, above the spleen and extending below the attachment of the muscle, were numerous hæmorrhagic spots, clustered and often fused: some were also seen on the right side: on the pleural aspect of the diaphragm none were visible (Mr. S. A.).

Microscopic examination of Liver on same day; I found the hepatic cells had undergone extensive fatty degeneration, some lobules being almost wholly disintegrated, and at the same time greatly atrophied. The inter-lobular tissue is then hyperplastic, large rows and collections of leucocytes being intermingled with the fibrous stroma, and encroaching on the exterior of the lobules; many hepatic cells were charged with dark green, yellow or black pigment granules and masses (which would account for the slaty hue of the organ): blood-vessels not distended.

Kidnies—granular choking of the tubes and incipient fatty degeneration of epithelium: Malpighian tufts not affected.

Diaphragm at site of petechiæ—muscular fibres partly normal, partly clouded with fine granules, not wasted, both appearances being seen side by side; + acetic acid the granules are found to be fatty, no oil-drops visible, nuclei of sarcolemma persist.

Blood—whenever examined no spirillum seen; at first some of the scanty proplasm was vacuolated; at last large granule-cells or protoplasmic masses were seen containing dark granules, free granules in the serum and increase of leucocytes: at all times deficient or slow fibrillation. Chart No. 33, Plate VII.

Relapsing Remittent (? Typho-malarial) fever. CASE 5.—M., 30, Goanese cook, out of work, admitted Oct. 1879, having had fever (seemingly paroxysmal), at his home for a week: general condition low, e.t. 101°-8, p. 76 weak, much headache and pain in limbs, tongue coated but moist, no implication of liver or spleen, one stool, no iliac gurgling, no eruption or jaundice, or sweats. The pyrexia soon began to decline, remitting daily and after 8 days the temperature was at normal. During this time he became weaker, the heart's systole was very feeble, tongue brown and dryish, night delirium, pupils normal. Urine free from albumen, the chlorides in excess and bile-pigment; it was clear and specific, gravity 1020. On estimated 12th and three succeeding days, some bright scarlet spots appeared on the r. chest, neck and forearm, and there was also a decided mottling of the skin; application of cupping glasses did not bring out more spots, but the cup-marks were deep and lasting. After 2 days of apyrexia, during which the man's appetite was good, the chlorides in the urine diminished and general improvement was beginning, the fever returned with chills in spite of the large doses of quinine that were given; and with a preliminary ague-like paroxysm, it became semi-continuous (19th day of illness), remaining so for 10 days, when the paroxysmal character reappeared, and by deeper remissions the normal level was reached 9 days later. This recurrent event lasted 19 days, or somewhat longer than the first attack, so far as known. The persistence of high temperature at near 102° F. was not accounted for by any apparent local disease, nor was the man's strength so much reduced as might be expected: he continued to eat meat; the urine was pale and clear; pupils normal, no delirium; chills and sweats were not marked until after the fever became paroxysmal; spleen not affected and liver not enlarged, the abdomen was retracted and some emaciation came on; the heart's action continued feeble, but there was no dilatation, or murmur. At the beginning of the relapse and on the next two days there was a plentiful eruption of pink spots and a few appeared at the close, some mottling of the skin also was present. About the middle of the relapse, there was vomiting three-quarters of an hour after taking food, leading to the idea of duodenal irritation, but no local uneasiness existed; at the end there were marks of slight hemiplegia of the r. side, with wasting of the limbs and impaired cutaneous sensibility. The man had a pallid, earthy-tinted countenance, but none of the usual signs of cerebral lesion were noted: the urine seemed healthy, and no other local lesion was found. Convalescence was slow, but it was progressing when he left hospital. No spirillum in the blood. Chart No. 34, Plate VII.

DESCRIPTION OF THE CLINICAL CHARTS IN PLATES IV., V., VI. AND VII.

Temperatures according to Fahrenheit's scale and the Centigrade: Pulse-numbers and Respirations per minute. The Day of Disease is that stated by the patient. State of the Blood: when the Spirillum was seen the sign is +, when not seen it is -; when not seen in fresh blood, but seen in dried blood after the addition of acetic acid, the sign is ±; when no examination was made, there is no entry. The observations here recorded, are those made at morning and evening. I have added the line of normal temperature, 98°·5 F.; and also above the stage of disease, and below some of the chief symptoms noted at each stage.

PLATE IV.

- No. 1. Single or Abortive attack of moderate severity, the result of inoculation at the autopsy of a fever-patient. Case No. 6 in the list at p. 403.
- No. 2. Attack with one Relapse. Patient a lad æt. 14; illness moderately severe, and no clear signs of 2nd relapse. The decline of the pulse after invasion-attack is shown, and course of respiration; fuller details of the Relapse in Plate I, page 358.
- No. 3. Attack with two Relapses. The case is described at page 59. The movements of temperature at close of the earlier crises are noticeable, as due to transitory complications; there was no sign of further relapse during three weeks after the latest here shown.
- No. 4. Attack with three Relapses. Details at page 65. The relapses are seen to be both briefer and longer delayed at each recurrence; the final stages were not witnessed in hospital. There were transitory complications after the first and second attacks, and the type of fever was low.
- No. 5. An unusual Variety of the Invasion-attack. Contagion in hospital; for details see page 161, CASE XXIX.
- No. 6. A Variety of First Relapse. M., 28, a muscular Arab sailor: the earlier febrile intermissions were as pronounced as many true crises, and yet the blood-spirillum was always to be seen. CASE XXXV., p. 165.
- No. 7. A Variety of Second Relapse, showing similar disintegration of the pyrexia. See CASE XXXIV., p. 164.
- No. 8. An Ague-like paroxysm occupying the position of a first relapse; not uncommon. Patient M., 30, Hindoo from the Deccan: general symptoms slight, but the spleen became distinctly larger, though not tender, at this time; the spirillum was seen in the fresh blood, prior to acme of the attack.
- No. 9 and 9a. Latent Relapse, according to the ordinary temperature-chart; but by means of the 6-hour observations which were made, a distinct febrile paroxysm was detected, occurring in the night. Details in CASE XXXVI., p. 166.

PLATE V.

- No. 10. An intercalated Relapse, coming after the Invasion-attack. Details in CASE XXXII., p. 164.
- No. 11. Defervescence of Invasion by Lysis, with subsequent complication and then a Relapse. Details in CASE XXII., page 82.
- No. 12. Febrile Complication of Asthma with bronchitis, the result being effacement of the first apyretic interval. Details in CASE LXXIV., p. 206.
- No. 13. Complication of Hectic fever (phthisis pulmonalis), also tending to obscure specific pyrexia. See CASE LXXVI., p. 207.

- No. 14. A rare Complication of *Spirillum* fever with a Remittent of typhus-like aspect. The blood-scrutiny was incomplete, but it shows specific pyrexia to have been present at one time. CASE XCIX., p. 234.
- No. 15. An Invasion-attack ending fatally at near acme. The pyrexia was not so irregular as is frequent in fatal first attacks. Patient, F., 22, ward nurse, of slender physique, but on active duty when seized in her ward. Fever came on suddenly with chills and vomiting; general symptoms pronounced and the spleen was unusually tender and enlarged, there was jaundice, bilious vomiting, constipation and rigidity of the abdominal walls, very quick breathing and the characteristic *facies*. Spirilla abundant and found after death, retaining their activity for 30 hours longer in specimens of the blood. The liver and spleen alone were examined, and they were much enlarged and congested.
- No. 16. Fatal first attack in an infant, showing much irregularity of temperature. The mother suckled this child during her own specific illness (a pronounced relapsing attack, at the close of which the infant became affected). Many spirilla present and pulse very frequent, but respirations not more than 20-30 per minute; at the autopsy, no coarse lesion found.
- No. 17. Fatal first attack in an aged man, showing much irregularity of temperature. The illness supervened 16 days after his admission for chronic bronchitis, and doubtless arose from contagion in hospital. On the first day there was only feverishness at night, and at no time was the pyrexia high or sustained; the general symptoms, also, were mild, but much tenderness (without fulness) was present in both hypochondria; the blood-parasite abounded and furnished the sole means of diagnosis. At autopsy—liver and spleen hardly enlarged.
- No. 18. First attack ending fatally, with a prompt rebound of temperature after the crisis. CASE XLI., page 172.
- No. 19. First attack proving fatal immediately after the crisis. CASE XLII., page 173. This chart also serves for comparison of malarious fever with the spirillar.

PLATE VI.

- No. 20. Secondary fever ensuing after the first crisis of a spirillar attack. CASE XL. page 172.
- No. 21. Secondary fever following the first Relapse. CASE XLIII., p. 174.
- No. 22. Secondary fever much prolonged, succeeding to a first Relapse. CASE XLIV., p. 175.
- No. 23. Fatal consecutive fever (enteritis) following the Relapse. CASE LXXXVII., p. 216.
- No. 24. *Spirillum* fever complicated with Pneumonia, supervening during the first Apyretic Interval and blending with the Relapse. CASE LXX., p. 204.
- No. 25. Pneumonic complication in another fatal case. *Typhus biliosus*. So far as witnessed, the pyrexia was highly irregular: see page 239.
- No. 26. Pneumonia and non-spirillar fever directly following a specific attack. CASE LXIX., p. 204.
- No. 27. Dysentery attending spirillum fever: pyrexia irregular. CASE LXXXVIII., p. 219.
- No. 28. Typhus-like fever seen at Bombay. *Vide* page 437, Appendix B.
- No. 29. Enteric fever at Bombay. *Vide* page 440, Appendix B.
- No. 30. Fatal Malarious fever. *Vide* page 441, Appendix B.

PLATE VII.

- No. 31. Mild Ague. See Appendix B, page 441.
- No. 32. Mild Remittent fever. See Appendix B, page 442.
- No. 33. Fatal Remittent fever. See Appendix B, page 442.
- No. 34. Remittent fever with relapse, as seen at Bombay. Appendix B, page 443.
- No. 35. Cerebro-spinal Meningitis. See Appendix B, page 436.
- No. 36. Tropical Enteric fever at Bombay. See Appendix B, page 439.

DAY OF DISEASE	TEMPERATURE (°C)	PULSE (b/min)
1	100	105
2	100	105
3	100	105
4	100	105
5	100	105
6	100	105

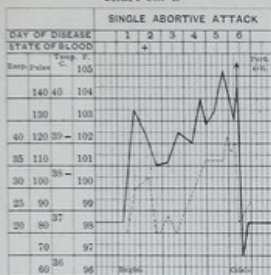
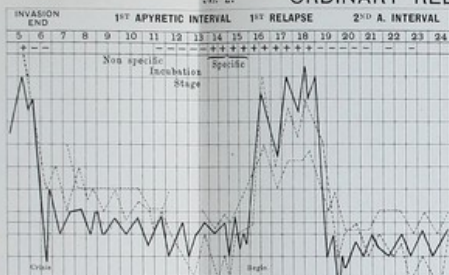


Figure 1 is a line graph showing the course of fever in a patient with typhoid fever. The x-axis represents time in days, from 5 to 24. The y-axis represents temperature. The graph is divided into three phases: 1st Apyretic Interval (Days 5-11), 1st Relapse (Days 12-18), and 2nd A. Interval (Days 19-24). The temperature curve shows a sharp rise at day 5, a fall to a baseline, a second rise at day 12, and a third rise at day 18. The temperature remains elevated during the relapse and then falls again at day 21. The graph is labeled "Non specific incubation Stage" and "Specific".



INVASION 1ST INTERVAL 1ST RELAPSE 2ND INTERVAL 2ND RELAPSE

8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35 36 37 38 39

Weight 59 lbs 59 lbs

Boundary, Serpentine, Crisis, Tremor, Burning in abdomen, Faint

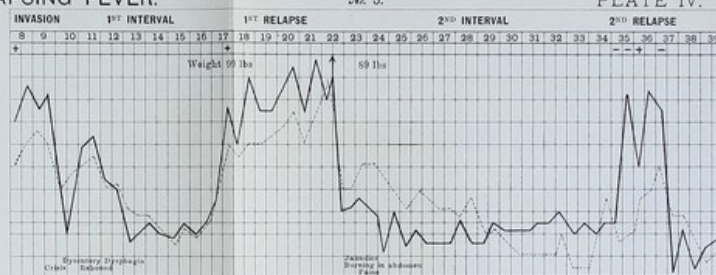
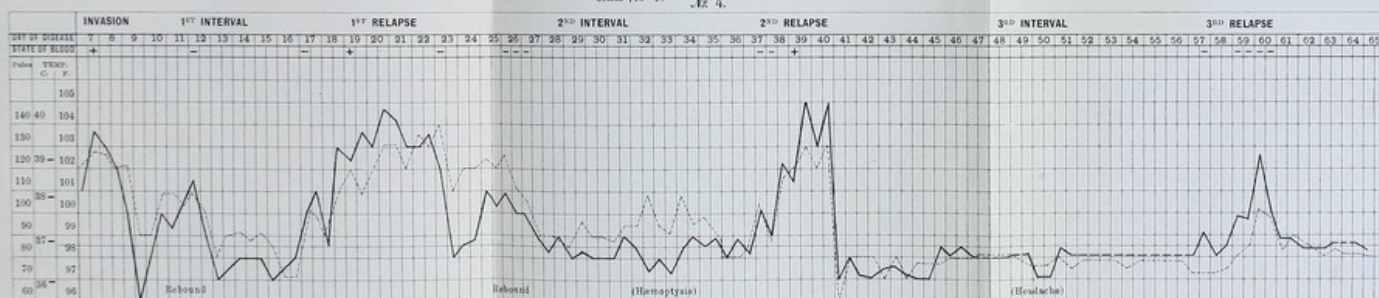
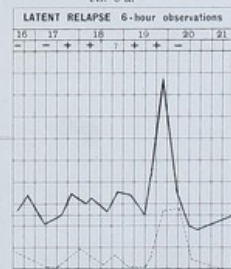
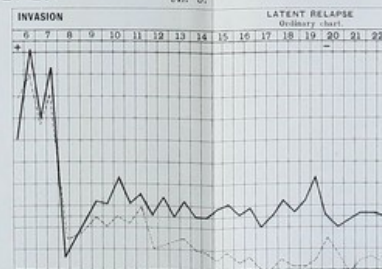
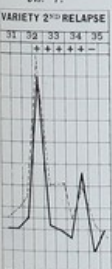
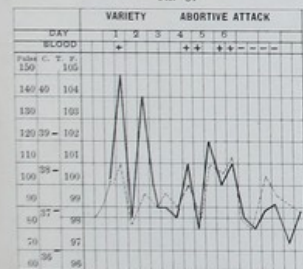


Figure 4 is a line graph titled "COURSE OF DISEASE AND STATE OF BLOOD". The X-axis represents the "DAY OF DISEASE" from 0 to 65. The Y-axis represents "Pulse" (60-140) and "Temp." (96-105). The graph is divided into several phases: "INVASION" (Days 0-7), "1ST INTERVAL" (Days 8-18), "1ST RELAPSE" (Days 19-24), "2ND INTERVAL" (Days 25-36), "2ND RELAPSE" (Days 37-41), "3RD INTERVAL" (Days 42-57), and "3RD RELAPSE" (Days 58-65). The graph shows two lines: a solid line for Pulse and a dashed line for Temp. The Pulse line shows significant fluctuations, with peaks around Day 10, Day 21, Day 40, and Day 60. The Temp line shows a general downward trend from Day 0 to Day 10, followed by a period of relative stability around 98-99 degrees, and then a slight increase during the 3rd Relapse phase. Labels "Rebound", "Hemoptysis", and "Hematuria" are placed below the graph at specific points.



4293.



IN ALL CHARTS TEMPERATURE AS CONTINUOUS LINE: PULSE AS DOTTED LINE: RESPIRATION AS INTERRUPTED LINE.

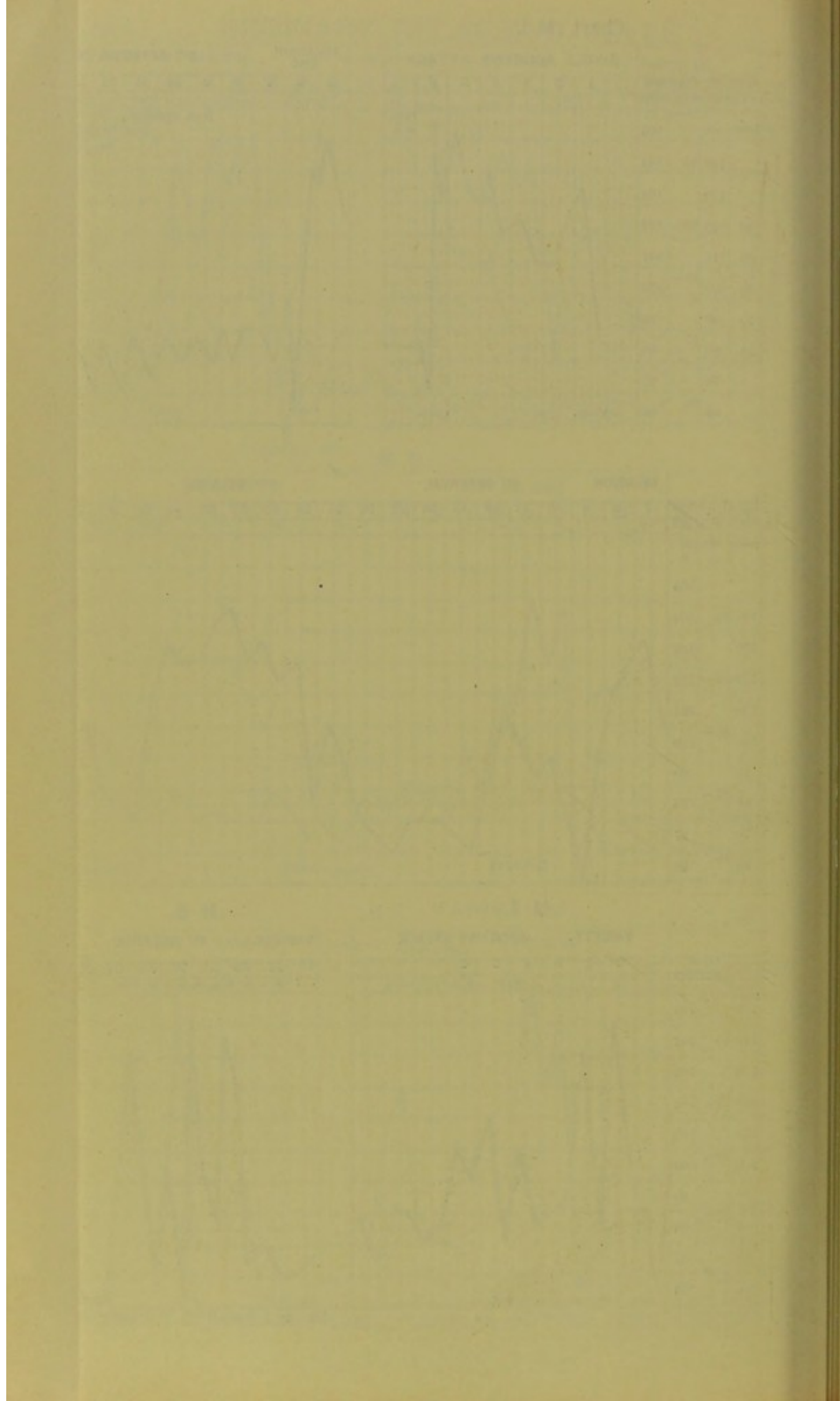


Chart .12 10. VARIETIES OF RELAPSING FEVER .12 11.

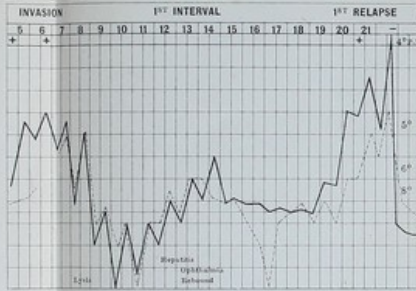
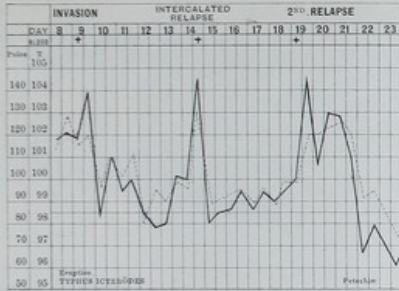
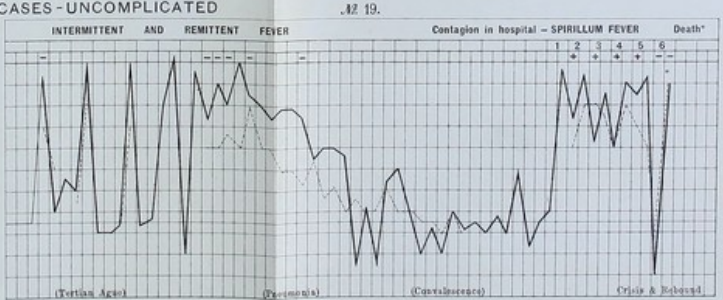
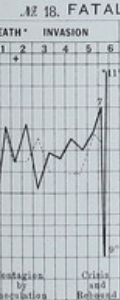
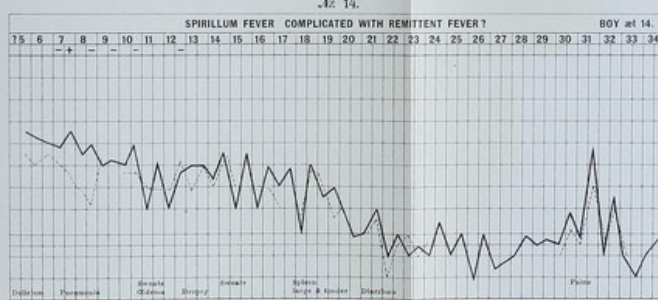
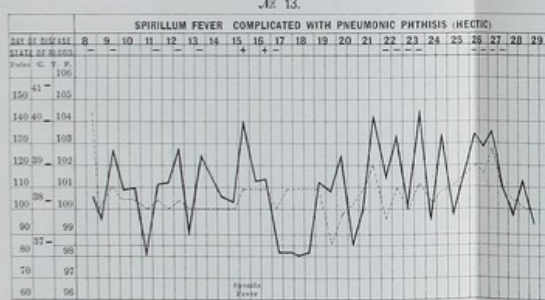
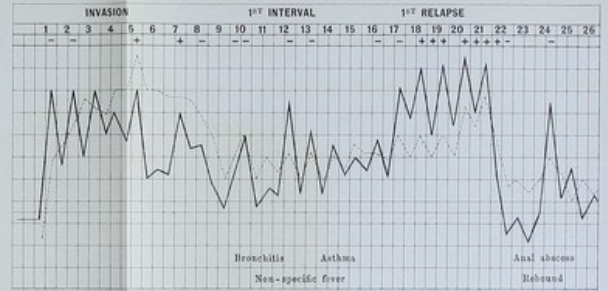
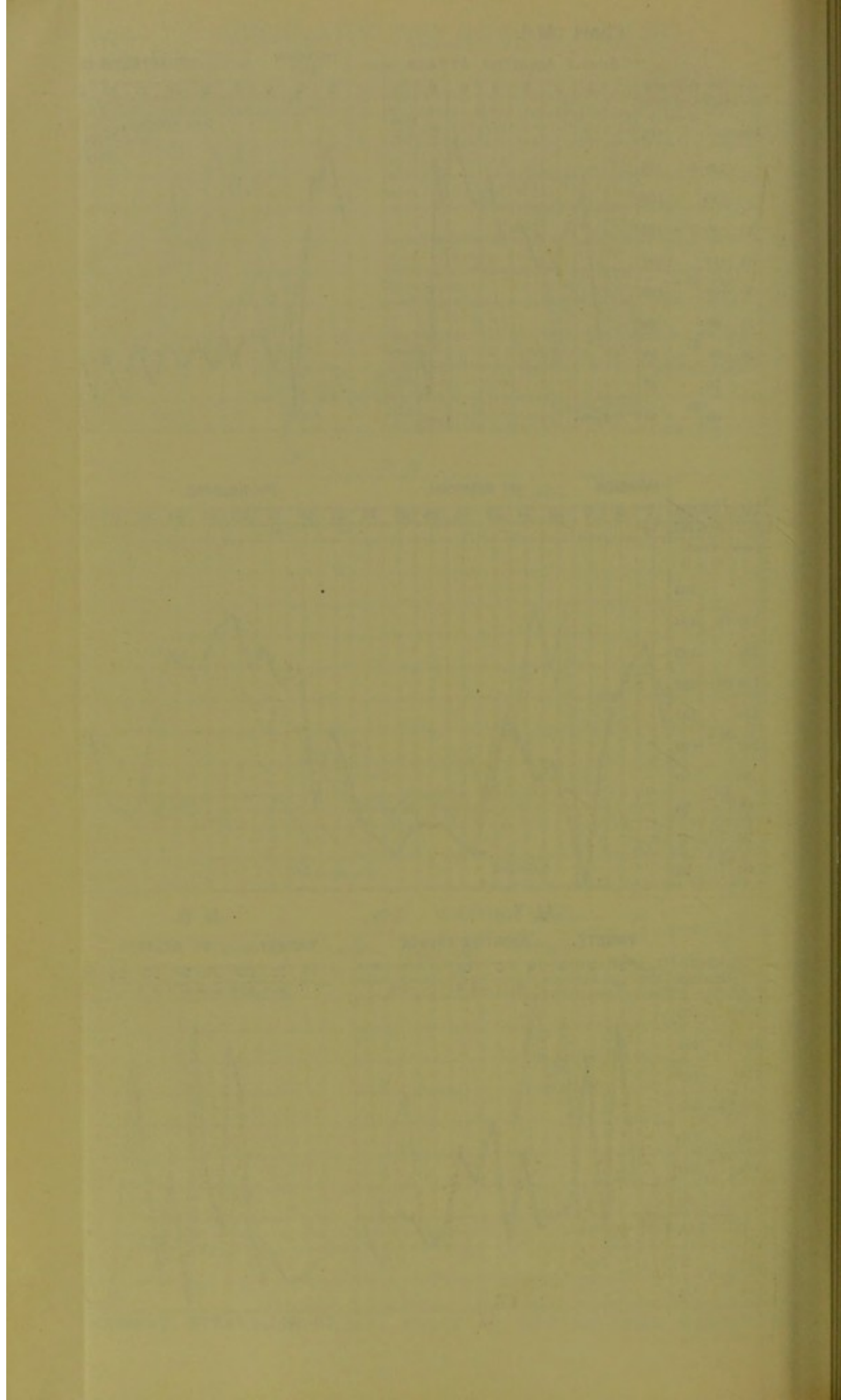


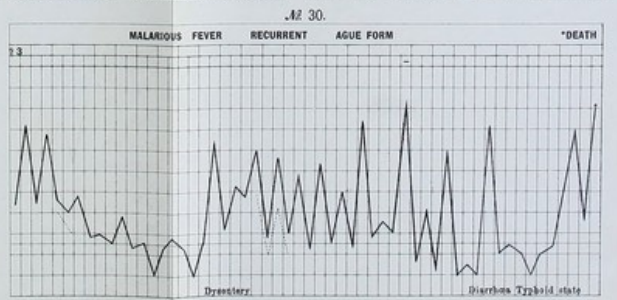
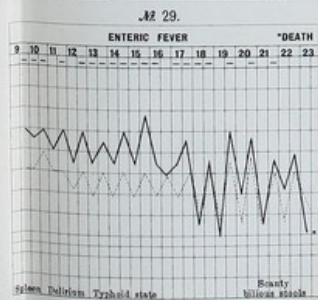
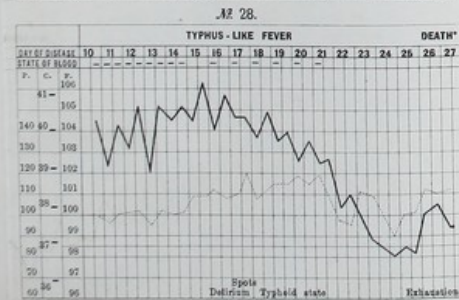
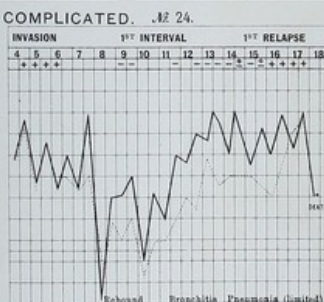
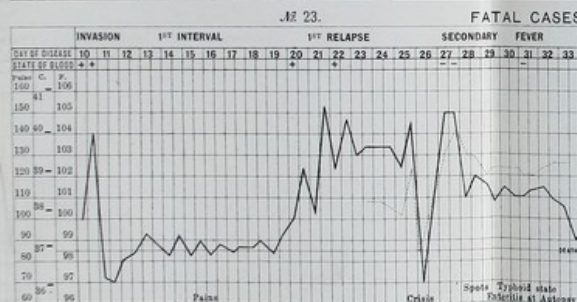
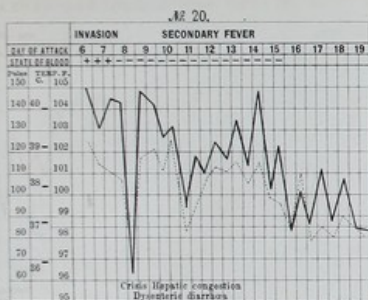
Chart .12 12. COMPLICATION Chart .12 12. PLATE V.





RELAPSING FEVER COMPLICATED WITH SECONDARY PYREXIA.

PLATE VI.



Handwritten text at the top of the page, possibly a title or header.

First section of handwritten text, appearing as a paragraph or list.

Second section of handwritten text, continuing the narrative or list.

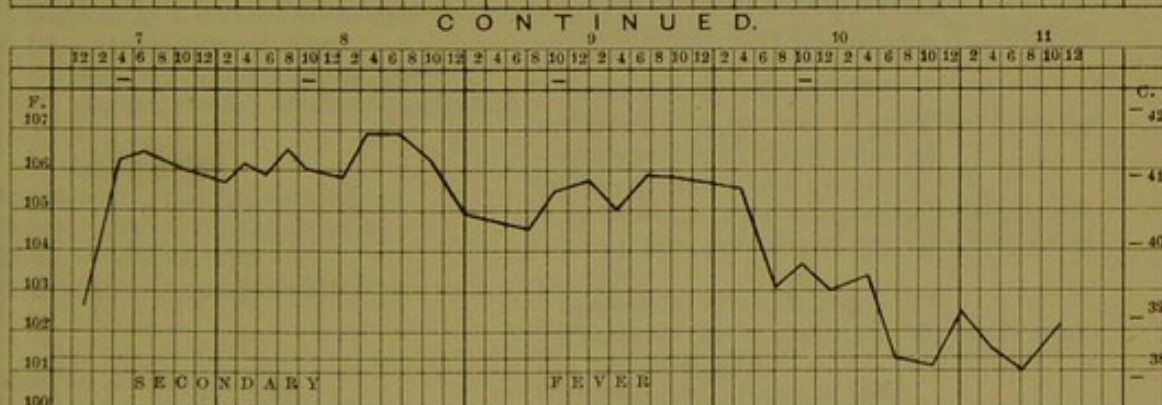
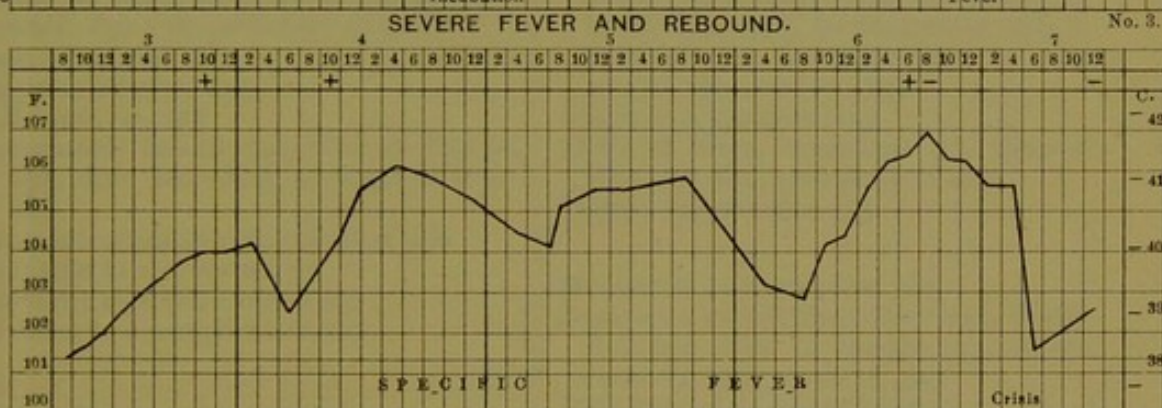
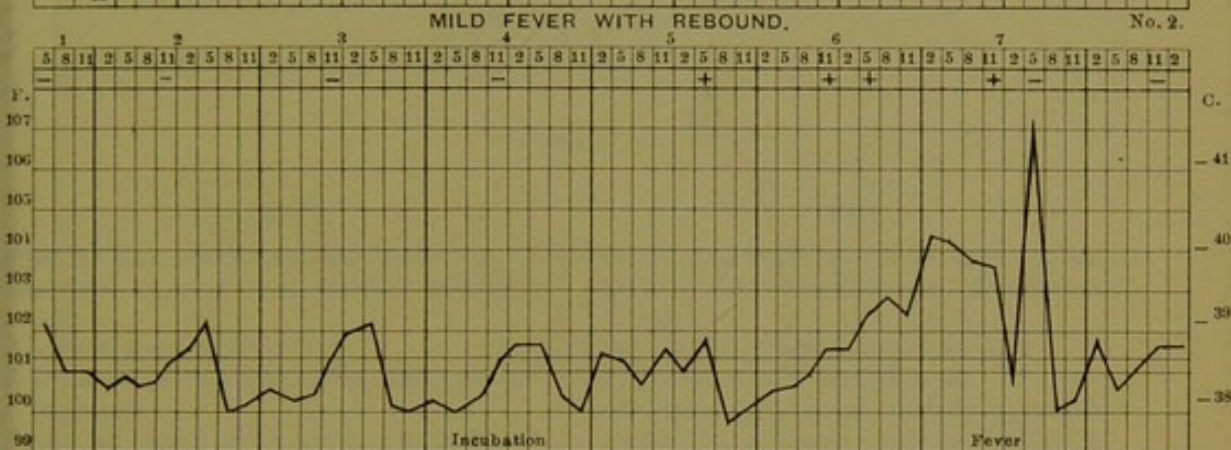
Third section of handwritten text, showing more detail or a new entry.

Fourth section of handwritten text, possibly a conclusion or summary.

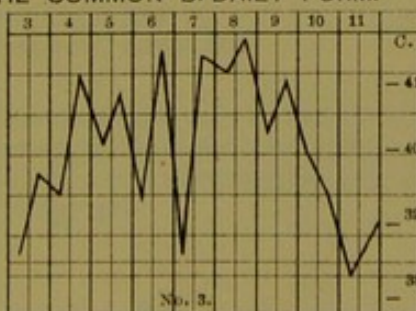
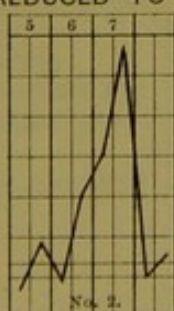
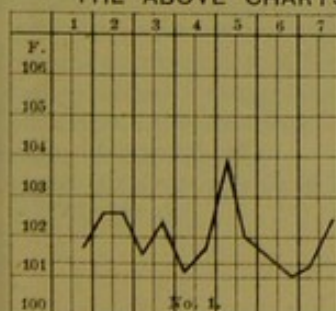
Final section of handwritten text at the bottom of the page.

SPIRILLUM FEVER IN THE MONKEY.

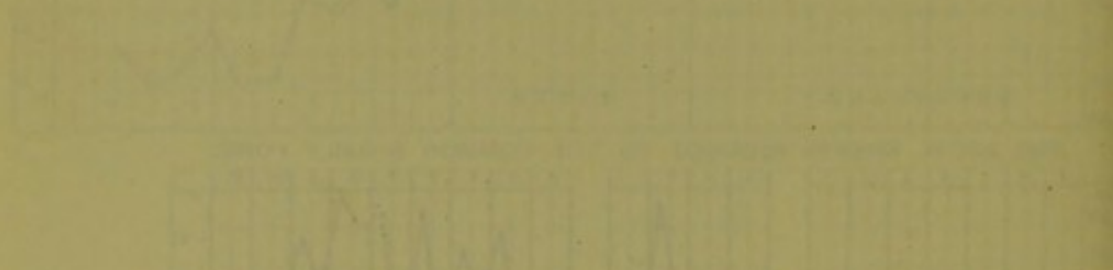
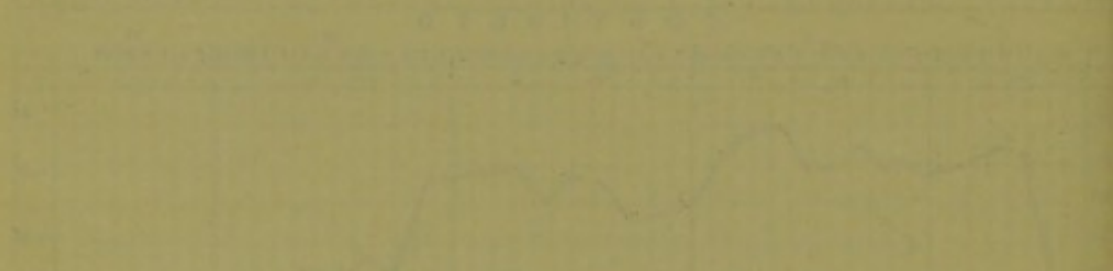
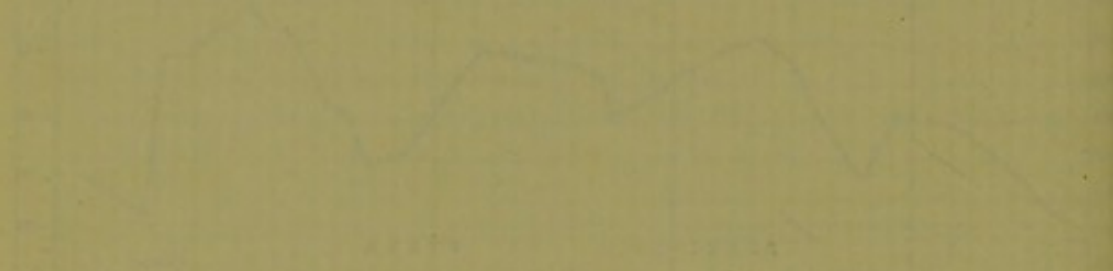
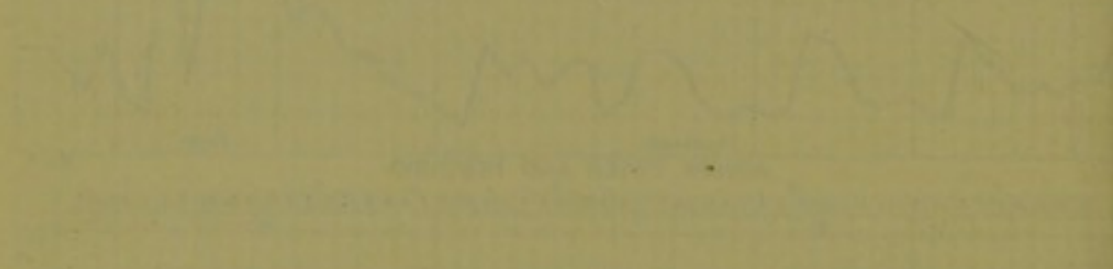
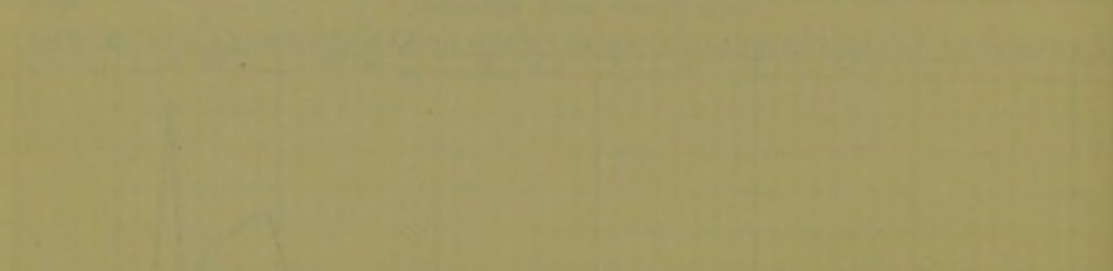
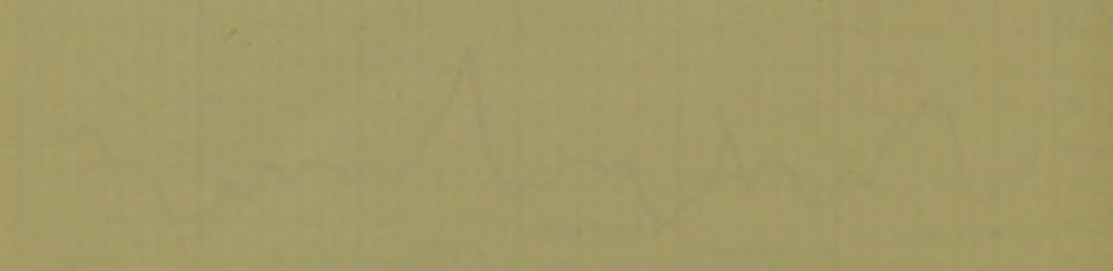
PLATE VIII.



THE ABOVE CHARTS REDUCED TO THE COMMON BI-DAILY FORM.



THEORY OF THE EARTH
CHAPTER I. OF THE ORIGIN OF THE EARTH.



INDEX.

ABD

ABDOMINAL symptoms reviewed, 117
 Abortion as a result of spirillar infection 226
 Abortive form of fever 129
 Accidents of the fever 170, their pathology 419
 Acme of fever, symptoms 72, pyrexial state, &c. 136, pathology of 418
 Age of patients 369
 Ague at Bombay 441
 Albuminuria, 225
 Anatomical lesions 248, ditto in Europe 295, ditto according to stage of fever 286
 Antecedents of specific infection 228
 Appetite as a symptom 90
 Apyretic intervals defined 33
 Asthma as a complication 206

 BED-SORES as a complication 198
 Blood, examination of, 333
 „ detailed description of, 339
 „ general aspect of during infection 336
 „ healthy 336
 Bombay city, normal data of, 14
 Bowels, state of during fever 95
 Brain, lesions of, 250
 Bronchitis as a complication 199

 CÆCUM, lesions of, 284
 Cast and race of patients 370
 Cerebral hæmorrhage as a complication 186, its brain-lesions 254
 Cerebral meningitis as a lesion 252
 Cerebro-spinal meningitis at Bombay 437
 Climate as a predisposing influence 372
 Colon, lesions of, 284
 Complications of spirillum fever 170, their pathology 419

ENT

Concurrence with spirillum fever of enteric f. 233, of typhus 234, of malarious f. 235
 Consecutive or secondary fever 171
 Constipation as a symptom 95
 Contagion 375, local modifying conditions 375, amongst town residents 376, in families 378, in tenements 384, in hospital 389, by inoculation of man 403, of the monkey 429
 Cough as a symptom 103
 Crisis of fever, symptoms 76, pyrexial state 137 &c., pathology of 418
 Culture experiments 433

 DEAFNESS as a symptom 184
 Death-rate of spirillum fever 241, death-date 242, ditto in Europe 247; death, cause of, 243, mode of, 243
 Definition of spirillum fever 33
 Delirium as a complication, its varieties 176
 Dementia as a sequel 183
 Description, general, of spirillum fever 34
 Designation of the disease 34
 Desquamation as a symptom 198
 Destitution as a predisposing influence 371
 Diagnosis, general, of the spirillum fever 300
 Diagnosis, clinical, 309
 Diarrhœa as a symptom 96, as a complication 217
 Duodenum, lesions of, 281
 Dysentery as a complication 218
 Dysphagia 211
 Dyspnoea, acmal 206

 ENDOTHELIAL cells in the blood 346
 Enteric fever at Bombay 438
 Enteritis as a complication 215, its pathology 420

EPI

Epidemic at Bombay, origin of 25, concurrent epidemics elsewhere in India 27
 Epidemiology of spirillum fever 421
 Epigastric symptoms 108
 Epistaxis as a symptom 191
 Eruptions on the skin 194
 Etiology of spirillum fever 369
 Eye, lesions of, 192

FIBRILLATION of the blood 340
 Filaments free in the blood 346
 First apyretic interval 42, pyrexial state 143
 First relapse 43, pyrexial state 145
 Fomes as a means of contagion 409
 Fourth relapse 48, pyrexial state 156

GANGRENE, spontaneous, 198
 Gastric hæmorrhage as complication 213
 Germs of the spirillum 363
 Granule-cells in the blood 342
 Granules, free, in the blood 346

HÆMATEMESIS 213

Headache 85
 Heart, action of, 97; lesions of 208, 252
 Hepatic congestion and abscess as complications 220, lesions 263
 Hepatitis as a complication 220
 Hiccup 212
 History of the epidemic, in the Mofussil 1, at Bombay 14

ICTERIC fever 237
 Identification of spirillum fever 306
 Ileum, lesions of, 282
 Increscant febrile attacks 162
 Incubation-stages of spirillum fever 33, aspect of blood in 336, presence of spirillum in 354
 Infarcts of spleen 270
 Intercalated relapses 163
 Intestinal canal, lesions of, 279
 Invasion-attack of fever 34, pyrexial state 132, varieties of 160

JAUNDICE as a symptom 221
 Jejunum, lesions of, 282

KIDNIES, lesions of, 274

LARYNGITIS as a complication 199
 Latent relapses 165

REC

Liver, symptoms 106, lesions 263
 Lungs, respiration-symptoms 103, lesions 255
 Lymphatic system, lesions of, 285
 Lysis defervescence 81, pyrexial state 141, pathology 419

MANIA, mental hebetude, as complications 183
 Miliary eruption 93
 Modifications of spirillum fever 231
 Monkey, induced fever in, 429
 Mortality of spirillum fever 241

NAUSEA as a symptom 92
 Normal temperature and pulse of man 126
 Numbness and hyperæsthesia as complications 184

OCCUPATIONS of patients 310
 Odour from the skin 198
 Ophthalmia as a complication 192
 Overcrowding as a predisposing influence 374

PAINS as a symptom 86
 Pancreas, lesions of, 281
 Parotitis as a complication 210
 Pathology of spirillum fever 333
 Pericarditis as a complication 208
 Perturbatio critica 72, its pathology 418
 Petechiæ 197
 Pharyngitis as a complication 211
 Phthisis pulmonalis as a complication 207
 Plasma of the blood in spirillum fever 339
 Pleurisy as a complication 205
 Pneumonia as a complication 201
 Predisposing causes of spirillum fever 369
 Prodromata 69
 Prognosis of the disease 321
 Prophylaxis of the disease 325
 Protoplasm in the blood 345
 Pulse, normal 127, during disease 100
 Pyrexia, specific 125, compared with the pulse 156, compared with normal temperature 156, compared with pyrexia of inoculated monkey 157, pathology of 416

RACE in patients 370
 Rebounds of temperature 171
 Recognition of relapsing fever 300
 Rectum, lesions of, 284

REC

Recurrency of spirillum fever 413
 Red corpuscles of the blood 341
 Relapses defined 33, their number and proportion 128, varieties 162
 Relapsing fever, general history of, 28
 Remittent fever at Bombay 441
 Renal complications 225
 Repeated attacks of spirillum fever 168
 Residual fever 171
 Respiratory system, symptoms 103, lesions 255
 Rigor mortis 249

SEASON of the year as predisposing influence 372
 Second apyretic interval 45, temperature and pulse 152
 Second relapse 46, pyrexial state 153
 Secondary fever 171
 Sequelæ of spirillum fever 229
 Sex of patients 369
 Soil of fever localities 373
 Sphygmographic pulse-tracings 101
 Spirillum of the blood, description 348, its aspects at different stages of fever 354, its natural history 361, its range and relationship to disease 412
 Spirillum of saliva 362
 Spirillar infection, origin of, 421
 Spirochæte plicatilis 361
 Spleen, symptoms 111, complications 224; its lesions 269
 Spurious relapses 167

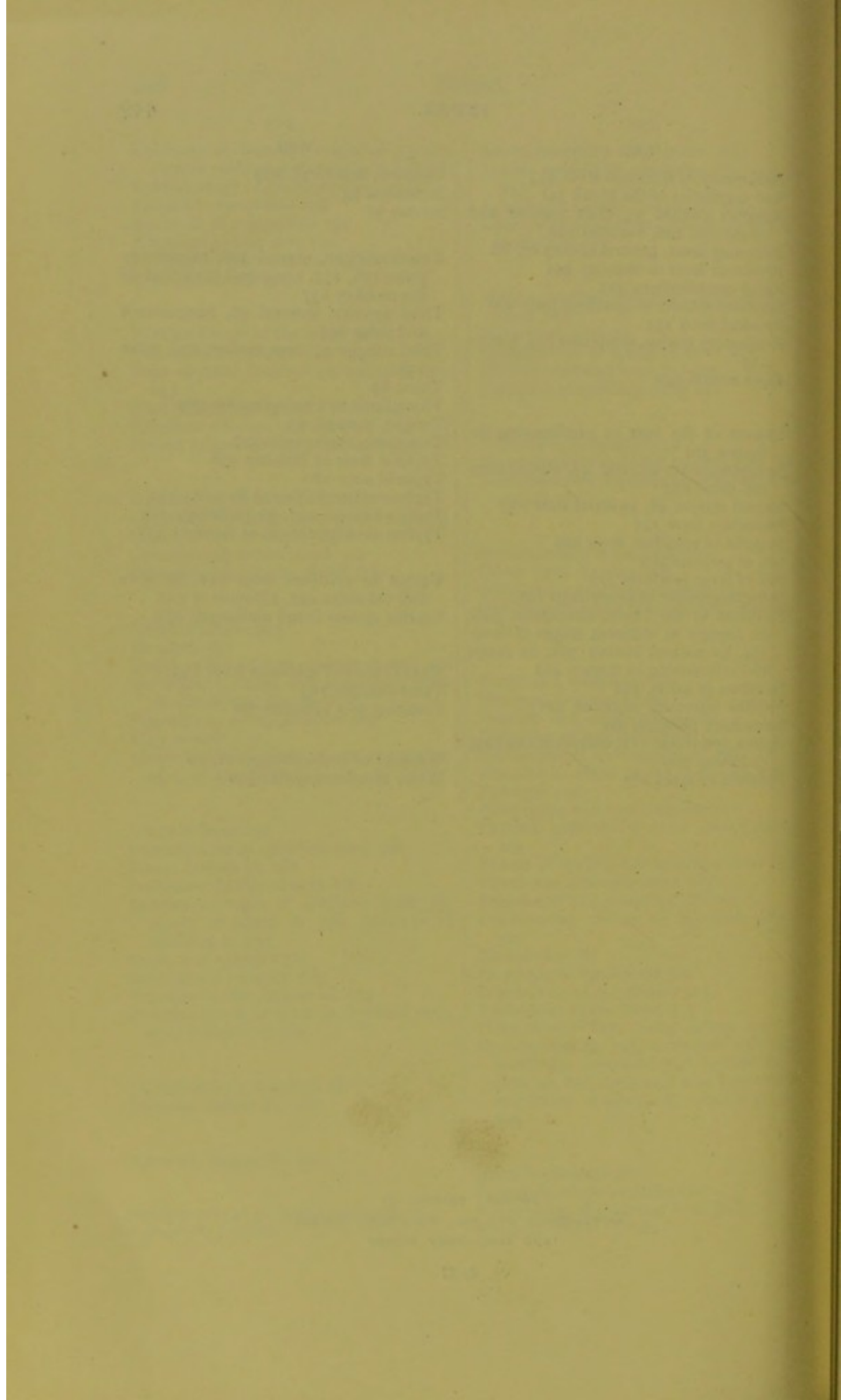
WHI

Stomach, lesions of, 279
 Sudamina 93
 Sweats 92
 TEMPERATURE, normal 126, relations to pulse 156, 157, compared with that of the monkey 157
 Third apyretic interval 46, temperature and pulse 155
 Third relapse 47, temperature and pulse 155
 Thirst 89
 Thrombosis as a complication 209
 Tongue, state of, 93
 Treatment, therapeutic 327
 Typhoid fever at Bombay 438
 Typhoid state 180
 Typho-remittent fever at Bombay 442
 Typhus biliosus 237, its pathology 419
 Typhus exanthematicus at Bombay 437

URINE in spirillum fever 118, its urea and chlorides 122, albumen in 124
 Uterine system found unchanged 279

VARIATIONS of spirillum fever 159
 Voice-changes 103
 Vomiting as a symptom 90

WEIGHT of body, changes in, 70
 White blood-corpuscles 342



WORKS BY THE SAME AUTHOR,

I.

The MICROSCOPIC STRUCTURE and MODE of
FORMATION of URINARY CALCULI. With 4 Plates.
8vo. 5s.

II.

On MYCETOMA ; or, the FUNGUS DISEASE of
INDIA. With 11 Coloured Plates, containing many Figures.
4to. 42s.

III.

MODERN INDIAN LEPROSY : being the Report of
a Tour in Kattiawar, 1876, with Addenda on Norwegian, Cretan,
and Syrian Leprosy. With Map and Plate. 8vo. 5s.

J. & A. CHURCHILL, New Burlington Street.

THE HISTORY OF THE

REIGN OF KING CHARLES THE FIRST

IN THE YEAR OF HIS REIGN

THE SECOND PART

OF THE HISTORY

Catalogue B]

*London, New Burlington Street
January, 1882*

SELECTION

FROM

J. & A. CHURCHILL'S GENERAL CATALOGUE

COMPRISING

ALL RECENT WORKS PUBLISHED BY THEM

ON THE

ART AND SCIENCE OF MEDICINE



N.B.—As far as possible, this List is arranged in the order in which medical study is usually pursued.

THE SCIENCE OF MEDICINE
AND THE ART OF NURSING

BY A. J. THORNTON

THE SCIENCE OF MEDICINE
AND THE ART OF NURSING

THE SCIENCE OF MEDICINE
AND THE ART OF NURSING

THE SCIENCE OF MEDICINE
AND THE ART OF NURSING



THE SCIENCE OF MEDICINE
AND THE ART OF NURSING

A SELECTION

FROM

J. & A. CHURCHILL'S GENERAL CATALOGUE,

COMPRISING

ALL RECENT WORKS PUBLISHED BY THEM ON THE
ART AND SCIENCE OF MEDICINE.

N.B.—J. & A. Churchill's Descriptive List of Works on Chemistry, Materia Medica, Pharmacy, Botany, Photography, Zoology, the Microscope, and other Branches of Science, can be had on application.

Practical Anatomy:

A Manual of Dissections. By CHRISTOPHER HEATH, Surgeon to University College Hospital. Fifth Edition. Crown 8vo, with 24 Coloured Plates and 269 Engravings, 15s.

Wilson's Anatomist's Vade-

Mecum. Tenth Edition. By GEORGE BUCHANAN, Professor of Clinical Surgery in the University of Glasgow; and HENRY E. CLARK, M.R.C.S., Lecturer on Anatomy at the Glasgow Royal Infirmary School of Medicine. Crown 8vo, with 450 Engravings (including 26 Coloured Plates), 18s.

Braune's Atlas of Topographi-

cal Anatomy, after Plane Sections of Frozen Bodies. Translated by EDWARD BELLAMY, Surgeon to, and Lecturer on Anatomy, &c., at, Charing Cross Hospital. Large Imp. 8vo, with 34 Photolithographic Plates and 46 Woodcuts, 40s.

An Atlas of Human Anatomy.

By RICKMAN J. GODLEE, M.S., F.R.C.S., Assistant Surgeon and Senior Demonstrator of Anatomy, University College Hospital. With 48 Imp. 4to Plates (112 figures), and a volume of Explanatory Text, 8vo, £4 14s. 6d.

Anatomy of the Joints of Man.

By HENRY MORRIS, Surgeon to, and Lecturer on Anatomy and Practical Surgery at, the Middlesex Hospital. 8vo, with 44 Lithographic Plates (several being coloured) and 13 Wood Engravings, 16s.

Surgical Anatomy:

A Series of Dissections, illustrating the Principal Regions of the Human Body. By JOSEPH MACLISE. Second Edition. 52 folio Plates and Text. Cloth, £3 12s.

Medical Anatomy.

By FRANCIS SIBSON, M.D., F.R.C.P., F.R.S. Imp. folio, with 21 Coloured Plates, cloth, 42s., half-morocco, 50s.

Manual of the Dissection of the

Human Body. By LUTHER HOLDEN, Consulting Surgeon to St. Bartholomew's and the Foundling Hospitals. Fourth Edition. 8vo, with 170 Engravings, 16s.

By the same Author.

Human Osteology:

Sixth Edition. 8vo, with 61 Lithographic Plates and 89 Engravings. 16s.

Also.

Landmarks, Medical and Surgi-

cal. Third Edition. 8vo, 3s. 6d.

The Student's Guide to Surgical

Anatomy: An Introduction to Operative Surgery. By EDWARD BELLAMY, F.R.C.S. and Member of the Board of Examiners. Fcap. 8vo, with 76 Engravings, 7s.

The Student's Guide to Human

Osteology. By WILLIAM WARWICK WAGSTAFFE, Assistant Surgeon to St. Thomas's Hospital. Fcap. 8vo, with 23 Plates and 66 Engravings, 10s. 6d.

The Anatomical Remembrancer; or, Complete Pocket Anatomist. Eighth Edition. 32mo, 3s. 6d.

Diagrams of the Nerves of the Human Body, exhibiting their Origin, Divisions, and Connections, with their Distribution to the Various Regions of the Cutaneous Surface, and to all the Muscles. By WILLIAM H. FLOWER, F.R.C.S., F.R.S., Hunterian Professor of Comparative Anatomy to the Royal College of Surgeons. Third Edition, with 6 Plates. Royal 4to, 12s.

Atlas of Pathological Anatomy. By Dr. LANCEREAUX. Translated by W. S. GREENFIELD, M.D., Professor of Pathology in the University of Edinburgh. Imp. 8vo, with 70 Coloured Plates, £5 5s.

A Manual of Pathological Anatomy. By C. HANDFIELD JONES, M.B., F.R.S.; and EDWARD H. SIEVEKING, M.D., F.R.C.P. Edited (with considerable enlargement) by J. F. PAYNE, M.D., F.R.C.P., Lecturer on General Pathology at St. Thomas's Hospital. Second Edition. Crown 8vo, with 195 Engravings, 16s.

Lectures on Pathological Anatomy. By SAMUEL WILKS, M.D., F.R.S., Physician to Guy's Hospital; and WALTER MOXON, M.D., Physician to Guy's Hospital. Second Edition. 8vo, with Plates, 18s.

Post-Mortem Examinations:

A Description and Explanation of the Method of performing them, with especial reference to Medico-Legal Practice. By Prof. VIRCHOW. Translated by Dr. T. P. SMITH. Second Edition. Fcap. 8vo, with 4 Plates, 3s. 6d.

Principles of Human Physiology. By W. B. CARPENTER, C.B., M.D., F.R.S. Ninth Edition. By HENRY POWER, M.B., F.R.C.S. 8vo, with 3 Steel Plates and 377 Wood Engravings, 31s. 6d.

A Treatise on Human Physiology. By JOHN C. DALTON, M.D., Professor in the College of Physicians and Surgeons, New York. Sixth Edition. Royal 8vo, with 316 Engravings, 20s.

Text-Book of Physiology.

By J. FULTON, M.D., Professor of Physiology, &c., in Trinity Medical College, Toronto. Second Edition. 8vo, with 152 Engravings, 15s.

Practical Histology.

By WILLIAM RUTHERFORD, M.D., F.R.S. Third Edition. Crown 8vo, with Engravings. (*In the Press.*)

Sanderson's Handbook for the Physiological Laboratory. By E. KLEIN, M.D., F.R.S.; J. BURDON-SANDERSON, M.D., F.R.S.; MICHAEL FOSTER, M.D., F.R.S.; and T. LAUDER BRUNTON, M.D., F.R.S. 8vo, with 123 Plates, 24s.

Histology and Histo-Chemistry of Man. By HEINRICH FREY, Professor of Medicine in Zurich. Translated by ARTHUR E. J. BARKER, Assistant-Surgeon to University College Hospital. 8vo, with 608 Engravings, 21s.

The Marriage of Near Kin,

Considered with respect to the Laws of Nations, Results of Experience, and the Teachings of Biology. By ALFRED H. HUTH. 8vo, 14s.

Medical Jurisprudence:

Its Principles and Practice. By ALFRED S. TAYLOR, M.D., F.R.C.P., F.R.S. Second Edition. 2 vols. 8vo, with 189 Engravings, £1 11s. 6d.

By the same Author.

A Manual of Medical Jurisprudence. Tenth Edition. Crown 8vo, with 55 Engravings, 14s.

Also.

Poisons,

In Relation to Medical Jurisprudence and Medicine. Third Edition. Crown 8vo, with 104 Engravings, 16s.

Lectures on Medical Jurisprudence. By FRANCIS OGSTON, M.D., Professor in the University of Aberdeen. Edited by FRANCIS OGSTON, JUN., M.D. 8vo, with 12 Copper Plates, 18s.

A Handy-Book of Forensic Medicine and Toxicology. By W. BATHURST WOODMAN, M.D., F.R.C.P., and C. MEYMOTT TIDY, M.D., F.C.S. 8vo, with 8 Lithographic Plates and 116 Engravings, 31s. 6d.

Microscopical Examination of Drinking Water. By JOHN D. MACDONALD, M.D., F.R.S., Assistant Professor in the Army Medical School. 8vo, with 24 Plates, 7s. 6d.

Sanitary Examinations

Of Water, Air, and Food. A Vade-Mecum for the Medical Officer of Health. By CORNELIUS B. FOX, M.D., F.R.C.P. Crown 8vo, with 94 Engravings, 12s. 6d.

Sanitary Assurance:

A Lecture at the London Institution. By Prof. F. DE CHAUMONT, F.R.S. With Short Addresses by J. E. ERICHSEN, F.R.S., Sir J. FAYRER, K.C.S.I., and R. BRUDENELL CARTER, F.R.C.S., &c. Royal 8vo, 1s.

A Manual of Practical Hygiene.

By E. A. PARKES, M.D., F.R.S. Fifth Edition. By F. DE CHAUMONT, M.D., F.R.S., Professor of Military Hygiene in the Army Medical School. 8vo, with 9 Plates and 112 Engravings, 18s.

Dangers to Health:

A Pictorial Guide to Domestic Sanitary Defects. By T. PRIDGIN TEALE, M.A., Surgeon to the Leeds General Infirmary. Third Edition. 8vo, with 70 Lithograph Plates (mostly coloured). 10s.

A Handbook of Hygiene and

Sanitary Science. By GEO. WILSON, M.A., M.D., Medical Officer of Health for Mid-Warwickshire. Fourth Edition. Post 8vo, with Engravings, 10s. 6d.

Also.

Healthy Life and Healthy

Dwellings: A Guide to Personal and Domestic Hygiene. Fcap. 8vo, 5s.

Contributions to Military and

State Medicine. By JOHN MARTIN, L.R.C.S.E., Surgeon Army Medical Department. 8vo, 10s. 6d.

Pay Hospitals and Paying

Wards throughout the World. By HENRY C. BURDETT, late Secretary to the Seamen's Hospital Society. 8vo, 7s.

By the same Author.

Cottage Hospitals — General,

Fever, and Convalescent: Their Progress, Management, and Work. Second Edition, with many Plans and Illustrations. Crown 8vo, 14s.

Dress: Its Sanitary Aspect.

A Paper read before the Brighton Social Union, Jan. 30, 1880. By BERNARD ROTH, F.R.C.S. 8vo, with 8 Plates, 2s.

Manual of Anthropometry:

A Guide to the Measurement of the Human Body, containing an Anthropometrical Chart and Register, a Systematic Table of Measurements, &c. By CHARLES ROBERTS, F.R.C.S. 8vo, with numerous Illustrations and Tables, 6s. 6d.

Madness:

In its Medical, Legal, and Social Aspects. Lectures by EDGAR SHEPPARD, M.D., M.R.C.P., Professor of Psychological Medicine in King's College. 8vo, 6s. 6d.

Idiocy and Imbecility.

By WILLIAM W. IRELAND, M.D., Medical Superintendent of the Scottish National Institution for the Education of Imbecile Children at Larbert, Stirlingshire. 8vo, with Engravings, 14s.

A Manual of Psychological

Medicine: With an Appendix of Cases. By JOHN C. BUCKNILL, M.D., F.R.S., and D. HACK TUKE, M.D., F.R.C.P. Fourth Edition. 8vo, with 12 Plates (30 Figures) and Engravings, 25s.

The Student's Guide to the Practice of Midwifery.

By D. LLOYD ROBERTS, M.D., F.R.C.P., Physician to St. Mary's Hospital, Manchester. Second Edition. Fcap. 8vo, with 111 Engravings, 7s.

Handbook of Midwifery for Mid-

wives: from the Official Handbook for Prussian Midwives. By J. E. BURTON, L.R.C.P. Lond., Senior Assistant Medical Officer, Ladies' Charity, &c., Liverpool. With Engravings. Fcap. 8vo, 6s.

Lectures on Obstetric Opera-

tions: Including the Treatment of Hæmorrhage, and forming a Guide to the Management of Difficult Labour. By ROBERT BARNES, M.D., F.R.C.P., Obstetric Physician to St. George's Hospital. Third Edition. 8vo, with 124 Engravings, 18s.

By the same Author.

A Clinical History of Medical and Surgical Diseases of

Women. Second Edition. 8vo, with 181 Engravings, 28s.

West on the Diseases of

Women. Fourth Edition, revised and in part re-written by the Author, with numerous Additions by J. MATTHEWS DUNCAN, M.D., Obstetric Physician to St. Bartholomew's Hospital. 8vo, 16s.

Observations on the Cæsarean

Section, Craniotomy, and on other Obstetric Operations, with Cases. By THOMAS RADFORD, M.D., late Honorary Consulting Physician, St. Mary's Hospital, Manchester. Second Edition, with Plates. 8vo, 10s.

Clinical Lectures on Diseases

of Women: Delivered in St. Bartholomew's Hospital, by J. MATTHEWS DUNCAN, M.D., F.R.S.E. 8vo, 8s.

By the same Author.

Papers on the Female Perineum, &c.

8vo, 6s.

The Principles and Practice of

Gynæcology. By THOMAS ADDIS EMMET, M.D., Surgeon to the Woman's Hospital, New York. Second Edition. Royal 8vo, with 133 Engravings, 24s.

The Student's Guide to the Diseases of Women. By ALFRED L. GALABIN, M.D., F.R.C.P., Assistant Obstetric Physician to Guy's Hospital. Second Edition. Fcap. 8vo, with 70 Engravings, 7s. 6d.

Notes on the Diseases of Women. Specially designed for Students preparing for Examination. By J. J. REYNOLDS, M.R.C.S. Fcap. 8vo, 2s. 6d.

By the same Author.

Notes on Midwifery: Specially designed to assist the Student in preparing for Examination. Fcap. 8vo, 4s.

Practical Gynæcology: A Handbook of the Diseases of Women. By HEYWOOD SMITH, M.D. Oxon., Physician to the Hospital for Women and to the British Lying-in Hospital. Second Edition. Crown 8vo, with Engravings. (*In the Press.*)

By the same Author.

Dysmenorrhea, its Pathology and Treatment. Crown 8vo, with Engravings, 4s. 6d.

Obstetric Aphorisms: For the Use of Students commencing Midwifery Practice. By JOSEPH G. SWAYNE, M.D. Seventh Edition. Fcap. 8vo, with Engravings, 3s. 6d.

Obstetric Medicine and Surgery: Their Principles and Practice. By F. H. RAMSBOTHAM, M.D., F.R.C.P. Fifth Edition. 8vo, with 120 Plates, 22s.

A Complete Handbook of Obstetric Surgery. Giving Short Rules of Practice in every Emergency. By CHARLES CLAY, late Senior Surgeon to St. Mary's Hospital, Manchester. Third Edition. Fcap. 8vo, with 91 Engravings, 6s. 6d.

Schroeder's Manual of Midwifery, including the Pathology of Pregnancy and the Puerperal State. Translated by CHARLES H. CARTER, B.A., M.D. 8vo, with Engravings, 12s. 6d.

Influence of Posture on Women in Gynecic and Obstetric Practice. By J. H. AVELING, M.D., Physician to the Chelsea Hospital for Women. 8vo, 6s.

A Handbook of Uterine Therapeutics, and of Diseases of Women. By E. J. TILT, M.D., M.R.C.P. Fourth Edition. Post 8vo, 10s.

By the same Author.

The Change of Life In Health and Disease: a Practical Treatise on the Nervous and other Affections incidental to Women at the Decline of Life. Third Edition. 8vo, 10s. 6d.

Ovarian and Uterine Tumours: Their Diagnosis and Surgical Treatment. By T. SPENCER WELLS, F.R.C.S., Consulting Surgeon to the Samaritan Hospital. 8vo, with Engravings. (*Nearly ready.*)

Rupture of the Female Perineum: Its Treatment, immediate and remote. By GEORGE G. BANTOCK, M.D., Surgeon to the Samaritan Hospital. 8vo, with 2 Plates, 3s. 6d.

Chronic Disease of the Heart: Its Bearings upon Pregnancy, Parturition, and Childbed. By ANGUS MACDONALD, M.D., F.R.S.E., Physician to the Edinburgh Royal Infirmary. 8vo, with Engravings, 8s. 6d.

The Female Pelvic Organs, Their Surgery, Surgical Pathology, and Surgical Anatomy, in a Series of Coloured Plates taken from Nature: with Commentaries, Notes, and Cases. By HENRY SAVAGE, Consulting Officer of the Samaritan Free Hospital. Fifth Edition. 4to. (*In the Press.*)

Lectures on Diseases of the Nervous System, especially in Women. By S. WEIR MITCHELL, M.D., Physician to the Philadelphia Infirmary for Diseases of the Nervous System. With 5 Plates. Post 8vo, 8s.

A Treatise on the Diseases of Children. For Practitioners and Students. By WILLIAM H. DAY, M.D., Physician to the Samaritan Hospital for Women and Children. Crown 8vo, 12s. 6d.

The Wasting Diseases of Children. By EUSTACE SMITH, M.D., Physician to the King of the Belgians, Physician to the East London Hospital for Children. Third Edition. Post 8vo, 8s. 6d.

By the same Author.

Clinical Studies of Disease in Children. Second Edition. Post 8vo. (*In the Press.*)

Infant Feeding and its Influence on Life; or, the Causes and Prevention of Infant Mortality. By C. H. F. ROUTH, M.D., Senior Physician to the Samaritan Hospital. Third Edition. Fcap. 8vo, 7s. 6d.

A Practical Manual of the Diseases of Children. With a Formulary. By EDWARD ELLIS, M.D. Fourth Edition. Crown 8vo, 10s.

By the same Author.

A Manual of what every Mother should know. Fcap. 8vo, 1s. 6d.

Lectures on Nursing. By W. R. SMITH, M.B., Physician to the Cheltenham Dispensary. Third Edition. Post 8vo, with Engravings. (*In the Press.*)

A Manual for Hospital Nurses

and others engaged in Attending on the Sick. By EDWARD J. DOMVILLE, Surgeon to the Exeter Lying-in Charity. Fourth Edition. Crown 8vo, 2s. 6d.

The Nurse's Companion :

A Manual of General and Monthly Nursing. By CHARLES J. CULLINGWORTH, Surgeon to St. Mary's Hospital, Manchester. Second Edition. Fcap. 8vo. (*In the Press.*)

Handbook for Nurses for the

Sick. By ZEPHERINA P. VEITCH. Second Edition. Crown 8vo, 3s. 6d.

Notes on Fever Nursing.

By J. W. ALLAN, M.B., Superintendent and Physician, City of Glasgow Fever Hospital. Crown 8vo, with Engravings, 2s. 6d.

Medicinal Plants :

Being descriptions, with original figures, of the Principal Plants employed in Medicine, and an account of their Properties and Uses. By Professor BENTLEY and Dr. H. TRIMEN. In 4 volumes, large 8vo, with 306 Coloured Plates, bound in Half Morocco, Gilt Edges. £11 11s.

Royle's Manual of Materia

Medica and Therapeutics. Sixth Edition, by JOHN HARLEY, M.D., Physician to St. Thomas's Hospital. Crown 8vo, with 139 Engravings, 15s.

A Manual of Practical Thera-

peutics. By E. J. WARING, C.B., M.D., F.R.C.P. Lond. Third Edition. Fcap. 8vo, 12s. 6d.

The Student's Guide to Materia

Medica. By JOHN C. THOROWGOOD, M.D., F.R.C.P. Fcap. 8vo, 6s. 6d.

Materia Medica and Therapeu-

tics : Vegetable Kingdom. By CHARLES D. F. PHILLIPS, M.D., late Lecturer on Materia Medica and Therapeutics to Westminster Hospital. 8vo, 15s.

Bazaar Medicines of India,

And Common Medical Plants : With Full Index of Diseases, indicating their Treatment by these and other Agents procurable throughout India, &c. By E. J. WARING, C.B., M.D., F.R.C.P. Third Edition. Fcap. 8vo, 5s.

Indian Notes.

The Voyage out; Travelling in India; Upper India; Stations; The Hills; Mineral Waters; Herbs and Simples. By F. R. HOGG, M.D., Surgeon-Major. Crown 8vo, 5s.

The National Dispensatory :

Containing the Natural History, Chemistry, Pharmacy, Actions and Uses of Medicines, including those recognized in the Pharmacopœias of the United States and Great Britain and Germany, with numerous references to the French Codex. By ALFRED STILLÉ, M.D., LL.D., and JOHN M. MAISCH, Ph.D. Second Edition. 8vo, with 239 Engravings, 34s.

Binz's Elements of Thera-

peutics : A Clinical Guide to the Action of Drugs. Translated by E. I. SPARKS, M.B., F.R.C.P. Crown 8vo, 8s. 6d.

A Companion to the British

Pharmacopœia. By PETER SQUIRE, F.L.S., assisted by his sons, P. W. and A. H. SQUIRE. Thirteenth Edition. 8vo. (*In the Press.*)

By the same Author.

The Pharmacopœias of the Lon-

don Hospitals, arranged in Groups for Easy Reference and Comparison. Fourth Edition. 18mo, 6s.

A New System of Medicine ;

Entitled *Reconisant Medicine*, or the State of the Sick. By B. BOSE, M.D., Indian Medical Service. 8vo, 10s. 6d.

By the same Author.

Principles of Rational Thera-

peutics. Commenced as an Inquiry into the Relative Value of Quinine and Arsenic in Ague. Crown 8vo, 4s. 6d.

Diseases of Tropical Climates,

And their Treatment : with Hints for the Preservation of Health in the Tropics. By JAMES A. HORTON, M.D., Surgeon-Major. Second Edition. Post 8vo, 12s. 6d.

Tropical Dysentery and Chronic

Diarrhœa—Liver Abscess—Malarial Cachexia—Insolation—with other forms of Tropical Diseases, &c. By Sir JOSEPH FAYRER, K.C.S.I., M.D., 8vo., 15s.

By the same Author.

Clinical and Pathological Obser-

vations in India. 8vo, with Engravings, 20s.

Endemic Diseases of Tropical

Climates, with their Treatment. By JOHN SULLIVAN, M.D. Post 8vo, 6s.

Family Medicine for India :

By WILLIAM J. MOORE, M.D., Honorary Surgeon to the Viceroy of India. Published under the Authority of the Government of India. Third Edition. Post 8vo, with 66 Engravings, 12s.

By the same Author.

Health Resorts for Tropical

Invalids, in India, at Home, and Abroad. Post 8vo, 5s.

The Elements of Indian Hygiene.

Intended to guide the Public in acquiring some knowledge in the all-important subject of the Preservation of Health and Prevention of Sickness. By JOHN C. LUCAS, F.R.C.S., H.M.'s Indian Medical Service. Crown 8vo, with Map of India, &c., 5s.

The Student's Guide to the Practice of Medicine.

By MATTHEW CHARTERIS, M.D., Professor of Materia Medica in the University of Glasgow. Third Edition. Fcap. 8vo, with Engravings on Copper and Wood, 7s.

Hooper's Physicians' Vade-Mecum.

A Manual of the Principles and Practice of Physic. Tenth Edition. By W. A. GUY, F.R.C.P., F.R.S., and J. HARLEY, M.D., F.R.C.P. With 118 Engravings. Fcap. 8vo, 12s. 6d.

Clinical Studies:

Illustrated by Cases observed in Hospital and Private Practice. By Sir J. ROSE CORMACK, M.D., F.R.S.E., Physician to the Hertford British Hospital of Paris. Two vols. Post 8vo, 20s.

Clinical Medicine:

Lectures and Essays. By BALTHAZAR FOSTER, M.D., F.R.C.P. Lond., Professor of Medicine in Queen's College, Birmingham. 8vo, 10s. 6d.

Clinical Lectures and Cases,

with Commentaries. By HENRY THOMPSON, M.D., F.R.C.P., Consulting Physician to Middlesex Hospital. With Temperature Charts. 8vo, 7s. 6d.

Clinical Medicine:

A Systematic Treatise on the Diagnosis and Treatment of Disease. By AUSTIN FLINT, M.D., Professor of Medicine in the Bellevue Hospital Medical College. 8vo, 20s.

By the same Author.

Phthisis:

In a series of Clinical Studies. 8vo, 16s.

Transfusion of Human Blood:

With Table of 50 cases. By Dr. ROUSSEL, of Geneva. With a Preface by Sir JAMES PAGET, Bart. Crown 8vo, 2s. 6d.

The Spectroscope in Medicine.

By CHARLES A. MACMUNN, B.A., M.D. 8vo, with 3 Chromo-lithographic Plates of Physiological and Pathological Spectra, and 13 Engravings, 9s.

The Microscope in Medicine.

By LIONEL S. BEALE, M.B., F.R.S., Physician to King's College Hospital. Fourth Edition. 8vo, with 86 Plates, 21s.

Also.

On Slight Ailments:

Their Nature and Treatment. 8vo, 5s.

A Manual of Medical Diagnosis.

By A. W. BARCLAY, M.D., F.R.C.P., Physician to St. George's Hospital. Third Edition. Fcap. 8vo, 10s. 6d.

The Student's Guide to Medical Diagnosis.

By SAMUEL FENWICK, M.D., F.R.C.P., Physician to the London Hospital. Fifth Edition. Fcap. 8vo, with 111 Engravings, 7s.

By the same Author.

The Student's Outlines of Medical Treatment.

Second Edition. Fcap. 8vo, 7s.

Also.

On Chronic Atrophy of the Stomach, and on the Nervous Affections of the Digestive Organs.

8vo, 8s.

The Student's Guide to Medical Case-Taking.

By FRANCIS WARNER, M.D., Assistant Physician to the London Hospital. Fcap. 8vo, 5s.

Asthma:

Its Pathology and Treatment. By J. B. BERKART, M.D., Assistant-Physician to the City of London Hospital for Diseases of the Chest. 8vo, 7s. 6d.

Notes on Asthma:

Its Forms and Treatment. By JOHN C. THOROWGOOD, M.D., Physician to the Hospital for Diseases of the Chest, Victoria Park. Third Edition. Crown 8vo, 4s. 6d.

Observations on the Result of

Treatment of nearly One Hundred Cases of Asthma. By T. L. PRIDHAM, M.R.C.S. Third Edition. 8vo, 2s. 6d.

Diseases of the Chest:

Contributions to their Clinical History, Pathology, and Treatment. By A. T. HOUGHTON WATERS, M.D., Physician to the Liverpool Royal Infirmary. Second Edition. 8vo, with Plates, 15s.

Winter Cough:

(Catarrh, Bronchitis, Emphysema, Asthma). By HORACE DOBELL, M.D., Consulting Physician to the Royal Hospital for Diseases of the Chest. Third Edition. 8vo, with Coloured Plates, 10s. 6d.

By the same Author.

Loss of Weight, Blood-Spitting, and Lung Disease.

Second Edition, to which is added Part VI., "On the Functions and Diseases of the Liver." 8vo, with Chromo-lithograph, 10s. 6d.

Also.

The Mont Dore Cure, and the Proper Way to Use it.

8vo, 7s. 6d.

Relapse of Typhoid Fever, especially with reference to the Temperature. By J. PEARSON IRVINE, M.D., F.R.C.P., late Assistant Physician to Charing Cross Hospital. 8vo, with Engravings, 6s.

Croonian Lectures on Some Points in the Pathology and Treatment of Typhoid Fever. By WILLIAM CAYLEY, M.D., F.R.C.P., Physician to the Middlesex and the London Fever Hospitals. Crown 8vo, 4s. 6d.

Manual of the Physical Diagnosis of Diseases of the Heart, including the use of the Sphygmograph and Cardiograph. By ARTHUR E. SANSOM, M.D., F.R.C.P., Assistant-Physician to the London Hospital. Third Edition. Fcap. 8vo, with 48 Engravings, 7s. 6d.

By the same Author.

The Antiseptic System in Medicine and Surgery: A Treatise on Carbolic Acid and its Compounds, etc. With 9 Plates (42 Figures), 8vo, 10s. 6d.

On Diseases of the Heart.

By THOS. B. PEACOCK, M.D., F.R.C.P.
(1) Malformations. 8vo, 10s. (2) Causes and Effects of Valvular Disease. 8vo, 5s.
(3) Prognosis in Valvular Disease. 8vo, 3s. 6d.

Medical Ophthalmoscopy:

A Manual and Atlas. By WILLIAM R. GOWERS, M.D., F.R.C.P., Assistant Professor of Clinical Medicine in University College, and Senior Assistant-Physician to the Hospital. Second Edition, with Coloured Autotype and Lithographic Plates and Woodcuts. 8vo., 18s.

By the same Author.

Epilepsy, and other Chronic Convulsive Diseases: Their Causes, Symptoms, and Treatment. 8vo, 10s. 6d.

Also.

Pseudo-Hypertrophic Muscular Paralysis: A Clinical Lecture. 8vo, with Engravings and Plate, 3s. 6d.

Also.

The Diagnosis of Diseases of the Spinal Cord. Second Edition. 8vo, with Coloured Plate and Engravings, 4s. 6d.

The Sympathetic System of Nerves: Their Physiology and Pathology. By Professor EULENBURG and Dr. P. GUTTMANN. Translated by A. NAPIER, M.D., F.F.P.S. 8vo, 5s. 6d.

Studies on Functional Nervous Disorders. By C. HANDFIELD JONES, M.B., F.R.S., Physician to St. Mary's Hospital. Second Edition. 8vo, 18s.

Diseases of the Stomach:

The Varieties of Dyspepsia, their Diagnosis and Treatment. By S. O. HABERSHON, M.D., F.R.C.P., late Senior Physician to Guy's Hospital. Third Edition. Crown 8vo, 5s.

By the same Author.

Pathology of the Pneumo-gastric Nerve, being the Lumleian Lectures for 1876. Post 8vo, 3s. 6d.

Also.

Diseases of the Abdomen,

Comprising those of the Stomach and other parts of the Alimentary Canal, Oesophagus, Cæcum, Intestines, and Peritoneum. Third Edition. 8vo, with 5 Plates, 21s.

Gout, Rheumatism,

And the Allied Affections; with a Chapter on Longevity and the Causes Antagonistic to it. By PETER HOOD, M.D. Second Edition. Crown 8vo, 10s. 6d.

Notes on Rheumatism.

By JULIUS POLLOCK, M.D., F.R.C.P., Senior Physician to the Charing Cross Hospital. Second Edition. Fcap. 8vo, with Engravings, 3s. 6d.

A Treatise on the Diseases of the Nervous System. By JAMES ROSS, M.D., Assistant-Physician to the Manchester Royal Infirmary. Two vols., 8vo, with Lithographs, Photographs, and 280 Wood Engravings. 42s.

Lectures on Diseases of the Nervous System. By SAMUEL WILKS, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital. Second Edition. 8vo.

(In the Press.)

Nervous Diseases:

Their Description and Treatment. By ALLEN McLANE HAMILTON, M.D., Physician at the Epileptic and Paralytic Hospital, Blackwell's Island, New York. Second Edition. Royal 8vo, with Illustrations. *(Just ready.)*

Headaches:

Their Nature, Causes, and Treatment. By WILLIAM H. DAY, M.D., Physician to the Samaritan Hospital for Women and Children. Third Edition. Crown 8vo, with Engravings, 6s. 6d.

Fits:

Diagnosis and Immediate Treatment of Cases of Insensibility and Convulsions. By JOHN H. WATERS, M.D., K.C., St.G.C., Surgeon to the C Division of Metropolitan Police. Crown 8vo, bound in leather, 4s.

On Megrim, Sick Headache and some Allied Disorders: a Contribution to the Pathology of Nerve Storms. By E. LIVEING, M.D., F.R.C.P. 8vo, 15s.

Nutrition in Health and Disease: A Contribution to Hygiene and to Clinical Medicine. By HENRY BENNET, M.D. Third (Library) Edition. 8vo, 7s. Cheap Edition. Fcap. 8vo, 2s. 6d.

Food and Dietetics,

Physiologically and Therapeutically Considered. By F. W. PAVY, M.D., F.R.S., Physician to Guy's Hospital. Second Edition. 8vo, 15s.

By the same Author.

Croonian Lectures on Certain Points connected with Diabetes. 8vo, 4s. 6d.

Imperfect Digestion:

Its Causes and Treatment. By A. LEARED, M.D., Sixth Edition. Fcap. 8vo, 4s. 6d.

Indigestion:

What it is; what it leads to; and a New Method of Treating it. By JOHN B. GILL, M.D., formerly Surgeon to the Dover Hospital, &c. Second Edition. Fcap. 8vo, 4s. 6d.

The Climate of the Undercliff, Isle of Wight, as deduced from forty years' consecutive Meteorological Observations. By J. L. WHITEHEAD, M.D. Royal 8vo, 5s.

The Riviera:

Sketches of the Health-Resorts of the North Mediterranean Coast of France and Italy, from Hyères to Spezia; with Chapters on the General Meteorology of the District, its Medical Aspect and Value, &c. By EDWARD I. SPARKS, M.B., F.R.C.P. Crown 8vo, 8s. 6d.

Winter and Spring

On the Shores of the Mediterranean. By HENRY BENNET, M.D. Fifth Edition. Post 8vo, with numerous Plates, Maps, and Engravings, 12s. 6d.

By the same Author.

Treatment of Pulmonary Consumption by Hygiene, Climate, and Medicine. Third Edition. 8vo, 7s. 6d.

The Ocean as a Health-Resort:

A Practical Handbook of the Sea, for the use of Tourists and Health-Seekers. By WILLIAM S. WILSON, L.R.C.P., Second Edition, with Chart of Ocean Routes, &c. Crown 8vo, 7s. 6d.

Davos Platz, and the Effects of High Altitude on Phthisis. By ALFRED WISE, M.D. Fcap. 8vo, 2s. 6d.

Principal Health-Resorts

Of Europe and Africa, and their Use in the Treatment of Chronic Diseases. By THOMAS MORE MADDEN, M.D., M.R.I.A. 8vo, 10s.

Handbook of Medical and Surgical Electricity. By HERBERT TIBBITS, M.D., F.R.C.P.E., Senior Physician to the West London Hospital for Paralysis and Epilepsy. Second Edition. 8vo, with 95 Engravings, 9s.

By the same Author.

A Map of Ziemssen's Motor Points of the Human Body: A Guide to Localised Electrification. Mounted on Rollers, 35 x 21. With 20 Illustrations, 5s.

Lectures on the Clinical Uses of Electricity. By J. RUSSELL REYNOLDS, M.D., F.R.S., Physician to University College Hospital. Second Edition. Post 8vo, 3s. 6d.

A System of Practical Surgery.

By Sir WILLIAM FERGUSSON, Bart., F.R.S. Fifth Edition. 8vo, with 463 Engravings, 21s.

Surgical Emergencies:

Together with the Emergencies Attendant on Parturition and the Treatment of Poisoning. By PAUL SWAIN, F.R.C.S., Surgeon to the Royal Albert Hospital, Devonport. Third Edition. Crown 8vo, with 117 Engravings, 5s.

A Course of Operative Surgery.

By CHRISTOPHER HEATH, Surgeon to University College Hospital. With 20 Plates drawn from Nature by M. LÉVEILLÉ, and coloured by hand under his direction. Large 8vo, 40s.

By the same Author.

The Student's Guide to Surgical Diagnosis. Fcap. 8vo, 6s. 6d.

Also.

Manual of Minor Surgery and Bandaging. For the use of House Surgeons, Dressers, and Junior Practitioners. Sixth Edition. Fcap. 8vo, with 115 Engravings, 5s. 6d.

Also.

Injuries and Diseases of the Jaws. Second Edition. 8vo, with 164 Engravings, 12s.

Outlines of Surgery and Surgical Pathology. By F. LE GROS CLARK, F.R.S., assisted by W. W. WAGSTAFFE, F.R.C.S. Second Edition. 8vo, 10s. 6d.

The Practice of Surgery :

A Manual. By THOMAS BRYANT, Surgeon to Guy's Hospital. Third Edition. Two vols. Crown 8vo, with 672 Engravings (many being coloured), 28s.

The Surgeon's Vade-Mecum :

A Manual of Modern Surgery. By ROBERT DRUITT, F.R.C.S. Eleventh Edition. Fcap. 8vo, with 369 Engravings, 14s.

Illustrations of Clinical Surgery.

By JONATHAN HUTCHINSON, Senior Surgeon to the London Hospital. In occasional fasciculi. I. to XIV., 6s. 6d. each. Fasciculi I. to X. bound, with Appendix and Index, £3 10s.

The Principles and Practice

of Surgery. By WILLIAM PIRRIE, F.R.S.E., Professor of Surgery in the University of Aberdeen. Third Edition. 8vo, with 490 Engravings, 28s.

Surgical Enquiries :

Including the Hastings Essay on Shock, the Treatment of Inflammations, and numerous Clinical Lectures. By FURNEAUX JORDAN, F.R.C.S., Professor of Surgery, Queen's College, Birmingham. Second Edition, with numerous Plates. Royal 8vo, 12s. 6d.

Treatment of Wounds :

Clinical Lectures. By SAMPSON GAMGEE, F.R.S.E., Surgeon to the Queen's Hospital, Birmingham. Crown 8vo, with Engravings, 5s.

By the same Author.

Fractures of the Limbs,

And their Treatment. 8vo, with Plates, 10s. 6d.

On Dislocations and Fractures.

By JOSEPH MACLISE, F.R.C.S. Uniform with "Surgical Anatomy." 36 folio Plates and Text. Cloth, £2 10s.

Lectures on Diseases of Bones

and Joints. By CHARLES MACNAMARA, F.R.C.S., Surgeon to, and Lecturer on Surgery at, Westminster Hospital. Crown 8vo, with Engravings, 10s. 6d.

Clubfoot :

Its Causes, Pathology, and Treatment. By WM. ADAMS, F.R.C.S., Surgeon to the Great Northern Hospital. Second Edition. 8vo, with 106 Engravings and 6 Lithographic Plates, 15s.

By the same Author.

On Contraction of the Fingers,

and its Treatment by Subcutaneous Operation; and on Obliteration of Depressed Cicatrices, by the same Method. 8vo, with 30 Engravings, 4s. 6d.

Orthopædic Surgery,

And Diseases of the Joints. By L. A. SAYRE, M.D., Professor of Orthopædic Surgery in Bellevue Hospital Medical College. 8vo, with 274 Engravings, 20s.

Osteotomy :

With an Enquiry into the Etiology and Pathology of Knock-knee, Bow-leg, and other Osseous Deformities of the Lower Limbs. By WILLIAM MACEWEN, M.D., Surgeon and Lecturer on Clinical Surgery to the Glasgow Royal Infirmary. 8vo, with 51 Engravings, 7s. 6d.

Lectures on Orthopædic Sur-

gery. By BERNARD E. BRODHURST, F.R.C.S., Surgeon to the Royal Orthopædic Hospital. Second Edition. 8vo, with Engravings, 12s. 6d.

By the same Author.

On Anchylosis, and the Treat-

ment for the Removal of Deformity and the Restoration of Mobility in Various Joints. Fourth Edition. 8vo, with Engravings, 5s.

Orthopraxy :

The Mechanical Treatment of Deformities, Debilities, and Deficiencies of the Human Frame. By H. HEATHER BIGG, Assoc. Inst. C.E. Third Edition. 8vo, with 319 Engravings, 15s.

The Orthopragms of the Spine :

An Essay on the Curative Mechanisms applicable to Spinal Curvature, etc. By ROBERT HEATHER BIGG, Assoc. Inst. C.E. 8vo, with Engravings, 5s.

A Manual of the Principles and

Practice of Ophthalmic Medicine and Surgery. By T. WHARTON JONES, F.R.C.S., F.R.S. Third Edition. Fcap. 8vo, with 9 Coloured Plates and 173 Engravings, 12s. 6d.

A Manual of Diseases of the

Eye. By C. MACNAMARA, F.R.C.S., Surgeon to Westminster Hospital. Third Edition. Fcap. 8vo, with Coloured Plates and Engravings, 12s. 6d.

On Diseases and Injuries of the

Eye: A Course of Systematic and Clinical Lectures to Students and Medical Practitioners. By J. R. WOLFE, M.D., F.R.C.S.E., Senior Surgeon to the Glasgow Ophthalmic Institution; Lecturer on Ophthalmic Medicine and Surgery in Anderson's College. With 10 Coloured Plates and 157 Wood Engravings. 8vo, £1 1s.

Hintson Ophthalmic Out-Patient

Practice. By CHARLES HIGGINS, Ophthalmic Assistant-Surgeon to, and Lecturer on Ophthalmology at, Guy's Hospital. Second Edition. Fcap. 8vo, 3s.

Liebreich's Atlas of Ophthal-

moscopy: Composed of 12 Chromolithographic Plates (containing 59 Figures). The Text translated by H. ROSBOROUGH SWANZY, M.B. Second Edition. 4to, 30s.

The Student's Guide to Diseases of the Eye. By EDWARD NETTLESHIP, F.R.C.S., Ophthalmic Surgeon to St. Thomas's Hospital. Second Edition. Fcap. 8vo, with Engravings, (*In the Press.*)

Glaucoma :

Its Causes, Symptoms, Pathology, and Treatment. By PRIESTLEY SMITH, M.R.C.S., Ophthalmic Surgeon to the Queen's Hospital, Birmingham. 8vo, with Lithographic Plates, 10s. 6d.

A Manual of Ophthalmoscopy for the use of Students. By DR. DAGUENET. Translated by C. S. JEAFFRESON, Surgeon to the Newcastle-on-Tyne Eye Infirmary. With Engravings. Fcap. 8vo, 5s.

Essays in Ophthalmology.

By GEORGE E. WALKER, F.R.C.S., Surgeon to St. Paul's Eye and Ear Hospital, &c., Liverpool. Post 8vo, 6s.

Hare-Lip and Cleft Palate.

By FRANCIS MASON, F.R.C.S., Surgeon to, and Lecturer on Practical Surgery at, St. Thomas's Hospital. 8vo, with 66 Engravings, 6s.

By the same Author.

The Surgery of the Face.

8vo, with 100 Engravings, 7s. 6d.

A Practical Treatise on Aural Surgery. By H. MACNAUGHTON JONES, M.D., Professor of the Queen's University in Ireland, Surgeon to the Cork Ophthalmic and Aural Hospital. Second Edition. Crown 8vo, with 63 Engravings, 8s. 6d.

By the same Author.

Atlas of Diseases of the Membrana Tympani. In Coloured Plates, containing 62 Figures, with Text. Crown 4to, 21s.

Diseases and Injuries of the Ear. By WILLIAM B. DALBY, F.R.C.S., Aural Surgeon to, and Lecturer on Surgery at, St. George's Hospital. Second Edition. Fcap. 8vo, with Engravings, 6s. 6d.

Throat Diseases,

And the Use of the Laryngoscope: A Handbook for Practitioners and Senior Students. By W. DOUGLAS HEMMING, F.R.C.S.E. With Engravings. Fcap. 8vo, 2s. 6d.

Lectures on Syphilis of the Larynx (Lesions of the Secondary and Intermediate Stages). By W. MACNEILL WHISTLER, M.D., Physician to the Hospital for Diseases of the Throat and Chest. Post 8vo, 4s.

Diseases of the Throat and Nose. A Manual. By MORELL MACKENZIE, M.D. Lond., Senior Physician to the Hospital for Diseases of the Throat and Chest. Vol. I. Diseases of the Pharynx, Larynx, and Trachea. Post 8vo, with 112 Engravings, 12s. 6d.

By the same Author.

Diphtheria :

Its Nature and Treatment, Varieties, and Local Expressions. 8vo, 5s.

The Ear :

Its Anatomy, Physiology, and Diseases. By CHARLES H. BURNETT, A.M., M.D., Aural Surgeon to the Presbyterian Hospital, Philadelphia. 8vo, with 87 Engravings, 18s.

A Treatise on Vocal Physiology and Hygiene, with especial reference to the Cultivation and Preservation of the Voice. By GORDON HOLMES, L.R.C.P. Edin., Physician to the Municipal Throat and Ear Infirmary. Second Edition. With Engravings. Crown 8vo, 6s. 6d.

By the same Author.

A Guide to the Use of the Laryngoscope in General Practice. Crown 8vo, with 15 Engravings, 2s. 6d.

Ear and Throat Diseases.

Essays by LLEWELLYN THOMAS, M.D., Surgeon to the Central London Throat and Ear Hospital. Post 8vo, 2s. 6d.

Sore Throat :

Its Nature, Varieties, and Treatment. By PROSSER JAMES, M.D., Physician to the Hospital for Diseases of the Throat. Fourth Edition. Post 8vo, with Coloured Plates and Engravings, 6s. 6d.

A System of Dental Surgery.

By JOHN TOMES, F.R.S., and CHARLES S. TOMES, M.A., F.R.S. Second Edition. Fcap. 8vo, with 268 Engravings, 14s.

Dental Anatomy, Human and Comparative: a Manual. By CHARLES S. TOMES, M.A., F.R.S. Second Edition. Crown 8vo, with many Engravings.

(Just ready.)

A Practical Treatise on Operative Dentistry. By JONATHAN TAFT, D.D.S., Professor in the Ohio College of Dental Surgery. Third Edition. With 134 Engravings. 8vo, 18s.

Dental Materia Medica and Therapeutics. By JAMES STOCKEN, L.D.S.R.C.S., late Lecturer to the National Dental Hospital. Third Edition. Fcap. 8vo. (*In the Press.*)

The Student's Guide to Dental Anatomy and Surgery. By HENRY SEWILL, M.R.C.S., L.D.S. Fcap. 8vo, with 77 Engravings, 5s. 6d.

A Manual of Dental Mechanics. By OAKLEY COLES, L.D.S.R.C.S., Second Edition. Crown 8vo, with 140 Engravings, 7s. 6d.

By the same Author.

Deformities of the Mouth.

Third Edition, 8vo, with 83 Wood Engravings and 96 Drawings on Stone, 12s. 6d.

Mechanical Dentistry in Gold and Vulcanite. By F. H. BALKWILL, L.D.S.R.C.S. 8vo, with 2 Lithographic Plates and 57 Engravings, 10s.

Lectures on Dermatology:

Delivered at the Royal College of Surgeons, by Sir ERASMUS WILSON, F.R.S. 1870, 6s.; 1871-73, 10s. 6d.; 1874-75, 10s. 6d.; 1876-78, 10s. 6d.

Eczema:

By MCCALL ANDERSON, M.D., Professor of Clinical Medicine in the University of Glasgow. Third Edition. 8vo, with Engravings, 7s. 6d.

Eczema and its Management:

A practical Treatise based on the Study of 2,500 Cases of the Disease. By L. DUNCAN BULKLEY, M.D., Physician for Skin and Venereal Diseases at the New York Hospital. 8vo, 12s. 6d.

Psoriasis, or Lepra.

By GEORGE GASKOIN, M.R.C.S., Surgeon to the British Hospital for Diseases of the Skin. 8vo, 5s.

On Certain Rare Diseases of the Skin: being Vol. 1 of "Lectures on Clinical Surgery." By JONATHAN HUTCHINSON, Senior Surgeon to the London Hospital, and to the Hospital for Diseases of the Skin. 8vo, 10s. 6d.

Leprosy in British Guiana:

An Account of West Indian Leprosy. By JOHN D. HILLIS, F.R.C.S., M.R.I.A., Medical Superintendent of the Leper Asylum, British Guiana. Imp. 8vo, with 22 Lithographic Coloured Plates and Wood Engravings. £1 11s. 6d.

Photographic Illustrations of Skin Diseases. Sixty Cases from Life.

By GEORGE H. FOX, M.D. 4to, £5 5s.

Cancer Life:

Its Causes, Progress, and Treatment. A General and Historical Treatise. By R. MITCHELL, M.R.C.S. 8vo, 7s. 6d.

On Cancer:

Its Allies, and other Tumours, with special reference to their Medical and Surgical Treatment. By F. ALBERT PURCELL, M.D., Surgeon to the Cancer Hospital, Brompton. 8vo, with 21 Engravings, 10s. 6d.

Atlas of Skin Diseases:

By TILBURY FOX, M.D., F.R.C.P. With 72 Coloured Plates. Royal 4to, half morocco, £6 6s.

Certain Forms of Cancer,

With a New and Successful Mode of treating it. By A. MARSDEN, Senior Surgeon to the Cancer Hospital. Second Edition. 8vo, with Coloured Plates, 8s. 6d.

Diseases of the Urinary Organs:

Clinical Lectures. By Sir HENRY THOMPSON, F.R.C.S., Emeritus Professor of Clinical Surgery in University College. Fifth Edition. 8vo, with 2 Plates and 71 Engravings, 10s. 6d.

By the same Author.

Diseases of the Prostate:

Their Pathology and Treatment. Fourth Edition. 8vo, with numerous Plates, 10s.

Also.

Practical Lithotomy and Li-

thotritry; or, an Inquiry into the best Modes of Removing Stone from the Bladder. Third Edition. 8vo, with 87 Engravings, 10s.

Also.

The Preventive Treatment of Calculous Disease, and the Use of Solvent Remedies. Second Edition. Fcap. 8vo, 2s. 6d.

Diseases of the Testis, Spermatic Cord, and Scrotum. By THOMAS B. CURLING, F.R.S., Consulting Surgeon to the London Hospital. Fourth Edition. 8vo, with Engravings, 16s.

Fistula, Hæmorrhoids, Painful Ulcer, Stricture, Prolapsus, and other Diseases of the Rectum: Their Diagnosis and Treatment. By WILLIAM ALLINGHAM, Surgeon to St. Mark's Hospital for Fistula. Fourth Edition. 8vo, with Engravings, 10s. 6d.

Cancer of the Rectum:

Its Pathology, Diagnosis, and Treatment. By W. HARRISON CRIPPS, F.R.C.S., Surgeon to the Great Northern Hospital, &c. Crown 8vo, with Lithographic Plates, 6s.

Hydrocele:

Its several Varieties and their Treatment. By SAMUEL OSBORN, late Surgical Registrar to St. Thomas's Hospital. Fcap. 8vo, with Engravings, 3s.

By the same Author.

Diseases of the Testis.

Fcap. 8vo, with Engravings. 3s. 6d.

Parasites:

A Treatise on the Entozoa of Man and Animals, including some Account of the Ectozoa. By T. SPENCER COBBOLD, M.D., F.R.S. 8vo, with 85 Engravings, 15s.

The Surgery of the Rectum.

By HENRY SMITH, Professor of Surgery in King's College, Surgeon to the Hospital. Fourth Edition. Fcap. 8vo, 5s.

Lectures on the Surgical Disorders of the Urinary Organs.

By REGINALD HARRISON, F.R.C.S., Surgeon to the Liverpool Royal Infirmary. Second Edition, with 48 Engravings. 8vo, 12s. 6d.

By the same Author.

The Prevention of Stricture and of Prostatic Obstruction.

8vo, with Engravings, 2s. 6d.

Lithotomy and Extraction of

Stone. By W. POULETT HARRIS, M.D., Surgeon-Major H.M. Bengal Medical Service. 8vo, with Engravings, 10s. 6d.

Diseases of the Bladder,

Prostate Gland, and Urethra, with a practical view of Urinary Diseases, Deposits, and Calculi. By F. J. GANT, Senior Surgeon to the Royal Free Hospital. Fourth Edition. Crown 8vo, 10s. 6d.

Renal and Urinary Diseases:

Clinical Reports. By WILLIAM CARTER, M.B., Physician to the Liverpool Southern Hospital. Crown 8vo, 7s. 6d.

Syphilitic Nervous Affections:

Their Clinical Aspects. By THOMAS BUZZARD, M.D., F.R.C.P. Physician to the National Hospital for Paralysis and Epilepsy. Second Edition. Post 8vo.

(In the Press.)

Pathology of the Urine,

Including a Complete Guide to its Analysis. By J. L. W. THUDICHUM, M.D., F.R.C.P. Second Edition, rewritten and enlarged. 8vo, with Engravings, 15s.

Genito-Urinary Organs, including Syphilis:

a Practical Treatise on their Surgical Diseases, designed as a Manual for Students and Practitioners. By W. H. VAN BUREN, M.D., and E. L. KEYES, M.D. Royal 8vo, with 140 Engravings, 21s.

Lectures on Syphilis.

By HENRY LEE, Consulting Surgeon to St. George's Hospital. 8vo, 10s.

Harveian Lectures on Syphilis.

By JAMES R. LANE, F.R.C.S., Surgeon to St. Mary's Hospital. Second Edition. Fcap. 8vo, 3s. 6d.

Photographic Illustrations of

Cutaneous Syphilis. Seventy Cases from Life. By GEORGE H. FOX, M.D. 4to, £5 5s.

Urinary and Reproductive Or-

gans: their Functional Diseases. By D. CAMPBELL BLACK, M.D. Second Edition. 8vo, 10s.

A Treatise on Syphilis.

By WALTER J. COULSON, Surgeon to the Lock Hospital and to St. Peter's Hospital for Stone. 8vo, 10s.

By the same Author.

Stone in the Bladder:

Its Prevention, early Symptoms, and Treatment by Lithotripsy. 8vo, 6s.

Also.

Coulson on Diseases of the Bladder and Prostate Gland.

Sixth Edition. 8vo, 16s.

The Reproductive Organs

In Childhood, Youth, Adult Age, and Advanced Life, considered in their Physiological, Social, and Moral Relations. By WILLIAM ACTON, M.R.C.S. Sixth Edition. 8vo, 12s.

Student's Primer on the Urine.

By J. TRAVIS WHITTAKER, M.D., Clinical Demonstrator at the Royal Infirmary, Glasgow. With 16 Plates etched on Copper. Post 8vo, 4s. 6d.

A Manual of the Laws affecting

Medical Men. By ROBERT G. GLENN, LL.B., Barrister-at-Law. 8vo, 14s.

The Medical Adviser in Life

Assurance. By EDWARD H. SIEVEKING, M.D., F.R.C.P., Physician to St. Mary's and Lock Hospitals, &c. Crown 8vo, 6s.

A Dictionary of Medical Science:

Containing a concise Explanation of the various Subjects and Terms of Medicine, &c.; Notices of Climate and Mineral Waters; Formulæ for Officinal, Empirical, and Dietetic Preparations; with the Accentuation and Etymology of the Terms, and the French and other Synonyms. By ROBLEY DUNGLISON, M.D., LL.D. New Edition. Royal 8vo, 28s.

A Medical Vocabulary:

Being an Explanation of all Terms and Phrases used in the various Departments of Medical Science and Practice, giving their Derivation, Meaning, Application, and Pronunciation. By ROBERT G. MAYNE, M.D., LL.D. Fifth Edition. Fcap. 8vo, 10s. 6d.

Abridged Medical Account

Books. The "Expedite" Method. By JAMES MACNAB, L.R.C.S.E. *Index Ledger*. Royal 4to. For four years, 21s. *Visiting List*. Cloth, 2s. 6d.; Leather, 3s. 6d.

Medical Education

And Practice in all parts of the World. By HERBERT JUNIUS HARDWICKE, M.D., M.R.C.P. 8vo, 10s.

INDEX.

- Acton's Reproductive Organs, 14
 Adams (W.) on Clubfoot, 11
 Contraction of the Fingers, 11
 Allan on Fever Nursing, 7
 Allingham on Diseases of the Rectum, 13
 Anatomical Remembrancer, 4
 Anderson (McC.) on Eczema, 13
 Aveling's Influence of Posture on Women, 6
 Balkwill's Mechanical Dentistry, 13
 Bantock on Rupture of the Female Perineum, 6
 Barclay's Medical Diagnosis, 8
 Barnes on Obstetric Operations, 5
 on Diseases of Women, 5
 Beale's Microscope in Medicine, 8
 Slight Ailments, 8
 Bellamy's Surgical Anatomy, 3
 Bennet (J. H.) on the Mediterranean, 10
 on Pulmonary Consumption, 10
 on Nutrition, 10
 Bentley and Trimen's Medicinal Plants, 7
 Berkart on Asthma, 8
 Bigg (H. H.) on Orthopraxy, 11
 Bigg (R. H.) on the Orthopragms of Spine, 11
 Binz's Elements of Therapeutics, 7
 Black on the Urinary Organs, 14
 Bose's Rational Therapeutics, 7
 Recognisant Medicine, 7
 Braune's Topographical Anatomy, 3
 Brodhurst's Anchylosis, 11
 Orthopædic Surgery, 11
 Bryant's Practice of Surgery, 11
 Bucknill and Tuke's Psychological Medicine, 5
 Bulkeley on Eczema, 13
 Burdett's Cottage Hospitals, 5
 Pay Hospitals, 5
 Burnett on the Ear, 12
 Burton's Midwifery for Midwives, 5
 Buzzard's Syphilitic Nervous Affections, 14
 Carpenter's Human Physiology, 4
 Carter (W.) on Renal Diseases, 14
 Cayley's Typhoid Fever, 9
 Charteris' Practice of Medicine, 8
 Clark's Outlines of Surgery, 10
 Clay's Obstetric Surgery, 6
 Cobbold on Parasites, 13
 Coles' Dental Mechanics, 13
 Deformities of the Mouth, 13
 Cormack's Clinical Studies, 8
 Coulson on Stone in the Bladder, 14
 on Syphilis, 14
 on Diseases of the Bladder, 14
 Cripps' Cancer of the Rectum, 13
 Cullingworth's Nurse's Companion, 7
 Curling's Diseases of the Testis, 13
 Dagenet's Manual of Ophthalmoscopy, 12
 Dalby's Diseases and Injuries of the Ear, 12
 Dalton's Human Physiology, 4
 Day on Diseases of Children, 6
 on Headaches, 9
 De Chaumont's Sanitary Assurance, 4
 Dobell's Lectures on Winter Cough, 8
 Loss of Weight, &c., 8
 Mont Dore Cure, 8
 Domville's Manual for Nurses, 7
 Druitt's Surgeon's Vade-Mecum, 11
 Duncan on the Female Perineum, 5
 on Diseases of Women, 5
 Dunglison's Medical Dictionary, 14
 Ellis's Manual for Mothers, 6
 of the Diseases of Children, 6
 Emmet's Gynecology, 5
 Eulenburg and Guttman's System of Nerves, 9
 Fayer's Observations in India, 7
 Tropical Dysentery and Diarrhœa, 7
 Fenwick's Chronic Atrophy of the Stomach, 8
 Medical Diagnosis, 8
 Outlines of Medical Treatment, 8
 Fergusson's Practical Surgery, 10
 Flint on Phthisis, 8
 on Clinical Medicine, 8
 Flower's Diagrams of the Nerves, 4
 Foster's Clinical Medicine, 8
 Fox's (C. B.) Examinations of Water, Air, and Food, 4
 Fox's (G. H.) Photographs of Cutaneous Syphilis, 14
 Skin Diseases, 13
 Fox's (T.) Atlas of Skin Diseases, 13
 Frey's Histology and Histo-Chemistry, 4
 Fulton's Text-Book of Physiology, 4
 Galabin's Diseases of Women, 6
 Gamgee's Fractures of the Limbs, 11
 Treatment of Wounds, 11
 Gant's Diseases of the Bladder, 14
 Gaskoin on Psoriasis or Leprosy, 13
 Gill on Indigestion, 10
 Glenn's Laws affecting Medical Men, 14
 Godlee's Atlas of Human Anatomy, 3
 Gowers' Diseases of the Spinal Cord, 9
 Epilepsy, 9
 Medical Ophthalmoscopy, 9
 Pseudo-Hypertrophic Muscular Paralysis, 9
 Habershon's Diseases of the Abdomen, 9
 Stomach, 9
 Pneumogastric Nerve, 9
 Hamilton's Nervous Diseases, 9
 Hardwicke's Medical Education, 14
 Harris on Lithotomy, 14
 Harrison's Surgical Disorders of the Urinary Organs, 14
 Prevention of Stricture, 14
 Heath's Injuries and Diseases of the Jaws, 10
 Minor Surgery and Bandaging, 10
 Operative Surgery, 10
 Practical Anatomy, 3
 Surgical Diagnosis, 10
 Hemming on the Laryngoscope, 12
 Higgins' Ophthalmic Out-patient Practice, 11
 Hillis' Leprosy in British Guiana, 13
 Hogg's Indian Notes, 7
 Holden's Dissections, 3
 Human Osteology, 3
 Landmarks, 3
 Holmes' (G.) Guide to Use of Laryngoscope, 12
 Vocal Physiology and Hygiene, 12
 Hood on Gout, Rheumatism, &c., 9
 Hooper's Physicians' Vade-Mecum, 8
 Horton's Tropical Diseases, 7
 Hutchinson's Clinical Surgery, 11
 Rare Diseases of the Skin, 13
 Huth's Marriage of Near Kin, 4
 Ireland's Idiocy and Imbecility, 5
 Irvine's Relapse of Typhoid Fever, 9
 James on Sore Throat, 12
 Jones' (C. H.) Functional Nervous Disorders, 9
 Jones' (C. H.) and Sieveking's Pathological Anatomy, 4
 Jones' (H. McN.) Aural Surgery, 12
 Atlas of Diseases of Membrana Tympani, 12
 Jones' (T. W.) Ophthalmic Medicine and Surgery, 11
 Jordan's Surgical Enquiries, 11
 Lancereaux's Atlas of Pathological Anatomy, 4
 Lane's Lectures on Syphilis, 14
 Lee (H.) on Syphilis, 14
 Leared on Imperfect Digestion, 10
 Liebreich's Atlas of Ophthalmoscopy, 11
 Liveing's Megrin, Sick Headache, &c., 10
 Lucas's Indian Hygiene, 8
 Macdonald's (A.) Chronic Disease of the Heart, 6
 Macdonald's (J. D.) Examination of Water, 4
 Macewen's Osteotomy: Knock-knee, Bow-leg, &c., 11
 Mackenzie on Diphtheria, 12
 on Diseases of the Throat and Nose, 12
 MacLise's Dislocations and Fractures, 11
 Surgical Anatomy, 3
 MacMunn's Spectroscope in Medicine, 8
 Macnab's Medical Account Books, 14
 Macnamara's Diseases of Bones and Joints, 11
 the Eye, 11
 Madden's Principal Health-Resorts, 10
 Marsden on Cancer, 13
 Martin's Military and State Medicine, 5
 Mason on Hare-Lip and Cleft Palate, 12
 on Surgery of the Face, 12
 Mayne's Medical Vocabulary, 14
 Mitchell (R.) on Cancer Life, 13
 Mitchell's (S. Weir) Nervous System in Women, 6
 Moore's Family Medicine for India, 7

(Continued on the next page.)

Moore's Health Resorts for Tropical Invalids, 7
 Morris' (H.) Anatomy of the Joints, 3
 Nettleship's Diseases of the Eye, 12
 Ogston's Medical Jurisprudence, 4
 Osborn on Diseases of the Testis, 13
 — on Hydrocele, 13
 Parkes' Practical Hygiene, 5
 Pavy on Diabetes, 10
 — on Food and Dietetics, 10
 Peacock's Diseases of the Heart, 9
 Phillips' Materia Medica, 7
 Pirrie's Principles and Practice of Surgery, 11
 Pollock on Rheumatism, 9
 Pridham on Asthma, 8
 Purcell on Cancer, 13
 Radford's Cesarean Section, 5
 Ramsbotham's Obstetrics, 6
 Reynolds' (J. J.) Diseases of Women, 6
 — Notes on Midwifery, 6
 Reynolds' (J. R.) Clinical Electricity, 10
 Roberts' (C.) Manual of Anthropometry, 5
 Roberts' (D. Lloyd) Practice of Midwifery, 5
 Ross's Diseases of the Nervous System, 9
 Roth on Dress: Its Sanitary Aspect, 5
 Roussel's Transfusion of Blood, 8
 Routh's Infant Feeding, 6
 Royle and Harley's Materia Medica, 7
 Rutherford's Practical Histology, 4
 Sanderson's Physiological Handbook, 4
 Sansom's Diseases of the Heart, 9
 — Antiseptic System, 9
 Savage on the Female Pelvic Organs, 6
 Sayre's Orthopædic Surgery, 11
 Schroeder's Manual of Midwifery, 6
 Sewill's Dental Anatomy, 13
 Sheppard on Madness, 5
 Sibson's Medical Anatomy, 3
 Sieveking's Life Assurance, 14
 Smith's (E.) Wasting Diseases of Children, 6
 — Clinical Studies, 6
 Smith's (Henry) Surgery of the Rectum, 14
 Smith's (Heywood) Dysmenorrhœa, 6
 — Gynæcology, 6
 Smith (Priestley) on Glaucoma, 12
 Smith (W. R.) on Nursing, 6
 Sparks on the Riviera, 10
 Squire's Companion to the Pharmacopœia, 7
 — Pharmacopœias of London Hospitals, 7
 Stillé and Maisch's National Dispensatory, 7

Stocken's Dental Materia Medica, 12
 Sullivan's Tropical Diseases, 7
 Swain's Surgical Emergencies, 10
 Swayne's Obstetric Aphorisms, 6
 Taft's Operative Dentistry, 12
 Taylor's Medical Jurisprudence, 4
 — Poisons in relation to Medical Jurisprudence, 4
 Teale's Dangers to Health, 5
 Thomas on Ear and Throat Diseases, 12
 Thompson's (Sir H.) Calculous Disease, 13
 — Diseases of the Urinary Organs, 13
 — Diseases of the Prostate, 13
 — Lithotomy and Lithotripsy, 13
 Thompson's (Dr. H.) Clinical Lectures, 8
 Thorowgood on Asthma, 8
 — on Materia Medica, 7
 Thudichum's Pathology of the Urine, 14
 Tibbits' Medical and Surgical Electricity, 10
 — Map of Motor Points, 10
 Tilt's Change of Life, 6
 — Uterine Therapeutics, 6
 Tomes' (C. S.) Dental Anatomy, 12
 — (J. & C. S.) Dental Surgery, 12
 Van Buren on the Genito-Urinary Organs, 14
 Veitch's Handbook for Nurses, 7
 Virchow's Post-mortem Examinations, 4
 Wagstaffe's Human Osteology, 3
 Walker's Ophthalmology, 12
 Waring's Indian Bazaar Medicines, 7
 — Practical Therapeutics, 7
 Warner's Guide to Medical Case-Taking, 8
 Waters' (A. T. H.) Diseases of the Chest, 8
 Waters (J. H.) on Fits, 9
 Wells (Spencer) on Ovarian and Uterine Tumours, 6
 West and Duncan's Diseases of Women, 5
 Whistler's Syphilis of the Larynx, 12
 Whitehead's (J. L.) Climate of the Undercliff, 10
 Whittaker's Primer on the Urine, 14
 Wilks' Diseases of the Nervous System, 9
 Wilks and Moxon's Pathological Anatomy, 4
 Wilson's (Sir E.) Anatomists' Vade-Mecum, 3
 — Lectures on Dermatology, 13
 Wilson's (G.) Handbook of Hygiene, 5
 — Healthy Life and Dwellings, 5
 Wilson's (W. S.) Ocean as a Health-Resort, 10
 Wise's Davos Platz, 10
 Wolfe's Diseases and Injuries of the Eye, 11
 Woodman and Tidy's Forensic Medicine, 4

The following CATALOGUES issued by J. & A. CHURCHILL will be forwarded post free on application:—

A. *J. & A. Churchill's General List of more than 600 works on Anatomy, Physiology, Hygiene, Midwifery, Materia Medica, Medicine, Surgery, Chemistry, Botany, &c., &c., with a complete Index to their Subjects, for easy reference.* N.B.—This List includes B, C, & D.

B. *Selection from J. & A. Churchill's General List, comprising all recent Works published by them on the Art and Science of Medicine.*

C. *J. & A. Churchill's Catalogue of Text Books specially arranged for Students.*

D. *A selected and descriptive List of J. & A. Churchill's Works on Chemistry, Materia Medica, Pharmacy, Botany, Photography, Zoology, the Microscope, and other branches of Science.*

E. *The Half-yearly List of New Works and New Editions published by J. & A. Churchill during the previous six months, together with Particulars of the Periodicals issued from their House.*

[Sent in January and July of each year to every Medical Practitioner in the United Kingdom whose name and address can be ascertained. A large number are also sent to the United States of America, Continental Europe, India, and the Colonies.]

AMERICA.—*J. & A. Churchill being in constant communication with various publishing houses in Boston, New York, and Philadelphia, are able, notwithstanding the absence of international copyright, to conduct negotiations favourable to English Authors.*

LONDON: NEW BURLINGTON STREET.

