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Dr. Walter B. James



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Fig. 1

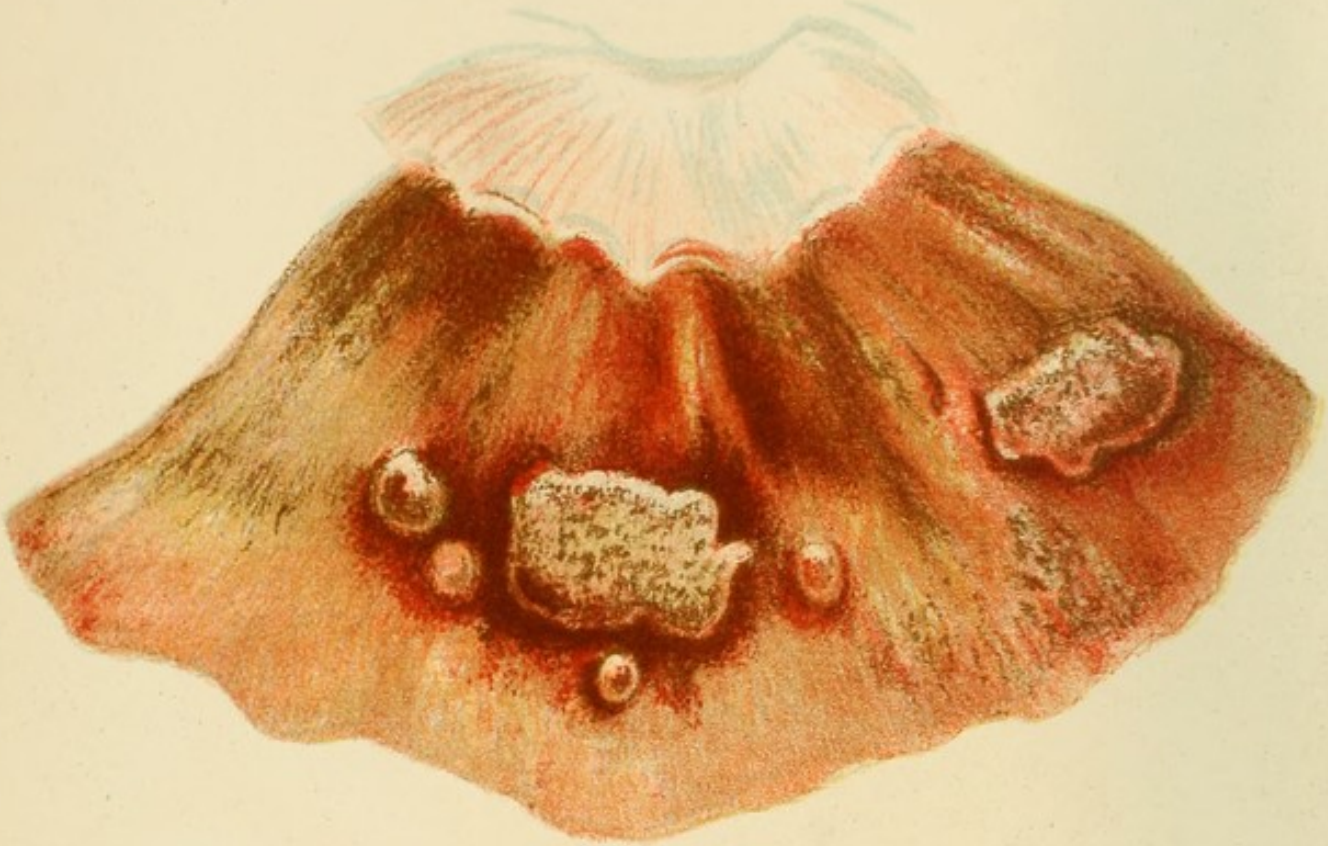


Fig. 2



E. P. Henry

INTESTINAL LESIONS

Lithographed by

Fig. 3



F TYPHOID FEVER.

Falconer Dublin

TEXT-BOOK

OF THE

ERUPTIVE AND CONTINUED FEVERS.

BY

JOHN WILLIAM MOORE, B.A., M.D., M.CH., UNIV. DUBL.;

FELLOW AND REGISTRAR OF THE ROYAL COLLEGE OF PHYSICIANS OF IRELAND;

PHYSICIAN TO THE MEATH HOSPITAL, DUBLIN;

JOINT PROFESSOR OF PRACTICE OF MEDICINE IN THE SCHOOLS OF SURGERY OF THE ROYAL COLLEGE OF SURGEONS IN IRELAND;

CONSULTING PHYSICIAN TO CORK-STREET FEVER HOSPITAL, DUBLIN, AND TO THE WHITWORTH HOSPITAL, DRUMCONDRA;

EX-SCHOLAR AND DIPLOMATE IN STATE MEDICINE OF TRINITY COLLEGE, DUBLIN.

WILLIAM WOOD & COMPANY,

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NEW YORK.

1892.

To the rebered Memory of

MY FATHER,

WILLIAM DANIEL MOORE,

M.D. Dubl. et Cantab., M.R.I.A.,

(*April 19, 1813—October 28, 1871*),

I DEDICATE THIS BOOK.

PREFACE.

It may be desirable to state very briefly the circumstances under which this work has come to see the light.

To write a Text-book on Fever while the splendid monograph of Charles Murchison is our possession to all time may well be deemed presumptuous. But, on the threshold, I would disclaim any intention of allowing the present work—"hoc parvum opusculum"—to enter the lists with that unrivalled masterpiece. However, even since Dr. William Cayley, in 1884, edited, with excellent good taste and literary skill, the posthumous edition of Murchison's Treatise, the study of the Continued Fevers has received a great impetus, and the necessity of a sound knowledge of Fever by every practitioner of medicine has been more and more recognised.

As regards the Eruptive Fevers, we possess no work at all analogous to, or comparable with, that of Murchison on the Continued Fevers. It is true that a very full and accurate description of these diseases is to be found in the third edition of Hilton Fagge's and Pye-Smith's "Text-book of the Principles and Practice of Medicine," published in 1891. The standard works on Practice of Medicine also contain a number of chapters devoted to this subject; and I am aware that a Text-book upon the Eruptive Fevers by Dr. Alexander Collie has been published within the last few years.

Notwithstanding all this, I make bold to say that full justice has not up to the present been done to so important and fascinating a theme. While I cannot pretend to have adequately supplied an admitted want in medical literature—

namely, a reliable and comprehensive Text-book of the Eruptive Fevers, yet I have endeavoured to focus in its pages the most recent views on the ætiology, bacteriology, symptoms, pathology, and treatment of this group of maladies.

Of late years the Spirit of Scientific Discovery has been abroad. In the realms of both Curative and Preventive Medicine its presence has been felt, but in no direction more than in the fertile field of Bacteriology. The microbic origin of enteric fever, erysipelas, pneumonia, cholera, and diphtheria, has been practically demonstrated, and kindred investigations are still engaging the earnest attention of the foremost thinkers in this country, on the Continent, and in America. We are not yet in a position fairly or accurately to gauge the gain to Humanity and to Science which will accrue from such researches as those of Burdon-Sanderson and E. Klein, of Robert Koch, of Louis Pasteur and Toussaint, and of other toilers in the vast field of original investigation, but they bear glowing testimony to the advance, by leaps and bounds, of Medical Science. They teach the lesson—so full of encouragement to the God-fearing and man-loving physician—that, in the not distant future, success will surely crown his noble effort to combat disease, to save life, and to stem the rising tide of human misery and despair.

The study of Fever has always had a peculiar attraction for me. Perhaps this arose in part from the fortunate circumstance that as a medical student I enjoyed the advantage of the philosophic teaching of William Stokes and was a daily witness of the lynx-eyed observation of Alfred Hudson in the wards of the Meath Hospital. These are names to conjure with from the bead-roll of authors upon Fever.

But above and beyond this personal consideration, it has always seemed to me that each individual case of Fever presented to the attending physician or to the clinical student an epitome of the Principles and Practice of Medicine.

"Fever," said Fordyce, nearly a century ago, "is a disease which affects the whole system; it affects the head, trunk, and extremities; it affects the circulation, absorption, and the nervous system; it affects the body, and it affects the mind; it is therefore a disease of the whole system, in the fullest sense of the term." This being so, we surely are in a position to study diseases of the three great cavities of the body, diseases of the circulation, of the lymphatic, digestive, and nervous systems in an individual case of fever. If our object is—as it should be—to accurately gauge the state of our fever patient, we cannot attain this end by a mere superficial examination. On the contrary, every known method of physical examination must be applied, and this too as regards every system of the body in rotation.

It would be equally disastrous to our fever patients were we, on the one hand, to treat a case which showed cerebral symptoms as if inflammation of the brain or its membranes was really present, were we, on the other hand, to act upon the unwarranted assumption that the kidneys in a given patient were sound and efficient.

In a word, there is no other disease which demands on the part of the physician a closer and more intelligent observation, a more minute and searching physical examination, a more subtle and refined train of reasoning, a more careful weighing of evidence for or against, and a more conscientious and painstaking management from start to finish.

The Royal College of Physicians of Ireland—to their credit be it spoken—many years ago recognised the paramount importance of a close study of fever by all candidates for the License to Practise Medicine granted by the College. The President and Fellows required, and still require, that every candidate for that License shall produce evidence of having for not less than three months studied Fever in a recognised Clinical Hospital containing fever-wards, and of having re-

corded, *from daily personal observation*, the notes of at least five cases of fever to the satisfaction of the attending clinical physician, as attested by his signature.

The University of Dublin requires of all candidates for the degree of Bachelor in Medicine or for the Diploma in Medicine a certificate of personal attendance on Fever Cases, with the names and dates of the cases attended.

The Royal University of Ireland requires evidence of personal attendance on at least ten fever cases during three consecutive months in a Fever Hospital of repute, or in the fever wards of a General Hospital.

The Irish Medical Licensing Bodies have thus nothing to be ashamed of in so important a matter.

At their Summer Session in 1890, the General Medical Council, on June 5, adopted the following resolution :—

> "RESOLVED,—That no qualification in Medicine ought to be granted without evidence of clinical instruction in Infectious Diseases."

This resolution was adopted in consequence of the receipt of a communication from the Local Authority for Glasgow under the Public Health (Scotland) Act. To this body belongs, therefore, the credit of initiating this important public recognition of the Clinical Study of Fever.

It is, therefore, plain that in future a personal study of fever will be an indispensable part of medical education.

This was one of the considerations which led me to write a book which, I hoped, might serve as a reliable guide to the student of fever.

In the spring of 1891, the Trustees of the Queen Victoria Jubilee Nursing Institute asked me to deliver a course of lectures on Fevers and Fever-Nursing to the nurses and probationers who were working under the auspices of the Institute among the sick-poor of Dublin and its suburbs in their own homes. The notes which I compiled for that

course of lectures first suggested the idea of publishing a book on the Nature and Treatment of Fever.

As to any special qualifications for my self-imposed task, I can lay claim to none, beyond the fact that for a period of thirteen years I was one of the Visiting Physicians to Cork-street Fever Hospital, Dublin.

It may, perhaps, be objected that in writing this book I have leaned too much on the clinical experience and on the literary labours of others. No doubt the following pages bristle with the names of authorities; but I have always endeavoured to test their statements by the touchstone of my own experience, nor have I hesitated to freely criticise any observations or opinions apparently unwarranted by certain facts, which appeared to me to be, perhaps, capable of a different interpretation.

It may be necessary to observe that all the Temperature Charts in the nine Plates which illustrate this book are, with three exceptions—the two charts of the temperature ranges in relapsing fever and the chart which shows a crisis in enteric fever, and which I owe to the kindness of Dr. H. T. Bewley—reproduced from among many hundreds taken in my own cases at Cork-street Fever Hospital and at the Meath Hospital.

The pleasant task remains of expressing my grateful acknowledgments to several friends who kindly helped me in my work. My cordial thanks are especially due to Dr. H. T. Bewley, Assistant Physician to the Adelaide Hospital, and Dr. Edward E. Lennon, Senior Clinical Assistant to the Meath Hospital, who undertook the labour of reading over the proof sheets as they issued from the press. To them, and also to Dr. James Little, Dr. T. W. Grimshaw, Registrar-General for Ireland, Dr. Walter G. Smith, and Dr. C. J. Nixon, I am deeply indebted for valuable suggestions and useful references.

Nor should I omit to mention the trouble taken in searching out books and original papers by Mr. S. W. Wilson, the Librarian of the Royal College of Physicians of Ireland on the Foundation of Sir Patrick Dun.

I desire also to acknowledge the courtesy of Dr. William Cayley, the editor of the third edition of Murchison's work on the "Continued Fevers," and of the eminent firm of publishers, Messrs. Longmans, Green, & Co., in permitting me to reproduce a series of very instructive Diagrams from that work.

In conclusion, I can but express the hope that the following pages will prove useful to some of that devoted band who have already entered, or are about to enter, upon the practice of the noblest of all Professions—the Church not even excepted.

JOHN WILLIAM MOORE.

40 FITZWILLIAM-SQUARE, WEST, DUBLIN,
December 21st, 1891.

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ERRATA.

Page 25--Line 7 from bottom, for "fastidium" read "fastigium."
Page 42—Line 4 from top, for "minute" read "second."
Page 202—Line 7 from top, for "not" read "now."
Pages 320 and 324—For "Moschutkovsky" read "Motschutkovsky."
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TEXT-BOOK

OF THE

ERUPTIVE AND CONTINUED FEVERS.

PART I.—INTRODUCTION.

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The word **Fever** means literally "a burning." It is derived from the Latin *Febris* (Gk. πυρετός), and in kindred forms appears in the chief modern European tongues. Thus, we have *la fièvre* in French; *das Fieber*, in German; *la febbre*, in Italian; *la fiebre*, in Spanish; *Feber*, in both Swedish and Norwegian or Danish. The Spanish *Calentura* expresses the same idea.[a]

The appropriateness of the term is apparent when we reflect that the essential fact in fever is preternatural body heat. This is in no small degree due to increased oxidation (combustion) of nitro-

[a] "Fieber (πυρετός = fever), *febris* eigentlich *ferbris* von *fervere* heiss sein; *appellamus à fervore febrin* (Varro)."—*Real-Encyclopädie der Gesammten Heilkunde.* Siebenter Band. Wien und Leipzig: Urban und Schwarzenberg. 1886. S. 171.

genous and carbonaceous substances furnished to the blood by the tissues, but mainly by the food.[a]

To the feverish state, the term **Pyrexia,** or **Feverishness,** is applied. Pyrexia is not a classical Greek word, but is connected with, and derived from, the future indicative tense of the verb πυρέσσω (fut. πυρέξω), to be in a state of πυρετός, or to be feverish.

Pyrexia, or "feverishness," is either *symptomatic* or *idiopathic.*

Symptomatic fever means the high temperature and general constitutional disturbances—such as shivering, headache, loss of appetite, nausea, vomiting, constipation or diarrhœa, thirst, prostration, sleeplessness, and so on—which accompany and depend on local inflammations: for example, abscess, synovitis, ophthalmia, peritonitis, or pleurisy. This form of fever is, accordingly, often described as secondary, or accidental, fever.

Idiopathic fever, on the contrary, means the high temperature and general constitutional disturbances which, although occasionally *complicated with* local inflammations—as, for example, the enteritis of typhoid fever, the sore throat of scarlatina, the dermatitis of smallpox—are independent of them, and result from the absorption from without of some poison, which under favouring circumstances multiplies in the system, or from the action on the nervous centres of a poison inbred in the body—what Murchison calls a "non-specific cause."

As in this case the morbid phenomena follow in the train of the fever, which results from a specific cause, idiopathic fever is called **primary, specific,** or **essential,** in contradistinction to symptomatic fever, which, as we have seen, is **secondary,** of **non-specific** or **accidental** origin.

From the earliest times **elevation of bodily temperature above normal** has ever been regarded as the cardinal fact in fever. Murchison says that "the abstract definition of Fever given by Hippocrates, Galen, and Avicenna, was 'Essentia vero febrium est—præter naturam caliditas' (Ἡ παρὰ φύσιν θερμασία)."[b]

[a] Murchison: *A Treatise on The Continued Fevers of Great Britain.* Third Edition. Page 14. London: Longmans, Green, & Co. 1884.

[b] *Loc. cit.*, page 14.

"Fever," wrote Virchow,[a] the Father of Modern Pathology, "consists essentially in elevation of temperature, which must arise in an increased tissue-change, and have its immediate cause in alterations of the nervous system."

To the same effect Traube[b] declares that "Fever consists essentially in an increased temperature of the blood;" and Samuel says:—"By fever we understand an elevation of temperature, which is evoked through lesion of the Heat-centres."[c] Again, in 1887, Dr. Donald Macalister[d] held that "the essential fact in Fever, the condition which is always present, is disorder of the body-heat." And, in the same year, the late Dr. Austin Flint[e] wrote:—"Fever is a condition of excessive production of heat, involving defective nutrition or inanition, an excessive production and discharge of nitrogenised excrementitious matters and carbonic acid, with waste and degeneration of the tissues, and partial or complete suppression of the production and discharge of water." Liebermeister[f] defines fever "as a symptom-group, at the foundation of which is an elevation of the temperature of the body produced by a morbid general increase of metamorphosis"—as Dr. Donald Macalister graphically says, the organism during the febrile process is like "a candle burning at both ends." Lastly, the late Dr. Hilton Fagge,[g] following Liebermeister, observed that "Pyrexia consists in a change in the normal function of heat-regulation, by which the production of heat and its loss are so balanced as to create and maintain, while the pyrexia lasts, a higher temperature instead of the normal temperature. One might imagine," he adds

[a] *Handbuch der speciellen Pathologie und Therapeutie.* Band I. *Das Fieber.* Erlangen. 1854.

[b] *Ueber Krisen und kritische Tage.* Berlin. 185[illegible].

[c] "Unter Fieber versteht man eine Temperaturerhöhung, welche durch Erkrankung der Warme-centren hervorgerufen ist." — *Real-Encyclopädie der gesammten Heilkunde.* Art. "*Fieber.*"

[d] *The Nature of Fever.* London: Macmillan & Co. 1887.

[e] *Fever: its Cause, Mechanism, and Rational Treatment. Medical News,* Saturday, September 10, 1887.

[f] Cf. *Handb. der Pathol. und Therap. des Fiebers.* 1875.

[g] *Principles and Practice of Medicine.* London: J. & A. Churchill. 1886. Vol. I., page 40.

theoretically, "the index of the regulating machinery to be shifted upwards, so that it is set—not at 98·4°—but at 101°, 102°, 103°, or even a still higher point."

And yet there exists **no absolutely pathognomonic symptom for fever.** Even that which is most constantly present, the elevation of temperature, may show itself without fever; it may, on the other hand, be wanting notwithstanding the fever.[a]

To the right understanding of the intimate nature of Fever, a knowledge of the **Theory of Animal Heat** is indispensable. It is now sufficiently well made out that the natural heat of the body is due to both vital and chemical processes, duly controlled by the nervous centres.

The physiological facts which have to be kept before us in considering the theory of Fever are well and clearly described by Dr. T. J. Maclagan[b] as follow:—(1.) Two main processes embraced under the general term **metabolism** (German, *Stoffwechsel*), are constantly going on in the system—**tissue formation** (that is, **repair**, or **anabolism**) and **tissue disintegration** (that is, **waste, retrograde metamorphosis**, or **katabolism**). (2.) During tissue disintegration various products destined for elimination are formed—the principal of these are urea, carbonic acid, and heat. (3.) Urea is eliminated chiefly by the kidneys, carbonic acid chiefly by the lungs, and heat chiefly by the skin. (4.) In the case of each, production and elimination are so well balanced that no accumulation takes place in the system. (5.) In the case of heat, this balance is so well maintained that the mean normal temperature is always the same (98·4° F.). (6.) The various processes here indicated are presided over by the nervous centres, and it is by the regulating power of these centres that the normal balance is maintained.

In fever this whole process is disturbed; there is increased formation of urea and carbonic acid, and the temperature rises above

[a] "Es giebt kein für das Fieber absolut pathognomonisches Symptom. Das constanteste selbst, die Temperaturerhöhung, kann vorhanden sein ohne Fieber, es kann fehlen trotz des Fiebers."—Samuel. Art. *Fieber*, in the *Real-Encyclopädie der gesammten Heilkunde*. Wien und Leipzig: Urban und Schwarzenberg.

[b] *Fever: a Clinical Study*. By T. J Maclagan, M.D. London: J. & A. Churchill. 1888. Page 2.

98·4° F. "The preternatural heat of fever," says Murchison, "is the result of vital and chemical action exalted above the standard of health, assisted, perhaps, by a disturbance of the processes by which heat is carried away."

The parts which go to form the **Thermal Apparatus** are, according to Dr. Maclagan:—

(1.) The tissues—and particularly the muscles—in which heat is formed.

(2.) The surface from which heat is eliminated—about 80 per cent. normally escaping by the skin, and less than 20 per cent. by the lungs.

(3.) A central controlling power in the nervous centres.

(4.) Nerves connecting this with the heat-forming parts of body.

(5.) Nerves connecting the heat-centre with the heat-eliminating surface.

Dr. Donald Macalister describes the **Thermal Nervous System** as consisting of three parts:—

(1) The Heat-adjusting Mechanism (Thermotaxis).

(2) The Heat-producing Mechanism (Thermogenesis).

(3) The Heat-discharging Mechanism (Thermolysis).

Disorder of the first implies irregularity of temperature only; disorder of the first and second implies, in general, heightened temperature and increased body-heat—that is, ordinary fever; disorder of the first, second, and third implies, in general, hyperpyrexia (or excessive fever), dangerous increase of heat, and steadily *rising* temperature.

In the ascending scale of evolution in the Animal Kingdom, we seem to rise from the thermolytic to the thermogenic, and thence to the thermotaxic nervous system. Cold-blooded animals possess a thermolytic mechanism—a nervous mechanism which controls the vessels and the breathing. We cannot easily throw a frog into an enduring fever. In young mammals, the thermogenic system is developed before the thermotaxic—hence a slight cause sends an infant into a "high fever"—a little restores it again. As the child grows, the thermotaxic mechanism is evolved, and its temperature becomes stable.

Dr. Macalister looks upon "Fever" as a "dissolution"—a relaxation of control from above downwards. Thermotaxis is the first part of the mechanism to be disturbed and overthrown. Thermogenesis then becomes more and more excessive, and, lastly, the thermolytic mechanism fails to act.

In recovery from a typical febrile attack, the thermolytic mechanism is first waked to adequacy, and the result is a critical sweat, relaxation of the vessels of the skin, and a sudden fall of surface temperature. Next, thermogenesis becomes less and less excessive; and, lastly, thermotaxis—the first to be disturbed and overthrown—is at length restored, the patient's temperature becoming more and more stable.

According to this theory, Fever or Pyrexia depends on impaired thermotaxis, exalted thermogenesis, and more or less complete failure of thermolysis.

In other words, rise of temperature above normal is due to—

α. Impairment of that inhibitory force by which the heat-producing process is kept within normal physiological bounds—that is, impaired thermotaxis;

β. Increased activity of the process by which heat is naturally formed—that is, exalted thermogenesis.

These are the **neurotic** and **metabolic theories**, respectively, of fever. They are not antagonistic, but each, while complete in itself, supplements the other.

"If the heat-centres alone are diseased," writes Samuel,[a] "the question is one only of a simple, pure, uncomplicated fever (*febris simplex pura*). If through the exciting cause other nervous centres are simultaneously affected with the heat-centres, then a complex, mixed fever (*febris complicata*, *febris mixta*) arises—in this case from its most frequent cause, usually designated as **Infective Fever (Infectionsfieber)**."

Professor Arnaldo Cantani,[b] of Naples, is not satisfied with the

[a] *Loc. cit.*

[b] *Ueber Antipyrese. Transactions of the Tenth International Congress*, Berlin, 1890. Vol. I., page 152. Cf. *The Medical Chronicle*, October, 1890. Manhester.

theory which makes the whole fever process depend on certain nerve-centres. The **local production of heat**, the result of processes of chemical combustion, forms an **essential factor**. The action of nerve-centres may produce a temporary rise of temperature, but not a continuous or remittent elevation. Fever must be looked upon as an **acute alteration of organic metabolism**, with increase of tissue combustion, and consequently also of heat and consumption of body. Fever is a general or essential, and—to a certain degree—beneficial **reaction of the organism** to changes in metabolism and in the blood, produced by the causative agent of the disease. This reaction is necessary to bring about cure in acute disease. Fever, therefore, may be useful as long as the consumption of tissue does not give rise to exhaustion, and the cardiac muscle does not suffer, or the nervous system is not threatened by hyperpyrexia. Thus the temperature alone is not a measure of the gravity of the disease—a moderate degree of fever may be due to a want of reactive power on the part of the organism, a high degree to the energy with which the organism defends itself against the invasion of the disease. **Pyrexia** may thus **actually be of use**—(1) by diminishing the vitality or virulence of the living causes of disease, and by raising the temperature of the tissues and of the blood; (2) by increasing the power of resistance of the tissue elements in their phagocytic activity; (3) by altering the nutritive soil in the tissues and rendering it less favourable for the growth and development of the germ of disease—in fact, by sterilising the body. At the close of his able Address Cantani uses these remarkable words:—"Das Fieber, das in so vielen Krankheiten der beste Verbündete des Arztes ist" ("The fever, which in so many diseases is the best ally of the physician"), and he quotes Boerhaave's question and answer—"Quid est febris? Est naturæ irritatæ conamen ad expellendum stimulum inconsuetum," as well as Borsieri's words—"Quos interdum morbos remedia non curant, febris curat."

Viewing the fever-process in this light, German writers now often speak of what they call "**Das Heil-Fieber.**"

The **Pathology of the infective or specific Fevers** may be thus described in general terms:—

1. A specific poison, called the "virus" or "contagium," enters the blood.

2. This poison affects the nervous system, by altering its blood-supply both in quantity and in quality, and disturbs its heat-producing, regulating, and discharging functions.

3. There is increased tissue change, while little fresh material is assimilated to compensate for the loss. The results are—rise of temperature, muscular prostration, loss of weight.

4. Rapid action of the heart results from nerve paralysis affecting the pneumogastric and cardiac ganglia, as well as from the irritation of poisoned blood. A pulse-rate of 70 beats per minute represents 100,800 beats of the heart in every 24 hours, but the fever-pulse of 120 per minute represents 172,800 beats in every 24 hours. This rapid heart action still further increases tissue waste.

5. Non-elimination of waste-products (or "ashes," as Dr. Lauder Brunton[a] aptly calls them) results from impaired functional activity of the skin, kidneys, and bowels, and this—together with the circulation of poisoned over-heated blood—causes the so-called nervous or ataxic symptoms of fever, as well as other complications.

6. A time comes when these waste-products, and the fever-poison also, are eliminated by the breath, the skin, the bowels, or the kidneys; when also the fever-poison perishes either through lack of nutrient material in the tissues of the patient's body or in consequence of the development of some chemical toxic principle by the specific micro-organisms which are themselves believed to be, or to give rise to, that fever-poison. When all this takes place, the nervous system resumes its normal functions, undue tissue waste is checked, the different organs and tissues recover their wonted activity, and the patient regains his health and strength. If this favourable change takes place quickly, we say that the fever has ended by **crisis**; if gradually and slowly, we speak of resolution of the fever by **lysis**.

[a] *Address in Medicine to the Brit. Med. Assoc.* 1891. Cf. *Brit. Med Journal*, Aug. 1, 1891, page 229.

In this sketch of the pathology of fever in the abstract, I have closely followed Murchison's aphorisms, and these are based on physiological, clinical, and pathological considerations which cannot be gainsaid.

The **essential phenomena** of fever are, according to Maclagan—

1. Wasting of the nitrogenous tissues.
2. Increased consumption of water.
3. Increased elimination of urea.
4. Increased rapidity of the circulation.
5. Preternatural body-heat ("calor præter naturam"—GALEN.)

If we bear these phenomena in mind, we shall be in a position, with Dr. Maclagan, to answer the questions: "**Why does the Fever Patient waste?**"—"**Why is he thirsty?**" We have already hinted at the causal relation to fever in which certain micro-organisms stand. These will be more minutely described in the second chapter, on the "Intimate Nature of Contagium." Suffice it here to state that these micro-organisms, or microbes, consist largely of **protoplasm.** They are reproduced in vast numbers in the system during the course of the disease to which they give rise, and in their growth they consume a large quantity of **nitrogen** and of **water.**

Voit has distinguished between the fixed or organ-albumen, which enters into the composition of the solid tissues and changes slowly, and the circulating, or store-albumen, which is contained in the blood and fluids of organs, and very readily undergoes change. This circulating or store-albumen consists of two parts—a *constructive* part, destined for the nutrition and building up of the tissues, and a *retrogressive* part, derived from tissue-waste and destined for excretion. The former is converted into solid nitrogenous tissue; the latter is converted into urea in the urea-forming glands, and as such is eliminated chiefly by the kidneys.

The **constructive store-albumen** is the source whence the "contagium" would by preference naturally take its nitrogen, and so the tissues are "starved" in fever—hence the **wasting** of fever.

But the protoplasm of the living and active contagium consists mainly of water, and for its growth a large consumption of water

is required. This is drawn from the tissues, and at the same time the water taken in by the patient is appropriated by the contagium. Hence the dry skin and the parched mucous membranes of fever, as well as the inordinate **thirst** and the loss of appetite complained of by the patient, the scanty urine, and the constipation.

The thirst of fever is the expression of a real want in the system, it is a craving for more water. Such is Maclagan's theory—ingenious indeed, but not yet capable of demonstration, or free from difficulties.

The further question arises—**How may water be supplied to the fever-patient?** A long personal experience has convinced me that no risk to the patient in fever arises either from the use of cold water as a beverage, or from frequent ablutions, such as are necessary if personal cleanliness is to be maintained. In the case of young children, especially, to withhold water in fever is downright cruelty. Taken internally, water is the most effectual assuager of consuming thirst, the best and safest diuretic, diaphoretic, aperient and eliminative, we can prescribe. Its administration in moderate quantities, given at frequent intervals, often allays delirium and induces sleep. Another way in which water may be given is in the form of ice, sucking fragments of which is most grateful to the patient, as well as beneficial, for it reduces temperature, relieves sore throat, and allays sickness and vomiting.

There can be no doubt also that, in fever, water is absorbed by the skin, when tepid sponging is practised, or the patient is placed in a warm, tepid, or even cold bath.

In his work on "Therapeutics,"[a] Dr. H. C. Wood, Professor of Materia Medica and Therapeutics in the University of Pennsylvania, speaks highly of the value of water as a diuretic in certain diseases. In an elaborate series of experiments, E. Roux[b] found that the ingestion of large quantities of water greatly increased the flow of urine, but did not sensibly affect the elimination of urea or uric acid, although that of the chlorides seemed to be augmented.

[a] *Therapeutics: its Principles and Practice.* By H. C. Wood, M.D., LL.D. Seventh Edition. London: Smith, Elder & Co. 1888. Page 706.

[b] *Archives Physiologiques.* 1874. Page 578.

In a paper entitled "Einfluss des Wassers," communicated to the *Zeitschrift der k. k. Gesellschaft der Aertze zu Wien* for April, 1854 (page 308), Dr. Böcker[a] arrived at the following conclusions:—

1. That water increases the interstitial metamorphosis of tissue and consequent loss of weight.
2. That the decomposed tissue is excreted partly in the urine and partly in the solid fæces.
3. That the water formed in the organism by the change of tissue is augmented, as well as the nitrogenous constituents of the excretions.

The investigations of J. Meyer[b] explain these discrepancies, and show how water may be of use in various diseases. He found that at times, *when the tissues were full of the products of disintegration*, the effect of water in increasing elimination was very marked, but that upon the wasting processes of the body the water exerted no influence. Having quoted these opinions, Dr. Wood goes on to say—"It would seem, therefore, that while we cannot by water produce tissue-disintegration, *we can by it wash out the retained products of tissue-change.*"

This is one of the most pressing indications of treatment in fever, and it is of the first importance to the physician to know that it is safely and efficiently fulfilled by the **internal administration of water.**

[a] *Brit. and Foreign Med.-Chir. Rev.* Vol. XIV., page 394. 1854.
[b] *Hoffmann und Schwalbe's Jahresbericht.* 1881. Page 345.

CHAPTER II.

The Intimate Nature of Contagion.

Communicable and Non-communicable Diseases.—The "Germ Theory of Disease."—Meanings of the term "Contagion."—Zymotic Diseases.—A typical example of Fermentation.—Nägeli's Classification of the decomposition-producing Fungi: Moulds, Yeasts, Fission-fungi (including Bacteria).—Characters and sub-divisions of the Fission-fungi, or Schizomycetes.—Non-pathogenic Micro-organisms or Microbes (Saprophytes).—Pathogenic Micro-organisms or Microbes (Parasites).—Division of the latter into—(1.) Non-infectious, but toxic, organisms; (2.) Truly infectious organisms.—Toxins, or Ptomaïns.—Crookshank's Classification of Bacteria.—Aërobic and Non-aërobic Micro-organisms (Pasteur): their food and drink.—Sporulation: "Resting Spores."—Explanation of supposed spontaneous, or *de novo*, origin of Infectious Fevers.—Specific, infective microbe, virus, or contagium.—Seat of the Development of the several Contagia.—Inoculation.—"Contagious" and "Infectious" Diseases.

Diseases may be roughly divided into two great classes—**communicable** diseases, or those capable of being imparted or communicated from one living being to another; and **non-communicable** diseases, or those not capable of being so imparted or communicated. Some of the former class are interchangeable, not only between one individual of the same species and another, but also between animals and men or between men and animals. Familiar examples are: hydrophobia, malignant pustule (anthrax, or charbon), glanders, tuberculosis, smallpox, and others.

On the threshold of our investigations into the intimate nature of contagion we are brought face to face with what has been called the "**Germ Theory of Disease.**"

The word "**Contagion**"[a] has been employed in two very different senses, which it is here necessary to discriminate. In the first place, it is a comprehensive term which includes, and is used to express, all the different agencies by which zymotic, or—as they are now generally called—micro-parasitic, diseases are known to

[a] Lat. *Contagio*; or in the poetical form, *Contagium*; from *cum*, and *tango*, *I touch* (*contingo*).

spread. But again the "contagion," or "contagium," of a disease is the poisonous principle, or **virus**, which when introduced into the system from without, by inoculation, or inhalation, or in the act of swallowing, is capable—under favourable circumstances—of multiplying itself and setting up the phenomena of the disease in question. It is to be noted that each disease has its own peculiar or special poison or virus, which is accordingly called "**specific.**" In recent years the generic term "**infective**" has been applied to any form of disease which is believed to be due to a specific cause. The word exactly represents the German expression, "Infections-krankheit."

The processes of typical infective diseases in the human body present many analogies to those of **fermentation** in an organic liquid. Hence the virus or contagium is often called the "ferment," or "leaven," or "zyme" (Gk. ζύμη), and the resulting diseases are called **zymotic.**

The analogy will become very apparent if we quote a typical example of fermentation. When the yeast plant (*Saccharomyces*, or *Torula cerevisiæ*) obtains access to a saccharine fluid and the temperature is suitable, the cells of the yeast rapidly multiply, and after a certain time—which corresponds with the **period of incubation** in an infective disease—changes are produced in the saccharine liquid, evidenced by the formation of alcohol and carbonic dioxide (CO_2), which eventually render it incapable of being further acted upon by that particular ferment.

According to the now generally accepted "Germ Theory," the contagia of the so-called Zymotic Diseases are microscopic living particles probably of a vegetable nature, which, for the most part, belong to the class of micro-organisms to which the name **Schizomycetes**, or **Fission-fungi** (Germ. **Spaltpilze**), has been given by Nägeli. That investigator divided the fungi engaged in producing fermentation and decomposition (or putrefactive fermentation) into—

Hyphomycetes, *Mucorini*, or "Moulds" (Germ. *Fadenpilze*).
Saccharomycetes, or "Yeasts" (Germ. *Hefenpilze*).
Schizomycetes, or "Fission-fungi" (Germ. *Spaltpilze*).

To the class of the "Fission-fungi," or "Cleft-fungi" (*Schizomycetes*[a]), belong the **Cocci, Bacteria, Leptotriches,** and **Cladotriches** (Zopf). The fission-fungi do not contain chlorophyll, and are composed of a kind of protoplasm, called **mycoproteïn** by Nencki, with an investing membrane chiefly of cellulose. Many of these micro-organisms—or, as the French call them, **microbes**[b]—exist in enormous numbers in the air, water, and soil in every region and climate, and to them, in conjunction with yeasts and moulds, the fermentative and putrefactive changes, to which organic matter is liable, are due.

Certain of these micro-organisms or microbes live on dead organic matter only, and are not, therefore, concerned in the *direct* production of disease. These are called **Non-pathogenic Microbes,** or **Saprophytes.**[c] Included amongst them are various micrococci, bacilli, &c.

Others of these micro-organisms prey either on living organic matter only, or on both living and dead organic matter. These are the disease-producing, or **Pathogenic Microbes,** or **Parasites.**[d] They, also, include various micrococci, bacilli, &c.

As true parasitic micro-organisms do not exist outside the living body, we are not at present in a position to study them by means of cultivations in artificial media such as gelatine, Agar-Agar (or Japanese gelatine), animal broth, and so on. To this group belong the micro-organisms of smallpox, cowpox, whooping-cough, measles, &c., of the intimate nature of which we at present of necessity know but little. These microbes are called "**Obligate Parasites.**"

Many pathogenic or parasitic organisms, on the other hand, do possess the power of attacking non-living matter, so affording evidence of a certain amount of saprophytism. Hence we are able to cultivate these outside the living body in artificial media. This class embraces the microbes of tubercle, diphtheria, anthrax, erysipelas, tetanus, and so on. They are called "**Facultative Parasites.**"

[a] Gk. σχίζω, *I cleave*; μύκης, *a fungus*, or *mushroom*.

[b] Gk. μικρός, *small*; βίος, *life*.

[c] Gk. σαπρός, *rotten*; φυτόν, *a plant*.

[d] Gk. παράσιτος, *one who eats beside* or *at the table of another*; *one who lives at another's expense*.

Another classification of the pathogenic or parasitic microbes is into—

(1) **Non-infectious**, but **toxic, organisms**; and

(2) **Truly infectious organisms.**

The latter, which include the microbes of some of the specific fevers, are found to pervade the blood and every organ of the body. The former, of which the microbes of diphtheria and tetanus are typical examples, act on the organism through certain poisonous principles which they produce during their lifetime or by their death. These poisonous principles are of an alkaloidal nature, and are called **Toxins** or **Ptomaïns.**[a]

Dr. Edgar M. Crookshank, in his *Manual of Bacteriology*,[b] says: "Bacteria may be grouped together according to the changes produced in the media in which they grow. Thus we have **pigment-forming, fermentative, putrefactive,** and **pathogenic bacteria.**"

The pigment-forming, or chromogenic bacteria, produce various colour-stuffs. Thus, the *Micrococcus prodigiosus* forms a blood-red growth in bread, potato, and other media. *Bacillus violaceus* produces a violet pigment, and *B. pyocyaneus* a delicate green. The magenta micrococcus forms a brilliant magenta growth, with a metallic lustre. In this species the pigment is retained within the cells. The surface of sterilised potato is a very favourable medium for the cultivation of these most interesting chromogenic micro-organisms.

The fermentative bacteria are also termed zymogenic, the putrefactive are saprogenic, and the pathogenic are the disease-producing microbes.

Some micro-organisms require free access of oxygen for their growth, and so they received the name of **aërobic**[c] from Pasteur. Others grow and flourish without free oxygen, and are **anaërobic**[d] (Pasteur). All require for their growth carbon, nitrogen, and

[a] Gk. *τὸ τόξικον, arrow poison; πτῶμα* (from *πίπτω*, I fall), *a fallen, i.e., a dead body.*

[b] London: H. K. Lewis. Second Edition. 1887. Pages 141 and 142.

[c] Gk. *ἀήρ, air; βίος, life.*

[d] Gk. *ἀν-*, the negative prefix in Greek.

water, as well as a certain temperature, which varies from 18° C. to 40° C. (64·4° F. to 104° F.) in the case of the pathogenic microbes. While all these microbes are capable of disintegrating organic combinations containing nitrogen, they, in their turn, help to produce certain definite chemical products for definite species.

These microbes, whether pathogenic or non-pathogenic, whether parasitic or saprophytic, grow and multiply enormously; but they are very short-lived, and are easily destroyed, perishing in vast numbers, often in consequence of the chemical compounds (**toxins** or **ptomaïns**) to which they themselves give rise.

How, then, are the several species preserved from extinction? By **sporulation.** Spores, the immature forms of the adult species, can resist extremes of temperature and of desiccation, or drying, which are destructive to the fully-developed organisms. So retentive of life are these spores that they are called "**Resting Spores.**" They represent seeds capable of retaining and germinating into microbes even after what would appear the most damaging influences—such as long lapse of time, drying, extremes of heat or cold, chemical reagents, and the like.

These spores are generally spherical when immature, oval when fully developed. They are of a glistening appearance, and take the ordinary dyes with difficulty, or not at all.

This spore-formation closes the cycle of the life-history of the microbe. The theory of sporulation explains those puzzling instances in which infectious diseases have been supposed to have arisen independently of a previous case—that is, spontaneously, or *de novo.*

The pathogenic organism of each of the infective diseases is **specific**—that is, it is peculiar to the particular disease, standing in a causal relation to it and to no other malady. Thus, the microbe of scarlet fever gives rise to scarlet fever and to it alone; that of smallpox, to smallpox and to it alone—"the seed, as a gardener would say, comes up true" (Dr. Robert Liveing). Hence these maladies are spoken of as "**specific**" or "**essential febrile diseases.**" This is an established clinical and pathological fact.

In each special, or specific, disease the virus, or contagium,

multiplies chiefly in those tissues—the mucous and epithelial—which are more especially subject to its action, and the infection is cast off from the body in large part with the excretions or secretions of those tissues. For example, the poison or virus of typhoid fever passes out of the system chiefly in the evacuations from the bowels; that of scarlet fever passes off chiefly by or from the skin. In the former case the contagium is propagated, not so much through the air as by means of water, milk, or other food to which the evacuations have gained access. In the latter case the contagium may be dormant in the clothes or furniture of the sick room for long periods, or it may be carried through the air from the diseased to the healthy.

In the case of some of the infective diseases the contagium does not seem to be capable of retaining an independent existence outside the animal body—the infective microbe is solely parasitic, not in any sense saprophytic. In these maladies the infection (or contagium) is conveyed by direct contact—that is, by inoculation from the diseased body into the tissues of the healthy: examples of this are—syphilis, gonorrhœa, hydrophobia, and glanders.

These infective diseases, then, are sometimes further artificially classified as **contagious**, or those transmitted only by direct inoculation or immediate contact, and **infectious**, or those transmitted through the air or by other carriers of infection (**fomites**[a]), such as wearing apparel, furniture, bedclothes, dust, skin-particles, fæces (as in enteric fever and cholera), and so on. It will be necessary to revert to this subject in the next chapter.

[a] Lat. *fomes, fomitis, touch-wood, tinder* (from *foveo, I warm*).

CHAPTER III.

Micro-parasitic Diseases.

Substitution of this term for "Zymotic Diseases."—"Infective Diseases."—Liebermeister's Classification: 1, Miasmatic; 2, Miasmatic-Contagious; 3, Parasitic-Contagious (Infectious or Contagious).—Endemic, Epidemic, Sporadic, and Pandemic: definition of these terms.—Theories of Immunity.—Natural and Acquired Immunity.—Metschnikoff's theory of Phagocytosis.—Phagocytes (Germ. "Fresszellen").—"Micro-Strife" (A. Wynter Blyth).—Acquired Immunity.—Classification of Micro-parasitic Febrile Diseases: 1, The Eruptive Fevers or Exanthemata; 2, The Continued Fevers; 3, The Intermittent Fevers.—Cyclical course of the Specific or Essential Fevers: 1, Incubation; 2, Invasion; 3, Eruption; 4, Defervescence; 5, Desquamation; 6, Convalescence.—Quarantine.—Isolation.—Influence of the Micro-parasitic Diseases on each other.

It was shown in the preceding chapter that each of the so-called Zymotic Diseases has its own peculiar disease-producing or pathogenic micro-organism, which is properly called a "parasite." Hence the term "**Micro-parasitic Diseases**" is now being adopted in place of the older expression "Zymotic Diseases." It was further shown that each of these affections has its own peculiar or special poison or virus, so that they are called "**Infective Diseases**" (Germ. *Infections-krankheiten*). The fever which attends them is in a sense not symptomatic: it is primary, or idiopathic, and thus we come to speak of them as the "**Essential**" or "**Specific Fevers.**"

Probably the best classification of these infective, or micro-parasitic, diseases, would be that adopted by the German School, as represented by Liebermeister:—

(1) Miasmatic.

(2) Miasmatic-Contagious.

(3) Parasitic-Contagious; that is, Infectious or Contagious.

1. **Miasmatic Diseases** are those which result from the introduction of a specific poison into the body from without—the poison being developed outside of the body, usually in marshy or swampy districts, or where there is an insufficiency of healthy vegetation and a waterlogged soil. The "morbific agent" is almost certainly

microbic.[a] It is usually inhaled, but may be swallowed with food or water exposed to marsh air. It is incapable, apparently, of reproduction in the animal body, it is not propagated by an infected person, and it produces in the system solely its own specific effects—fever, enlargement of the spleen, and a peculiar cachexia. This poison of miasmatic diseases is technically called **Malaria.**

Typical miasmatic diseases are "Intermittent Fever" or "Ague," in which periods of complete apyrexia or freedom from fever occur, and "Remittent Fever" or "Jungle Fever," in which the fever *remits* indeed from time to time, but does not absolutely disappear. Both these forms of disease are known as Malarial or Paludal Fevers (Latin: *Palus, paludis*, a swamp). They do not more immediately concern our present purpose.

2. **Miasmatic-Contagious Diseases** are those like enteric or typhoid fever, cholera, and perhaps influenza, which, although spreading through human intercourse, are thought not to be communicable *directly* from one person to another. This, as Liebermeister supposed, is in consequence of the inadequacy of the contagium to propagate the disease without undergoing some further change outside the body and so becoming a **miasm.**

3. **Parasitic-Contagious Diseases** are those which are capable of being transmitted from one person to another, or from one animal to another of either the same or another species, either by direct contact, or through the agency of air or water, or by other carriers of infection, technically called **fomites.**[b] In the former case, the diseases are strictly and truly **contagious**, like syphilis or hydrophobia; in the latter case they are described as "**common infectious diseases,**" like smallpox, measles, scarlet fever, typhus, and so on.

As regards their manner of prevalence, zymotic or micro-parasitic diseases are spoken of as **endemic, epidemic,** or **sporadic.** To these terms we may add a fourth, **pandemic,** which was used by Dr. Robert Lawson, Inspector-General of Hospitals, in connection with wide-spread epidemics, particularly of cholera.

[a] Klebs and Tommasi-Crudeli: *Bacillus malariæ.* And, more recently, Celli and Marchiafava: *Plasmodium malariæ.* See *Fortschritte der Medicin,* 1883 and 1885.

[b] See footnote on page 17.

1. **Endemic Diseases**[a] are those which arise and prevail in a given country, place, or neighbourhood, under conditions, circumstances, or agencies, peculiar to the locality in question, and which favour the development of the said diseases. For examples, we may instance among miasmatic diseases intermittent fever, or ague, and remittent fever, or jungle-fever; among the miasmatic-contagious diseases, cholera (in the Valley of the Ganges), enteric fever, and perhaps influenza.

2. **Epidemic Diseases**[b] are those which prevail among a people or community at a given time, and are produced by some special cause or causes not naturally, or generally, present in the affected locality. Such diseases spread more or less rapidly, by contagion or otherwise, so as to incapacitate or destroy great numbers of the people.

In illustration of the extent to which an epidemic may incapacitate a community we need only take the unhappily too familiar instance of **Influenza**, so expressively called by the French "La Grippe." In 1510, an epidemic of this disease swept over Europe, "not missing a family and scarce a person."[c] Mercatus asserts that before the beginning of autumn, 1557, influenza attacked all parts of Spain *at once*, so that the greatest part of the population in that kingdom were seized with it almost on the same day.[d] In 1782, again, Admiral Kempenfelt sailed from Spithead with a squadron on May 2. Influenza broke out on the 29th, and so many men were in consequence rendered incapable of duty that the whole squadron had to return into port about the second week in June.[e]

But, unfortunately, epidemic visitations destroy life as well as incapacitate the living. It is true that modern Preventive Medicine has lessened the evil to a remarkable extent, but nevertheless

[a] Gk. *ἐν, in* or *among*; *δῆμος, the people—ἐνδημέω, to live at* or *in a place.*

[b] Gk. *ἔπι, upon*; *δῆμος, the people—ἐμιδημέω, to be among a people, to be prevalent* or *epidemic.*—Hippocrates.

[c] Short's *Chronological History of the Weather.* Vol. I., page 204. Also *Dict. des Sciences méd.* Art. "*Grippe.*"

[d] See Report, by Dr. Glass, in Lettsom's Memoir of Dr. Fothergill. Quarto. Page 625.

[e] *Lectures on the Principles and Practice of Physic.* By Sir Thomas Watson, Bart., M.D. Third Edition. Vol. II., p. 41.

the death-roll from epidemic disease remains an *opprobrium medicinæ*. In 1871–72 small-pox caused 1,647 deaths in Dublin alone. The outbreaks of influenza in 1890 and 1891 raised the death-rate in many American and British towns to an appalling figure. It is estimated that the plague of London in 1666 slew 70,000 persons; but even this fades into insignificance when contrasted with the plague of 1348—a pestilence which carried off 100,000 souls in London alone and 40,000,000 from all Europe.

3. **Sporadic**[a] is a term applied to solitary, isolated, or scattered cases of specific fevers or micro-parasitic diseases, which occur independently of any known epidemic or contagious influences from accidental or undiscovered causes. Such outbreaks often take place in the wake of a past epidemic.

4. The term **Pandemic**[b] is used when a disease prevails in succession in a very large number of countries; when it sweeps, like a great wave, across large areas of the earth's surface: thus, in 1847–48 and again in 1889–91, there was a pandemic prevalence of influenza; and in 1869–73, a similar pandemic prevalence of smallpox was observed.

Theories of Immunity.—One of the most interesting facts connected with the micro-parasitic diseases is the **immunity** against a subsequent attack which their victims in most cases acquire. This freedom from a second attack by reason of the influence exerted by a first attack is not easily explained; nor is it observed to the same degree after the different diseases. Thus, an attack of smallpox confers almost absolute immunity from a second visitation. The protective efficacy of an attack of measles is less complete, a second attack of scarlatina is not uncommon, while an attack of erysipelas actually seems to predispose to a second. Among the Continued Fevers, the same rule of immunity in general holds good, and yet Dr. Charles Murchison in his own person suffered from two unequivocal attacks of maculated typhus.[c]

[a] Gk. *σπόρας, scattered* (from *σπείρω, I sow*).

[b] Gk. *πᾶς, all; δῆμος, the people.*

[c] *A Treatise on the Continued Fevers of Great Britain.* By Charles Murchison, M.D., LL.D., F.R.S. Third Edition, edited by W. Cayley, M.D., F.R.C.P. London: Longmans, Green, & Co. 1884. Page 84.

The importance of investigating this question of **acquired immunity** will be apparent when we reflect that to secure this immunity has been the object of the preventive treatment of zymotic disease by *inoculation* of an attenuated virus—that is, a virus of diminished or weakened poisonous properties—from the days of Lady Mary Wortley Montague[a] to those of Pasteur, Toussaint, and Koch.

The question is still a vexed one. It is well discussed by Dr. Edgar Crookshank in his "Manual of Bacteriology."[b] One theory is that during an attack of an infective disease some chemical substance—a toxin or a ptomaïn—is secreted, which remaining in the system defeats a future onset of the specific microbe of the disease.

Again, it is suggested that at their first invasion of the animal system the micro-organisms exhaust the pabulum in the blood and tissues which they require for their nutrition, and so leave the soil impoverished and unsuitable for the development hereafter of their successors of the same species.

Another theory is that the tissues are endowed with some power of vital resistance to the development of micro-organisms, and that this power exists in some species and individuals to a high degree, thus constituting what is called "**natural immunity.**" This theory, however, does not explain the acquired immunity of which we are now speaking, just as the second theory does not explain natural immunity. As Dr. Crookshank well remarks: "One would expect that the vital resistance would invariably be lowered by a previous attack, and increased liability be the constant result."

One more theory remains, and it constitutes a very romance in Pathology. It is the theory of **Phagocytosis**[c] and its results. In 1884, Elias Metschnikoff, of Odessa, communicated to Virchow's *Archiv. für pathologische Anatomie und Physiologie und für klinische Medicin* a paper on a "Blastomycetic Disease in Daphnia[d]—a

[a] See Chapter X., page 103.

[b] Second Edition. London: H. K. Lewis. 1887. Pages 169, *et seq.*

[c] Gk. *φαγέω, I devour; κύτος, a hollow*, hence, *a cell.* "Phagocytes" are "devouring cells."

[d] Ueber eine Sprosspilzkrankheit der Daphnien. Beitrag zur Lehre über den Kampf der Phagocyten gegen Krankheitserreger.

contribution to our knowledge of the War of the Phagocytes against Pathogenic Microbes." *Daphnia*, or "water-fleas," are a genus of small fresh-water entomostracans, one of the sub-classes of the *Crustacea*. Metschnikoff followed up this paper by a further communication on "The Relation of Phagocytes to the *Bacillus anthracis*."[a] According to the author, the leucocytes or white-blood corpuscles appear to have the power of destroying bacteria in some cases. If the bacilli of anthrax are inoculated in the frog, the white blood cells are seen to incorporate and destroy them until they entirely disappear, and the animal is not affected. If, however, the animal is kept at a high temperature after inoculation, the bacilli increase so rapidly that they gain the upper hand over the leucocytes, and the animal sickens and succumbs. In the septicæmia of mice the white blood cells are attacked and disintegrated by the bacilli in a like manner.

The leucocytes, then, may be regarded as a soldier-like community, one of whose special offices is to fight to the death the invading hordes of pathogenic microbes. Hence Metschnikoff's expressive term—"Der Kampf der Fresszellen (Phagocyten) gegen Krankheitserreger"—"The battle of the devouring cells against the causes of disease." These fighting leucocytes have been called **phagocytes**. They are endowed with powers of movement, and, amœba-like, are capable of pushing out and withdrawing portions of their protean bodies. Into the retracted portion of one of these phagocytes a microbe is received. By it it is finally enveloped, becomes cloudy, and finally disappears by complete absorption or digestion. When, on the other hand, victory inclines to the invader, then there is sufficient disturbance of function to produce pyrexia and other febrile symptoms.

Mr. A. Wynter Blyth, whose graphic description of "Micro-Strife"[b] we have closely followed, says that "by the same theory

[a] Ueber die Beziehung der Phagocyten zu Milzbrandbacillen. *Virchow's Archiv.* Vol. XCVII., page 502. 1884. "Anthrax" is also called "Splenic Fever," "Malignant Pustule," or "Wool-sorter's Disease."

[b] *A Manual of Public Health.* London: Macmillan & Co. 1890. Pp. 363, *et seq.*

the curious fact that an animal body having once recovered from one of these micro-parasitic diseases obtains a shorter or longer immunity is capable of a plausible explanation. The macrophages [phagocytes] have a rapid cycle of existence, a few hours may represent several generations, so that acquired properties are rapidly transmitted; those poisoned by the excretion of pathogenic microbes perish, those that more or less effectually resist, continue to live and propagate, until by a repetition again and again of this process, the body may be full of resistant living particles, and the foreign tribe is annihilated or expelled. If now a second inimical colony of the same kind obtains access to the body, it meets with the fighting descendants of the old heroes, and the attack is immediately repulsed, and this is the nature of protection conferred from a recovery from a first attack. Since it would seem that the weapon of the microbe is its venom, it is not surprising that the macrophages may be educated in their resistance by being dosed with the excretory products of pathogenic organisms; the method of education being—first, doses feeble in either quantity or strength, to be followed by successive doses of gradually increasing virulence, until no health disturbance is produced by an otherwise mortal dose."

Mr. Blyth goes on to give a practical example in illustration of this theory of acquired immunity. "The efficacy," he says, "of ordinary vaccination against smallpox may be explained on the theory that vaccine contains a number of degenerate colonies of the same genus as the smallpox microbe—these are conquered with comparative ease; nevertheless, the result is that a race of macrophages is left, which from their education successfully cope with the virulence of true variola." [a]

Such is the ingenious and suggestive theory of phagocytosis and its relation to acquired immunity.

Certain of the infective, or micro-parasitic, diseases are characterised by the appearance at a definite period in their course of a rash on the skin, or "an eruption." Others run a continued febrile course from the beginning, while in a third group periods of complete or partial freedom from fever—apyrexia—occur from time to time.

[a] *Loc. cit.* Page 365.

Bearing these clinical facts in mind we may adopt the following classification of micro-parasitic febrile diseases, exclusive of septicæmia:—

I. The Eruptive Fevers, or Exanthemata—

1. Variola, or Smallpox.
2. Varicella, or Chickenpox.
3. Morbilli, or Measles.
4. Scarlatina, or Scarlet Fever.
5. Epidemic Roseola, or Epidemic Rose Rash (Germ. Rötheln).
6. Erysipelas, or St. Anthony's Fire, or Rose.

II. The Continued Fevers—

1. Febricula, Simple Fever, or Ephemeral Fever.
   (This is sporadic in its mode of prevalence, and is by some authors supposed to be of non-specific origin).
2. Enteric, Typhoid, or Pythogenic Fever—
   (Specific, endemic, and locally epidemic).
3. Typhus Fever—
   (Specific, epidemic, and infectious).
4. Spirillum Fever, Famine Fever, or Relapsing Fever—
   (Specific, epidemic, and infectious).

III. The Intermittent Fevers—

1. Malarial Fever, or Ague—
   (Quotidian, Tertian, Quartan).
2. Remittent Fever, or Jungle Fever.

As regards the **behaviour of the temperature** in the foregoing diseases, the following terms are used:—

1. The **onset** or **invasion**, during which the temperature rises more or less rapidly and evenly. This is called by Wunderlich the initial period or *pyrogenetic stage.*[a]
2. The **acme**, or **fastidium**, during which the thermometer reads highest. The period of full development of the fever accompanying the disease.
3. The **cooling**, or **defervescence**, during which the tempera-

[a] *On the Temperature in Diseases: a Manual of Medical Thermometry.* By Dr. C. A. Wunderlich. Translated by W. Bathurst Woodman, M.D. London: The New Sydenham Society. 1871. Page 9.

ture falls more or less rapidly and evenly. The period of return to normal temperature.

4. The **crisis**, characterised by a sudden and abrupt fall of temperature and an amelioration in the other symptoms. This is rapid defervescence.
5. The **lysis**, during which the temperature gives way slowly and gradually, with morning remissions and evening exacerbations. This is protracted defervescence.
6. The **collapse stage**, characterised by marked subnormal temperature. This stage, although seemingly dangerous, is deceptive, and commonly ends in recovery.
7. The **hectic state**, denoted by habitual fever with large daily range of temperature. The febrile type is usually remittent, with one or two exacerbations in the course of every day, and is chronic or of long duration.

For convenience of description the cyclical course run by these specific fevers is portioned off, or mapped out, into the stages which follow—

1. **Incubation**—the "hatching" stage is sometimes spoken of as **the latent period**, because as a rule it is characterised by no special symptoms. The period of incubation is the time which elapses between the exposure to the poison of the disease and its reception into the system and the appearance of the earliest symptoms. This stage varies greatly in the different infectious fevers: its probable duration in each case will be found under the heading of each disorder, as well as in the Table at the close of this chapter.
2. **Invasion**—the stage during which the special symptoms, and in particular the fever, are developing. For this reason it is called by Wunderlich[a] the **pyrogenetic stage**, and in the case of the eruptive fevers, as it precedes, or *runs before*, the appearance of the rash, it is spoken of as the **prodromal**[b] **stage**. Like the period

[a] *Medical Thermometry.* New Syd. Soc. Pages 9,243.

[b] Gk. *πρόδρομος, a running before, a going in advance.*

of incubation, it also varies considerably in duration in the several diseases, begins with the earliest symptoms and terminates with the fastigium of the disorder, or (in the case of the eruptive fevers) with the appearance of the rash.

3. **Eruption**—the stage of the rash.

4. **Defervescence**[a]—the cooling stage, already described. The German equivalent, *Fieberabnahme*, is very expressive.

5. **Desquamation**[b]—the *peeling* stage (Germ. *Abschuppung* or *Absplitterung*), during which the skin is shed either in small particles, like fine bran, as in measles—when it is spoken of as "branny" or "furfuraceous" (Lat. *furfur*, *bran*) desquamation; or in flakes, as in scarlatina, when it is called "flaky," "membranous," or "lamellar" (Lat. *lamella*, *a small plate*) desquamation.

6. **Convalescence**[c]—restoration to health and strength—a stage which lasts from the termination of the disease and the final disappearance of its symptoms to the recovery of the strength lost through it by the patient.

The following Table will prove of use in connection with the foregoing definitions. The terms "Quarantine" and "Isolation," which head the last two columns in the Table, require explanation. "**Quarantine** (no longer used with its original signification of a *forty days'* [Fr. *Quarante*, forty] detention) means the segregation of *possibly* infected persons until after the period has elapsed at which they would (if infected) develop characteristic signs of the disease. **Isolation**, on the contrary, implies the continued separa-

[a] Lat. *defervesco, to cease boiling, leave off raging, cool down*—a Ciceronian word.

[b] Lat. *desquamo, to scale off, to peel off* (*Squama, a scale*, or *flake*).

[c] Lat. *convalesco, to recover from a disease, to regain health, to grow strong, gain strength.* These are all classical meanings of the word, frequently met with.

tion of the infectious patient from the healthy until after he has ceased to be infectious."[a]

It is right to mention that some authors include in the period of incubation the invasion-stage. According to them, therefore, incubation is divided into two stages, those of *latency* and of *invasion*. In this book incubation and latency are treated as synonymous terms, and invasion is treated as a separate and distinct period.

The doctrine promulgated by John Hunter[b] in the eighteenth century, that the presence of one of the micro-parasitic diseases protects the infected individual from an attack of another disease belonging to the same group cannot be entertained or accepted for a moment. All clinical experience is opposed to such a view, and in the following pages allusion will be made from time to time to instances of individuals contracting an eruptive or a continued fever while actually under treatment for another febrile disease.

In fact, it may be laid down as a general rule that the presence of one infective disease increases rather than diminishes the susceptibility of the sufferer to another by impairing his health and so lessening his powers of resistance. The curious fact that vaccination seems often to influence the progress of whooping-cough for the better does not militate against the view here put forward, for if it did the operation of vaccination would not have proved successful in such cases.[c]

[a] See Art. "*Disinfection*," by C. E. Shelly, M.A., M.D., in Fowler's *Dictionary of Practical Medicine*. London : J. & A. Churchill. 1890.

[b] Hunter's *Works*, Palmer's Edition. Vol. I., page 313. Vol. III., page. 4.

[c] Cf. Dr. Cachazo (*Wien. med. Blätter*, October 16, 1890), Dr. Emile Müller (*Gazette Médical de Strasbourg*, July 1, 1891), and Dr. Thomas Purcell (*Brit. Med. Journ.*, August 29, 1891, page 498).

TABLE I.

	Disease	Incubation	Invasion	Eruption	Mode of Defervescence	Duration of Fever	Form of Desquamation	Quarantine	Isolation
		Days	Days			Days			
The Eruptive Fevers, or Exanthemata	Smallpox	12	3	On 3rd day	Lysis	12–18	Flaky	18 days	Until every scab has fallen off
	Chickenpox	12	1–3	On 1st, 2nd, or 3rd day	Crisis	6–12	Branny	18 days	Until every scab has fallen off
	Measles	10 (7 to 14 days)	4	On 4th or 5th day	Crisis	8	Branny	16 days	4 weeks
	Scarlatina	1–7	1	On 1st or 2nd day	Lysis	9	Flaky	14 days	6 weeks to 2 months
	Rötheln	10–14	1–3	On 1st, 2nd, or 3rd day	Crisis	3	Branny	16 days	2 to 3 weeks
	Erysipelas	1–9	1–3	On 1st, 2nd, or 3rd day	Lysis	Var.	Branny or Flaky	12 days	Complete convalescence
The Continued Fevers	Typhoid	10–15 (2 to 28 days)	1 or more days	From 7th to 12th day, inclusive	Lysis	21–28 (24·3)	Branny	At least 10 days	Established apyrexia
	Typhus	12 (1 day to 20 days)	4–5	On 4th or 5th day	Crisis	14 (13·43)	Branny	14 days	Complete convalescence
	Relapsing or Spirillum	5–8 (1 day to 14 days)	1	None	Crisis	18[a] (17·9)	Branny	24 days	Complete convalescence

[a] First Paroxysm = 5·96 days; Intermission = 7·82 days; Second Paroxysm = 3·45 days.—*Murchison.*

CHAPTER IV.

The General Principles of Treatment of the Eruptive and Continued Fevers.

Preventable Diseases.—Preventive Treatment, or Prophylaxis.—Disinfection:—Antiseptics, Disinfectants, Germicides, and Deodorants.—Dr. Shelly's Classification of Disinfectants.—Dr. Emerson Reynolds' General Plan of Disinfection.—Official Regulations for Disinfection adopted in Berlin.—Disinfection within the Living Body.—Receptivity.—Exciting and Predisposing Causes of Disease.—Acquired and Natural, or Hereditary, Predisposition to Disease.—The chief Predisposing Causes of Febrile Disorders.

Part I.—Preventive Treatment, or Prophylaxis.

Having regard to the view that—(1) the diseases we are considering result from the introduction into the system of a specific micro-organism, which either acts as a poison itself or produces in the body a toxic agent of a chemical nature, while (2) the "susceptibility" or "receptivity" of the infected individual varies without limit, according to his healthy or unhealthy surroundings, his state of health at the time, and a natural or acquired immunity—we are justified in speaking of specific infectious (infective) diseases as **preventable.**

As this is so, it follows that the subject of treatment arranges itself under two headings—**preventive** and **curative.** Both these terms must be used with a certain amount of reservation—in a general rather than in a precise, an abstract, or an absolute sense.

From this point of view we may adopt the terms "**Preventive Medicine**" and "**Prophylaxis**"[a] with all reverence and with the utmost propriety.

The **prophylaxis** of the acute specific fevers consists in—(1) the destruction of the virus or contagium before it can enter the system; (2) the adoption of measures which will have the effect of lessening the receptivity of the individual, or—in other words—

[a] Gk. προφυλάσσω, fut. προφυλάξω, *to keep guard before, to guard against, to be on one's guard against;* and, in medical sense, *to take precautions against.*—(Plutarch).

of increasing his powers of resistance; and (3) the strict enforcement of rules relating to quarantine and isolation.

I. The **destruction of the virus** or **contagium** of infectious disorders is achieved by **disinfection**, using the term in its strict etymological sense to denote any process by which the contagium of a given disease may be destroyed or rendered inert, so as to render impossible the spreading of that disease.

Antiseptics must be distinguished from **disinfectants**. An **antiseptic** is a substance which prevents or retards putrefaction—that is, the decomposition of animal or vegetable bodies accompanied by the evolution of offensive gases.

A **true disinfectant**, on the contrary, is a substance which exerts a destructive action upon minute living organisms—the microbes of which we have been speaking so often, and upon dead organic matter.

A **germicide**, again, is a disinfectant which, when added, in not less than a certain proportion, to a culture of the particular microbe under observation, kills both it and its spores outright.

Apart from either antiseptics or disinfectants, we have a third class of chemical agents which remove bad smells, and hence are called **deodorants**. Although many substances are at the same time antiseptics, deodorants, and disinfectants—for example, chlorine, ozone, and some of the metallic salts—yet it would be a fatal error to suppose that the removal of a bad smell, probably the result of sepsis, or putrefaction, can in any sense be regarded as evidence of true disinfection. As a matter of fact, the volatile poisons of the infectious fevers are often odourless, and mere deodorants are not called for in dealing with them.

Dr. C. E. Shelly classifies disinfectants according to their mode of action as—

A. Oxidising agents, including (1) those which oxidise directly—*e.g.*, ozone, potassium permanganate, &c., and (2) those which oxidise indirectly by the abstraction of hydrogen—*e.g.*, chlorine, chloride of lime, &c.

B. De-oxidising agents—*e.g.*, carbolic acid, sulphurous acid gas, &c.

C. Substances which destroy the pathogenic virus by some other form of physical action—*e.g.*, corrosive sublimate, heat, &c.

"It is obvious," writes Dr. Shelly, "that the members of groups A and B must not be used together, since their modes of action are chemically antagonistic; thus, carbolic acid may be employed in conjunction with the fumes of burning sulphur, but neither should be used with Condy's fluid, chlorine gas, or sanitas.

"The contagia of the various forms of infection vary greatly as regards their virulence and their power of resistance to destructive agencies; the spores of *Bacillus anthracis* and the virus of scarlatina are tenacious of vitality, whilst the infection of measles is short-lived and can be destroyed by free exposure to fresh air and bright sunshine."[a] This last remark applies to the contagium of typhus fever also.

Dr. Emerson Reynolds, F.R.S., Professor of Chemistry in the University of Dublin, some years ago laid down the following **general plan of disinfection** in dealing with a case of infectious disease[b]:—

"The patient's stools and vomited matter should be immediately treated by free sprinkling with a roughly-powdered mixture of four parts of common sulphate of iron (copperas) and one part of sulphate of copper (blue stone). If necessary a little water may be thrown into the vessels, in order to facilitate solution of the salts. All articles of clothing, however trifling, pieces of cloth, books, toys, &c., used by the patient, should be set aside, the worthless articles and those of little value to be burned as soon as possible, and the clothing, &c., to be treated as we shall describe further on. Of all the bodies used to 'purify' the atmosphere of a sick chamber, chloride of lime, made into a paste with water and spread on plates, is, perhaps, least open to objection. If the smell proves disagreeable to a patient, 'Condy's fluid' may be substituted, though the non-volatility of the active ingredient (permanganate of potassium) renders it less effective. It is well to remember that, when used

[a] Art. "*Disinfection*," in Fowler's *Dictionary of Practical Medicine*.

[b] Cf. *Manual of Public Health for Ireland*. Dublin: Fannin & Co. 1875. Pages 263, *et seq*.

in this manner, both the above-named bodies probably act only as deodorants. Perfect cleanliness in the sick chamber and good ventilation are of vastly greater importance than the use of any known disinfectant that could be safely employed under the circumstances. But, if it be desired to try general disinfection, we decidedly give the preference to the occasional burning of ordinary incense, mixed with about one-sixth of its weight of benzoic acid, in different parts of the house, while carbolic or, better still, cresylic acid may be used in water-closets, &c. At the termination of an illness, the room or rooms occupied by the patient should be cleared of furniture, carpeting, curtains, &c., the paper removed from the walls, the chimney stopped, and windows well closed. A *large* earthenware basin is then placed in the centre of the room. If the apartment be of moderate size, put half a pound of copper wire, cut in short pieces, into the basin, and pour upon it three times its weight of aquafortis (strong commercial nitric acid), diluted with its own bulk of water. Action speedily commences, and ruddy fumes are evolved, consisting of oxides of nitrogen. The door of the room is now carefully closed, and not opened for a few days; then a shovel, containing a few pieces of red-hot coal, is brought into the room, and two or three ounces of sulphur are thrown on the coals. The sulphur burns, and produces abundance of sulphur dioxide, commonly called sulphurous acid; the door is again secured. After a day or two the room may be entered, the windows thrown open, the ceilings whitened, the walls papered, and the woodwork and floors washed with water containing a teaspoonful of benzoic acid per gallon.

"Articles of clothing which will admit of the treatment should be boiled with water first, and then be steeped for some days in fresh water, containing benzoic acid in the proportion of a large teaspoonful of the common acid for every five gallons of water. Let the articles then be boiled in the steep-water, and next washed as usual.

"Although the best and safest course unquestionably is to burn all clothes, bedding, hangings, &c., we may attempt the destruction of the contagion particles by heating to a much higher temperature than that of boiling water, though not to such a temperature as to

destroy the material operated upon. The temperature of 260° F. is about the highest that ordinary fabrics can bear for any length of time, and this heat may be applied by enclosing the articles in a large oven of brick or iron, raised to the required temperature by a coil of metal pipe, through which more or less of the hot gases from a small furnace can be directed, according to the temperature desired. The precise degree can be determined by a thermometer, whose stem passes through the door of the chamber. A tube should convey the effluvia of the clothes from the upper part of the oven to the ashpit of a furnace, in order that the gaseous or other exhalations may be burnt, and so prevented from polluting the atmosphere.

"It is comparatively difficult to regulate the temperature of a large chamber heated in this way, or to ensure the thorough treatment of the articles enclosed in it. We would much prefer to use several small steam-tight chambers, and to connect them directly with a boiler. The steam should be allowed to pass freely into each disinfecting chamber, from which it might issue at a pressure of about 20 lbs. A temperature of 230° F. is supposed to destroy all living organisms; but a temperature of 250° F. can be easily reached and maintained economically by the steam under pressure, and since the heat is also more evenly distributed in the steam than in the air chamber, we give the preference to the former. The time during which articles must be subjected to a high temperature varies with the thickness of material, and the facility with which it conducts heat. It is usual to leave such articles as pillows in the hot-air chambers for as much as five or six hours. We need scarcely add that every town should be provided with at least one chamber for disinfection with the aid of heat."

The writer of the foregoing remarks has recently (1890) described a new and powerful disinfectant, which is a liquid combination of sulphur dioxide, camphor, and various volatile aromatic bodies. This material—named **Thiocamf**—possesses the remarkable property of giving off a very large volume of effective germicides on mere exposure to the atmosphere, and can be advantageously used for disinfecting a room instead of the treatment with nitrous fumes described above. The air of the closed room is first moistened by

sprinkling hot water freely about; a sufficient quantity of Thiocamf is poured on a large flat dish or old tray, and the place is shut up for two days. The usual thorough cleansing of the apartment should then take place. About six fluid ounces of Thiocamf are required for a small bedroom, and double that amount if the room be large.

Although it would be difficult to improve upon Professor Emerson Reynolds' plan of disinfection just described, the importance of the subject justifies some account of the official regulations for disinfection adopted in Berlin. Under date of August 15, 1883, the municipal authorities of that great city published regulations, of which the following is a synopsis, for disinfection in cases of catching or contagious diseases:—

"Especially powerful disinfection methods are necessary in smallpox, diphtheria, cholera, typhoid and typhus fevers. Disinfection is also obligatory, although less powerful methods may be used in scarlet fever, dysentery, and measles. It may also be required in some cases of whooping-cough, consumption, contagious inflammation of the lungs and eyes, splenic fever, glanders, puerperal fever, hydrophobia, and septicæmia. The means of disinfection specified are strong potash soap, carbolic acid, corrosive sublimate, carbolic acid spray, chlorine, ventilation, dry heat, steam and burning. Corrosive sublimate is employed only in the most contagious forms of disease, and its application should be directed by a physician. For the disinfection of furnished apartments everything used by the sick person which can be washed should be placed with as little shaking or agitation as possible into a vessel placed in the sick chamber, containing a solution of the potash soap. They are to be removed in this vessel for cleansing.

"Bandages, &c., used in dressing wounds are to be burned, and the instruments are to be disinfected with carbolic acid. Except in special cases all the secretions should be placed in vessels containing soap solution. Offensive odours should not be combated by fumigations or perfumes, but by an abundant ventilation, and as far as possible by the removal of the causes which produce them. It is forbidden to keep food in the sick chamber. Persons who enter it should abstain from eating or drinking while there. At

their exit they should wash and cleanse the beard, the hair, and the clothing by means of a brush moistened with carbolic acid. When it is necessary to carry to the hospital a person affected with smallpox, cholera, or typhus, it must be done by vehicles furnished by the police. It is forbidden to make use of public vehicles. When the patient is convalescent he should be bathed with the potash soap.

"Clothing which has been in the chamber during the diseaes should be exposed to the vapours of chlorine before use. Corpses of persons dying of a contagious disease should be buried as soon as possible. Those dying from small-pox, diphtheria, typhus, typhoid, or cholera, should be wrapped in sheets thoroughly moistened by a solution of corrosive sublimate. For other diseases clothes moistened with a solution of the potash soap may be used. Before sending the linen, clothing, &c., of the patient, to be washed, the articles should be folded without shaking or making a dust, and placed in sheets dipped in the solution of corrosive sublimate, and, without unwrapping, should be boiled for half an hour in this solution of potash soap, in cases of smallpox, diphtheria, cholera, typhoid, typhus, splenic fever, glanders, or hydrophobia. After the sick room has been emptied in all cases of smallpox, and in cases of scarlatina and diphtheria, when specially directed by the police, the floors, walls, windows doors, furniture, &c., must be rubbed with cloths, sponges, or brushes dipped in the solution of corrosive sublimate, immediately after which everything which can be scraped is to be cleansed with the potash soap. Upholstered furniture, cushions, mattresses, curtains, and other similar articles which cannot be washed, are to be disinfected in special establishments by the use of dry heat or super-heated steam. The mattresses and cushions must be opened for this purpose.

"After everything in the room has been treated as indicated, chlorine gas is to be generated in the room, the doors and windows being closed as tightly as possible. The chlorine is generated by pouring hydrochloric acid upon chloride of lime in equal parts.

"A kilogramme (2 lbs. 3 ozs. 119·8 grs.) of chloride of lime is required for the disinfection of an average-sized room. To prevent injury to metal fixtures which cannot be removed, they are to be

covered with a coat of oil or of varnish. The apartment is to be kept closed for twelve hours, at the end of which time the chlorine which does not pass out sufficiently rapidly by the windows may be absorbed by cloths dipped in ammonia."

In a paper[a] read in the Section of Bacteriology at the Seventh International Congress of Hygiene and Demography, held in London in August, 1891, Stabsarzt Dr. Behring, of Berlin, said that recent researches on tetanus, diphtheria, anthrax, and such like diseases, had shown that it is possible, *even within the body*, to combat successfully the various specific poisons produced in these diseases. In short, we have now got so far as to be able to speak of a "**disinfection within the body.**" There are four possible ways in which it is conceivable that this internal disinfection may be effected: —

1. By killing the disease-producing germs.
2. By hindering their growth.
3. By counteracting their disease-producing properties—pathogenic organisms losing their power to produce poisonous products.
4. By antagonising the action of, or destroying altogether the various toxic products produced by the bacteria.

It is possible that the disinfecting action may be exercised, not directly on the toxic products, but indirectly on the cells of the body, and that the beneficial result may be due not so much to a destruction of the toxic products as to an increased resisting power conferred on the cells of the body, in virtue of which they are able to withstand the action of these products. This last mechanism of disinfection is not proved, while concrete examples can be brought forward to prove the occurrence of the four other methods.

As an example of disinfection within the body, Dr. Behring shows that in the case of anthrax being inoculated in a mouse, the fatal result may be postponed or altogether prevented by subsequent injection of 0·4 cubic centimetre of a mixture of corrosive sublimate (one part of a 0·04 per cent. solution) and chloroborate of sodium (three parts of a 10 per cent. solution).

These and such like observations are full of interest and bright with promise of great results in the not distant future.

[a] *Brit. Med. Journal*, August 22, 1891. Pages 406, *et seq.*

The 139th section of the Public Health (Ireland) Act, 1878 (41 & 42 Vict., cap. 52), authorises every sanitary authority, whether urban or rural, to "provide a proper place, with all necessary apparatus and attendance, for the disinfection of bedding, clothing, or other articles which have become infected," and to cause any articles brought for disinfection to be disinfected free of charge. The sanitary authority also "may provide for the conveyance of such articles to such place."

II. We have, in the next place, to consider what measures should be adopted with the view of **lessening the receptivity** of an individual when exposed to the virus of one of the specific fevers. Regarding the introduction of that virus, or morbific agent, into the body as in each case the *exciting cause* of the specific disease in question, we find that one or more *predisposing causes* usually co-exist when an individual contracts that disease. Two things are, in fact, required—first, an infecting morbific agent, virus, or contagium; and, secondly, a state of the body which renders it peculiarly liable to be affected injuriously by that morbific agent. It is this latter which constitutes **predisposition to disease.** As Dr. William B. Carpenter points out,[a] any habitual infraction of the laws of health will induce a *general* liability to disease, by producing a depressed condition of the vital activity, whereby the organism is rendered less capable of resisting the influence of morbific agents. In this way **acquired predisposition** is brought about, as distinguished from **hereditary predisposition** to disease, which seems to follow the same modified laws of *heredity* as the transmission of *family* peculiarities.

The chief *predisposing* causes of febrile disorders may be enumerated as follows:—

1. The **presence of an antecedent disease**, which interferes with the excretory organs, by which the effete and waste matters circulating in the blood are eliminated and cast forth from the body. To enable an individual to resist the poison of typhus, or typhoid, or scarlatina, for example, the great emunctories—the lungs, the

[a] *Quain's Dictionary of Medicine.* Art. *Predisposition to Disease.* London: Longmans, Green, & Co. 1883. Page 1,252.

liver, the intestinal glands, the kidneys, and the skin, must all be sound and able for their work.

2. The **introduction into the body of putrescent organic matter** in either food, or water, or air, will certainly render a person more susceptible to a specific poison like that of scarlatina or cholera.

3. An **excessive generation of effete matter within the body**, as during the puerperal state, or after severe surgical injuries or operations, will have a like effect in the case of the poisons of scarlatina and erysipelas.

4. **Excessive exertion** of mind or body, marked by the feeling of fatigue, has always ranked as a most potent predisponent, for example, to typhus fever.

5. **Overcrowding** and **defective ventilation** are amongst the most powerful predisposing causes of typhus. "All the historians of the great epidemics of typhus," wrote Murchison,[a] "testify to the intimate connection between its prevalence and overcrowding."

6. **Alcoholic Intemperance** induces a strong predisposition to zymotic disease. As Murchison admirably puts it[b]:—"Habitual intemperance deranges digestion, impairs nutrition, causes degeneration of the excreting organs, retards the elimination of carbonic acid and urea, and lowers the tone of the nervous system." "It is not surprising," he adds, "that under such circumstances the body becomes more susceptible of the poison of typhus."

7. Lastly, it is unnecessary to enlarge on the intimate connection which at all times and in all places has existed between **Famine** and **Pestilence**. Murchison says[c]: "Destitution and deficient alimentation are the most powerful predisposing causes of typhus." As to relapsing fever, the same author observes[d] that some of the appellations bestowed upon it in different countries indicate the popular opinion as to its origin." It is essentially the *Famine Fever* of the British Isles, and the *Armentyphus* and *Hungerpest* of Germany.

[a] *A Treatise on the Continued Fevers of Great Britain.* Third Edition. 1884. Page 72.

[b] *Loc. cit.* Page 70.

[c] *Loc. cit.* Page 73.

[d] *Loc. cit.* Page 341.

Dr. William B. Carpenter, having regard to the fact that the presence of nitrogenous matter in a decomposing or readily-decomposable state affords the best possible *pabulum*, either for the development of pathogenic micro-organisms, or for the action of ferments, as long ago as 1853[a], came to the conclusion that the common condition which all the predisposing agencies just mentioned tend to produce is the presence in the blood of an *excess* of those decomposing effete matters with which the circulating current is normally charged to a limited extent. Should an individual, with his blood in such a condition, become the host of a pathogenic microbe, it goes without saying that his guest will batten and thrive, be fruitful and multiply exceedingly, with the result that symptoms of the special disease will soon declare themselves.

The **preventive measures** to be adopted, so far as an individual is concerned, who is exposed or is likely to be exposed to the poison of one of the micro-parasitic or specific diseases, are the following:—

1. Close attention must be paid to the **general health.** The presence of any organic disease is to be carefully sought for and combated. The state of the digestion, of the bowels, and of the kidneys, should be looked to. The skin should be kept in healthy action by daily thorough ablutions and dry rubbing, as well as by suitable clothing. Should a person's circumstances or health forbid a daily bath, the surface of the body may be washed piecemeal with luke-warm water and soap—absolute personal cleanliness is indispensable, and particularly in the case of young children. Free Public Baths and Wash-Houses should be provided in all large centres of population.

2. **Wholesome, plain food,** an abundant supply of **pure water,** and **fresh air** ought, in a civilised community, to be claimed as a birthright by every man, woman, and child. In a recently-published work on "Bacteria and their Products,"[b] Dr. German Sims

[a] *The Predisposing Causes of Epidemics. Brit. and For. Med. Chir. Review.* Vol. XI. Page 159, *et seq.*

[b] London: Walter Scott. 1891. Page 201.

Woodhead observes:—"Duclaux's dictum that fresh air and sunlight are two of the most powerful agents that we have with which to combat the onslaught of disease, cannot be too strongly insisted on."

Full powers are given to sanitary authorities for the inspection of food, in England under sections 116–119 of the Public Health (England) Act, 1875; in Scotland under Section 26 of the Public Health (Scotland) Act; and in Ireland under sections 132–136 of the Public Health (Ireland) Act, 1878. As regards water supply, it is generally conceded that 30 gallons of pure water per head of the population should be provided, if health is to be maintained, especially in large towns. The question of fresh air we will discuss later on. Defective sewerage arrangements frequently lead to contamination of food, drink, and air, and therefore demand immediate attention.

3. From what was stated above, it is evident that the strictest **sanitary precautions** should be taken when a **puerperal woman** or a **wounded person** is exposed to the poison of any of the diseases we are considering.

4. "**Fatigue of mind or of body,**" says Murchison,[a] "is to be **scrupulously shunned** by persons who are necessarily exposed to the poison of typhus." And this is equally true of the other infectious fevers also. No physician, student of medicine, or nurse should enter a fever-ward tired, or after a sleepless night. Recreation of mind and body at stated intervals is an essential preventive measure in the case of those who attend fever patients. Their hours for sleep should be ample and undisturbed. They should *never sleep in the sick room*, and as much of their time as possible should be spent in the open air.

5. If health is to be maintained, whether among the poor or among the rich, **sufficient cubical air space** and **free ventilation** in the dwelling are essential. Adults spoil every hour about 5 cubic feet of air (140 litres)—that is, they render 5 cubic feet of air absolutely irrespirable. But 600 times that amount of air—that is, 3,000 cubic feet—will be required to keep the atmosphere "in

[a] *Loc. cit.* Page 272.

the highest degree of practicable purity."[a] In practice, it is found that from 500 to 750 cubic feet of air-space per head are required in a dwelling room; but, in addition, 2 cubic feet of fresh air per head should be admitted into the room every minute. This latter indication is effected by constant and occasional **ventilation** or "the art of supplying, without perceptible draught, the requisite quantity of pure air."[b] Happily for mankind, it is not possible to hermetically seal a dwellingroom—no matter how rigorously windows and doors are closed, air leaves such a room by the chimney and enters it by various chinks and crevices. This interchange of air, which is beyond control, is called **constant** or **natural ventilation.** The more decided change of air, which is called **occasional ventilation,** occurs when doors and windows are thrown open. This must be duly regulated so as to avoid a draught, which is caused when air at a temperature between 55° and 60° F. streams through an opening at a greater rate than two miles an hour, that is, three feet per second. It is necessary to observe that near a cold surface, such as a damp wall, a sensation of "draught," or chilliness, may be caused even when the air is almost still.

"The dwellings of the poor," says Murchison,[c] "ought to be so constructed as to ensure good ventilation. Closed courts, surrounded by high houses, are always objectionable. Every window-frame ought to be movable, and every room should be provided with means for *constant* ventilation. Human beings ought to be prohibited from living in underground cellars, where proper ventilation is impossible."

6. Among the most preventable predisposing causes of infectious disease is **alcoholic intemperance.** It is not so widely known as it should be that this deadly sin not only strongly predisposes to these diseases but also materially lessens the chance of recovery from them. The vice betrays and kills. Let us once more hear Murchison on this point:—"There is no greater error than to

[a] A. Wynter Blyth. *Loc. cit.* Page 63.
[b] A. Wynter Blyth.
[c] *Loc cit.* Page 269.

imagine that a liberal allowance of alcoholic stimulants fortifies the system against contagious diseases."[a]

7. "The prevention of **scarcity of food, loss of employment**, and other causes of destitution," writes the same great physician, "is not always within human power; but, under such circumstances, every means, both public and private, calculated to alleviate the distresses of the poor should be adopted. Moreover, no time is to be lost in affording relief; it is difficult to stay the plague when once it has begun. Care also must be taken that the funds collected for such purposes do not produce the very evils they are intended to avert. The poor naturally flock in greater numbers to those localities where most relief is to be obtained, and the result has often been increased crowding. The expediency of supplying relief to the poor in their crowded dwellings may, therefore, be questioned. A preferable plan would be to establish, during seasons of scarcity and when typhus is prevalent, **temporary buildings of wood or iron**, or **tents**, in the neighbourhood of large towns. Here overcrowding could be prevented, the poor could be supplied with abundance of fresh air and food, while the number of persons resorting thither for relief would prevent overcrowding in the towns. The expense of such a plan would certainly not exceed what the spread of an epidemic always entails."[b]

8. To this branch of the subject of prophylaxis belongs also the procedure commonly called "**inoculation**," the intention of which is to engraft in a healthy individual—whose *receptivity* in consequence would be low—a mild form of a particular infectious disease by means of an *attenuated virus* of that disease.

III. The third great means at our disposal in the attempt to check the spread of one of the infectious fevers is the strict enforcement of quarantine (using the term in its modern acceptation),[c] and of isolation.

1. In Table I., p. 29, under the heading, "**Quarantine**," the time is specified during which possibly infected persons should be segregated, or kept apart, so as to secure that they shall not do further

[a] *Loc cit.* Page 70.
[b] *Loc cit.* Page 269.
[c] See above. Page 27.

mischief by spreading infection should they themselves develop symptoms of disease. This risk is vastly increased in the case of a malady like measles, which is infectious, or "taking," from the first sneeze which the patient gives.

In private families, or those occupying the whole of one house, it will suffice that the member or members of the household suspected to have contracted a disease shall be kept apart from the rest of the family until such time as the incubation period of the particular disease shall have passed by without the development of suspicious symptoms.

Refuges for persons removed from infected tenement houses should be provided in every town where a number of families reside under one roof, in order to stay the spread of an epidemic. The sick, in such a case, should at once be removed to hospital, and the healthy to a refuge where they could be watched for the outbreak of further cases. In this way the infected house could be conveniently cleansed and disinfected. The Refuge should be arranged in *sets* of rooms, or "flats," as they are called in Scotland—so that each family might keep together. This would not only ensure the comfort of the family, but also limit the spread of the disease to the particular family among whom it arose.

2. When a member of a private family is proved, or even suspected, to be suffering under an infectious or "taking" disease, he should be **isolated**, preferably in a separate building, or, failing this, in an airy room at the top of the house. Outside the door of the sick chamber a sheet should be hung, and kept saturated with some liquid volatile disinfectant, like carbolic acid, sanitas, or chloride of lime. The room should be large, well ventilated, but warm (temperature about 60° Fahr), well lighted. It should contain an open fire-place. To secure ample air space, as well as to lessen the risk of infection through **fomites**, or "**carriers of infection**," all bed-curtains, carpets, and non-essential articles of furniture should be removed. The patient's surroundings should be kept scrupulously clean and pure; soiled linen and crockery should not be taken out of the sick room until they have been dipped in a disinfecting solution—carbolic acid, benzoic acid, sanitas, Condy's

fluid, or corrosive sublimate. The sick room may be sprayed with sanitas solution several times a day, or one of the "disinfecting cones," supplied by the Chemical Carbon Company, may be burned from time to time.

Among the poorer classes isolation—important and theoretically simple a plan as it is for the prevention of the spread of infectious diseases—is with difficulty carried out. The means employed for the purpose are—(1) the early removal of the sick to hospital in a properly constructed ambulance; (2) his admission, first to a reception-room, where his own clothes should be taken off, disinfected if necessary, and then removed to a proper store-room, and where the patient himself should be washed and made tidy before he is placed in bed in a suitable, airy, well-ventilated and properly heated ward; (3) his treatment in such a ward, reserved absolutely for cases of the disease from which he is suffering, visitors being forbidden admission except under the most pressing and urgent circumstances; and (4) lastly, his removal in due time to a Convalescent Home, where he may gain health and strength before he returns to his work.

The treatment of the infectious fevers in the wards of General or Medico-Chirurgical Hospitals, should, as far as possible, be avoided, and permanent **Epidemic Hospitals** should be provided by every Sanitary Authority, the accommodation in such hospitals being at the rate of one bed for every 200 of an urban population, and every 400 of a rural population.

A lengthened experience has led me to the conclusion that the most feasible method of attempting to limit the spread of "catching" or "taking" disease, is by a system of complete **isolation**, not only of the sick, but also of the convalescent. The convalescent from an infectious disease must be kept separate until the days of his purification are accomplished—in the words of the Mosaic Law:—"He shall dwell alone; without the camp shall his habitation be."[a]

In hospital and in private practice, I have had frequent opportunities of recognising the want which exists of suitable accommodation for infectious convalescents. Although we are just

[a] *Leviticus.* Chap. xiii., v. 46.

now primarily concerned with the protection of the healthy from the risk of infection, I may be permitted to advance four reasons why, in the interest of the convalescents themselves, we should provide for them a temporary home *outside the hospital precincts*:—

1. The health of convalescents is often seriously injured if they are kept in hospital beyond a certain period in their progress towards recovery.

2. The surroundings in even the best-managed epidemic hospitals cannot fail to exercise a depressing influence on the minds of patients who are recovering from illness.

3. It should not be forgotten that beds are being occupied by the convalescent which may perhaps be urgently required at the very moment for other patients in the earlier stages of their sickness.

4. Lastly, it is most desirable that those who toil for their daily bread should not leave hospital to return at once to their wonted employment. It frequently has happened that the sudden strain in this way put upon the bodily powers not yet recovered from a deadly conflict with sickness has led to disastrous results—in some instances the seeds of permanent organic disease have been thereby sown.

Patients in private practice also, equally with hospital patients, require "change of air" after an illness, and provision should be made for them in separate rooms in a **Convalescent Home for Infectious Diseases.**

So far as to the patients themselves. No one will need to be told how imperative it is in the interest of the public health that the convalescent from infectious maladies should be kept apart from the healthy until all risk of infection is over. This applies not less to private than to hospital patients. How can the needful isolation be kept up except through the medium of Convalescent Homes?

The Law is on my side in my advocacy of the means of isolation now indicated. Take, for example, the "Public Health (Ireland) Act, 1878." Section 140 requires (it is *compulsory*) that "every sanitary authority shall provide and maintain a carriage or carriages suitable for the conveyance of persons suffering under any infectious

disorder, and shall pay the expense of conveying therein any person so suffering to a hospital or other place of destination, and shall keep such carriage or carriages properly disinfected." Can any legal document be more explicit than this?

Section 141 of the same Act of Parliament provides for the removal of infected persons without proper lodging to hospital by order of any Justice of the Peace. Section 142 imposes a penalty on exposure of infected persons and things. Under Sub-section 5 of this Section, in the Northern Divisional Police Court, Dublin, on Saturday, July 12, 1879, before Mr. C. J. O'Donel, a man was fined £5—the full penalty—for permitting a "wake" to be held in his room in a tenement house in Church-street, Dublin, on the body of his son, who died of confluent smallpox on the 16th of the previous month. Section 143 imposes a penalty on failing to provide for the disinfection of a public conveyance. In the same Police Court as before, on Saturday, February 21, 1880, the same Magistrate fined the driver of a hackney car £5—the full penalty—for failing to have his car disinfected after conveying a smallpox patient on it to the Hardwicke Fever Hospital, Dublin, on February 5.

Section 155 empowers any Sanitary Authority (whether urban or rural), with the sanction of the Local Government Board, to provide for the use of the inhabitants of its district, "*hospitals or temporary places for the reception of the sick or convalescent.*" These are the very words of the section, and nothing can be more definite or comprehensive than they are. The section requires amendment only so far as it does not contemplate or authorise the provision of refuges for those still healthy who have been removed from infected dwellings.

To sum up—the **Prophylaxis**, or **Preventive Treatment** of acute specific infectious disorders consists in—

1. **Disinfection**;
2. The **removal** or **neutralisation of the Predisposing Causes** of this class of diseases;
3. The strict **enforcement of Quarantine** and of **Isolation.**

CHAPTER V.

The General Principles of Treatment of the Eruptive and Continued Fevers.

Part II.—Curative Treatment.

Principles of Treatment of Fevers.—1. To NEUTRALISE THE FEVER POISON: sanitary surroundings, mineral acids, antiseptics.—2. To PROMOTE ELIMINATION: fresh air, diluents, diuretics, diaphoretics, aperients or laxatives, beneficial effects of common salt (chloride of sodium).—3. To REDUCE TEMPERATURE: hygienic measures—bloodletting (general or local), saline cathartics, diaphoretics, antipyretics (Cantani's Views on Antipyresis); hydriatic measures—tepid sponging, the wet-pack, the application of ice-cold compresses, the cold bath, the ice-cradle (Fenwick).—4. To MAINTAIN NUTRITION: food to be both nutritious and digestible, peptonised food, times for feeding the fever patient, feeding through the nares or by the rectum; administration of alcoholic stimulants—indications for their use, signs of their agreeing with the patient, avoidance of exhaustion.—5. To RELIEVE DISTRESSING SYMPTOMS: headache, sleeplessness, nervous excitement, delirium, stupor, convulsions, hyperæsthesia, rheumatoid and neuralgic pains, thirst, persistent vomiting, tympanites or meteorism, hiccough, diarrhœa, intestinal hæmorrhage.—6. To OBVIATE AND COUNTERACT LOCAL COMPLICATIONS AND SEQUELÆ.

If we bear in mind the bacterial origin—proved in some cases, inferred by analogy in others—of the diseases we are considering, their clinical history, and the pathological appearances they present, we may with Murchison lay down the following **Principles of Treatment:**—

1. To **neutralise the fever poison** and improve the state of the blood.
2. To **promote elimination**, not merely of the fever poison, but also of the products of tissue-change, or metamorphosis.
3. To **reduce temperature** and to lessen the frequency of the heart's action.
4. To **maintain the nutrition** of the body and to **stimulate** when necessary the heart's action by food and stimulants.

5. To **relieve distressing symptoms** as they arise—such as headache, sleeplessness, pain, restlessness, prostration, stupor, delirium, cough, constipation, diarrhœa, thirst, and so on.
6. To **obviate and counteract local complications**, or those secondary affections which intervene in the course of the primary malady, upsetting its normal course and imperilling the patient's life, or prolonging the fever movement to a greater or less extent.

It will be necessary to consider each of these indications for treatment in some detail:—

1. The **sanitary surroundings** of a patient suffering under a specific fever should be above suspicion. In the previous chapter we have already dealt with this topic at some length from the standpoint of Preventive Medicine, and it is a happy reflection that all the measures we take to arrest the spread of infection from the sick chamber will also directly benefit its stricken tenant for the time being. Fresh air and warmth and daylight are magical remedies in disease. The darkened sick room is a mistake, except perhaps in meningitis or measles, or when the patient complains that light hurts his eyes (photophobia). A patient will sleep better by night in a room where a broad difference between day and night has been made. Of one thing we may be perfectly sure, that it is madness to treat a typhoid fever patient in the house where he has contracted his illness, with its tainted atmosphere and its probably poisoned water supply. Nothing is more striking than the marvellous improvement which often follows a fever patient's removal to an airy, well-warmed, wholesome ward of a hospital.

Among drugs which tend to fulfil the first object we set before us in treating febrile diseases, the various so-called "**mineral acids**" enjoy a well-established and classical reputation. Probably the most useful of all is the dilute hydrochloric acid.[a] In cases where the bowels are loose, dilute sulphuric acid or the aromatic sulphuric

[a] "Half a drachm of the dilute acid with a like quantity of the tincture and syrup of orange may be given in solution every three hours."—Murchison. *Treatise on the Continued Fevers of Great Britain.* Page 274.

acid may be substituted with advantage. Dilute nitric and phosphoric acids also come under the same heading. They may be combined with antiseptics like quinine or ferric chloride, or with cardiac tonics like digitalis or nux vomica, or with nervine tonics like strychnin or hydrochloric solution of arsenic.

Antiseptics are prescribed to meet the same indication. Those chiefly in use are carbolic acid, sulphurous acid, sulphite of sodium, the sulpho-carbolates (which enjoyed a considerable reputation in the last two epidemics of small-pox in Dublin), bismuth, salicylate of sodium, chlorate of potassium, permanganate of potassium, and—*facile princeps*—quinine. Inhalation of oxygen is well worth a trial as a blood purifier in septic fevers with impaired lung-action and cyanosis.

It may, perhaps, be necessary to explain that, in the words of Dr. J. Burney Yeo,[a] "the idea of an *antiseptic* treatment of certain forms of disease has been greatly misunderstood or intentionally misrepresented by those who for some inscrutable reason dislike it; and those of us who have been bold enough to entertain this idea have been credited with the crude intention of attempting to slay these parasitic morbific agents 'in a direct and simple manner,' and we have been gravely told that our so-called antiseptic methods are so murderous that our patients and not the microbes fall victims to them." Dr. Yeo shows that every analogy in nature points to the possibility of effectually modifying the life-history and activities of all living things by even slight modifications of their environment. The real aim of the antiseptic treatment of the infective diseases is to modify or counteract the injurious activities of the living parasitic agents of these diseases. Antiseptic methods, too, may act either by preventing the formation of the poisonous animal alkaloids, or ptomaïns, to which pathogenic microbes seem, or are believed, to give rise, or by destroying these ptomaïns when formed, or, lastly, by promoting their discharge from the body.

[a] *The Treatment of Typhoid Fever, especially by "Antiseptic" Remedies.* London : Cassell & Co. 1891. Pages 5–8.

2. In attempting to meet the second indication of restoring or increasing the eliminative power of the various organs which are commonly called the "**great emunctories**," we rely on fresh air, diluents (of which the best and safest is pure cold water), diuretics, salines, diaphoretics, emetics (now very seldom employed except, perhaps, in the early stage of typhoid fever), and simple purgatives or laxatives.

The fever patient is fastidious in his choice of drinks, but he may be allowed to partake freely of such liquids as water, iced water, toast water, barley water, raspberry vinegar, well-diluted lemon-juice, or the juice of oranges when in season. Soda-water and milk or seltzer-water and milk make a nutritious beverage. All these **diluents** should be taken in moderate quantities at a time, so as not to distend the stomach and so cause discomfort.

The **diuretics** employed should, as far as possible, be non-stimulating, such as digitalis, nitre-whey (prepared by boiling two drachms of nitrate of potassium in a pint of milk and straining), *potus imperialis* (prepared by dissolving 60 to 120 grains of acid tartrate of potassium—"cream of tartar"—in a pint of boiling water and flavouring with lemon and sugar), tea and coffee, citrate of caffein, or, better still, "*diuretin*," (sodio-theobromin salicylate) given in 15 grain doses thrice a day in mixture with peppermint water and syrup.

Diaphoretics are not often employed at the present day in the treatment of fever, and scarcely ever should be given after the first few days of illness. Common nitre, and spirit of nitrous ether are those probably most frequently used. But as a rule it will be found that tepid sponging from time to time will keep the skin acting sufficiently.

Throughout an attack of fever, it is desirable that the bowels should act regularly, but only mild methods should be adopted to secure the end in view. Very often copious draughts of water alone will suffice; a glycerine enema or suppository may be used (except in typhoid fever, when a simple enema will be safer); a small dose of calomel (1 to 5 grains) with sugar of milk or rhubarb powder (except in scarlet fever); a teaspoonful of castor oil with one of

glycerine in milk, repeated every 6 or 8 hours if necessary; or a dose of the new "Mistura Olei Ricini" of the British Pharmacopœia—these are all safe **aperients** or **laxatives.** Young children bear a dose of calomel—1 to 3 grains—uncommonly well, and it usually has an excellent effect.

The good effects of giving common salt—"chloride of sodium"—in fever are possibly due to its antiseptic properties and to its property of increasing elimination. Murchison used to order large quantities of salt to be mixed with the patient's beef tea, and found it in most cases greatly relished and apparently beneficial.

3. The third indication for treatment—namely, the reduction of temperature—may be met in the first place by the adoption of the hygienic measures already described. The patient should lie on a hair-mattress, or a woven wire-mattress, covered with a moderate quantity of bed-clothes. The temperature of the sick room should not much exceed 60° F.

Bloodletting, whether general (venesection) or local (leeching or wet cupping), was, in days gone by, a favourite means of reducing temperature, and to the present day the fall of temperature which accompanies an intestinal hæmorrhage in typhoid fever seems to warrant the adoption of this mode of treatment in hyperpyrexia, or excessive fever. But in practice, bloodletting is found to be far too depressing to be safe, except in rare instances. For example, in certain cases of acute pneumonic fever, venesection occasionally certainly saves life, and local depletion by leeching or wet cupping relieves the early headache of typhus or smallpox, lessens the amount of eruption in the latter disease, and restores kidney action in the nephritis of scarlatina.

Saline cathartics or **diaphoretics** are sometimes employed to reduce temperature, but these classes of remedies are inadmissable when we have to deal with a failing heart. We then resort to the so-called **antipyretics**, of which quinine, digitalis, acetanilide (antifebrin), phenacetin, and phenazone (antipyrin) are the most striking representatives. If acetanilide and phenazone are given in large doses—10 to 20 grains—they are apt to cause cardiac

depression and even cyanosis, but this objection does not hold good in the case of phenacetin. Even 3 to 5 grains of phenazone have produced very unpleasant effects in a child.

In Cantani's Address on "Antipyresis" to the Tenth International Medical Congress at Berlin, to which I have before referred, he says that the proper remedies for fever would be such as act on the cause of the disease. In this way quinine acts in malaria, mercury in syphilis. The **antipyretic** remedies—as antipyrin (phenazone)—have no special action on the causes of fever. They lower temperature by increasing radiation of heat from the body, and they diminish heat-production. They do harm by interrupting the course of the fever, diminishing the means of defence of the human organism, for a diminution in the production of heat is equivalent to a diminution of the vitality of the human organism and of its power of resistance. In excessive fever (hyperpyrexia), however, the cardiac muscle and the nervous system suffer; and under these circumstances, a reduction of temperature is desirable, if, by abstraction of heat, heat production is not diminished. The "**cold water treatment**" (*die hydriatische Wärme-entziehung*), by cold baths, the cold pack, cold douches, the internal administration of large quantities of cold water, either by the mouth or by enema, act in this way. They absorb a large amount of heat from the body, whilst they rather augment the formation of heat. It is not known if the good effects which these remedies produce are due to increased tissue waste and elimination of excrementitious matters, including ptomaïns and leucomaïns. Certain diseases, attended with high temperature, are often successfully treated with **diaphoretics**, which cause an increased excretion of noxious matters, but which scarcely—if at all—increase heat production.

Cantani[a] holds that for certain fevers there are **specific remedies**, which attack the cause of the disease itself. There are, however, no general antipyretics. The most that can be done is to diminish the accumulation of heat in the febrile body without lowering the production of heat. To effect this, the **cold water treatment** can

[a] See abstract of Cantani's Address in Sajous' *Annual of the Universal Medical Sciences for* 1891. Vol. I., pages 34–36.

be recommended, whilst the chemical antipyretics must be looked upon with suspicion as general remedies for fever.

"The **application of cold water** to the surface of the body," says Dr. Samuel Fenwick,[a] "is the most certain means of reducing an elevated temperature, and there are various methods of employing this measure, each of which is specially adapted for particular cases. The chief of these are *sponging*, the *wet-pack*, the *application of cloths wet in ice-cold water*, the *cold-bath*, and the *ice-cradle*."

Tepid sponging is most grateful and refreshing, and reduces the temperature of the surface about $1\frac{1}{2}°$ or 2° F. To carry it out, the patient is laid on a folded blanket and the surface of the body is thoroughly *mopped* with a sponge squeezed out of tepid or warm water, to which a varying proportion of vinegar or aromatic vinegar has been added. The sponging may be continued for 10 or 15 minutes and renewed twice or three times in the 24 hours. The **wet-pack**, recommended by Brand of Stettin, is applied as follows:—A sheet is quickly wrung out of ice-cold water and wrapped several times round the patient's body, a couple of dry blankets are then applied outside the wet sheet, and a Macintosh outside all. The pack is very refreshing and often induces sleep. The Gräfenberg plan, mentioned with approval by Hebra and Mayr in their account of the treatment of scarlatina, is merely a variety of this "wet-pack," in which the head is enveloped in wet napkins, and the patient is given cold water to drink. Ice, also, is applied (*Eisumschläge*) if intense congestion is present. Dr. Sydney Ringer is the special advocate of the continuous **application of cloths wet with ice-cold water** and rung nearly dry. They should be applied one below the other from the chest downwards. This is an effectual method of lowering temperature.

Immersion in cold baths is the method now most in fashion, particularly on the Continent, and in the treatment of typhoid fever. It is contra-indicated in hæmorrhage from the bowels. When the patient's temperature reaches 104°, he is placed in a bath having a temperature of from 50° to 70° F., or better, as recommended by

[a] *Outlines of Medical Treatment.* Third Edition. London: J. and A. Churchill. 1891. Page 424.

von Ziemssen, in one with a temperature of some 10° below that of the body, but which after the patient's immersion is gradually cooled down to 68° by adding cold water. The patient should remain in the bath for half an hour, or until shivering comes on, when his limbs should be rubbed by the attendants, he should be quickly dried and put into a warm bed. The fullest account of this method of treatment will be found in Liebermeister's article on "Typhoid Fever" in the first volume of von Ziemssen's "Cyclopædia of the Practice of Medicine" (page 208.)

Dr. Fenwick[a] thus describes the "ice-cradle":—"I was led to adopt a method of reducing the temperature in fever which I have employed for many years at the London Hospital and in private practice, and which seems to me to possess the advantages of the cold bath without its drawbacks. It is generally called in the hospital the 'ice-cradle,' and consists of an ordinary iron surgical cradle, of sufficient width to allow the patient to move easily beneath it, and long enough to cover the whole body. In it are suspended three or four small zinc pails filled with ice; the bottoms of the pails being covered with a piece of lint, so as to prevent any condensed moisture from dripping upon the patient's body. A light coverlet is thrown over the cradle, an aperture being left at either end, in order to allow a free circulation of air through it. Under this the patient lies, either naked or covered with a very thin opaque gauze, whilst a hot water-bottle placed against the feet adds to his comfort, and assists in warding off any tendency to chilliness. If the iron framework is not obtainable, a good substitute can easily be made with a piece of stout wire, or a couple of wooden hoops, such as children are accustomed to play with; a string or wire being fastened along the centre, from which the pails may be suspended."

The ice-cradle has been used for many years at the London Hospital with satisfactory results. Many severe cases of typhoid fever have lain beneath it for a fortnight, or even longer—the temperature remaining constantly below 104°. Its efficacy was demonstrated by its temporary removal, when the thermometer in the axilla would rise to 105° or upwards. It is not applicable in

[a] *Loc. cit.* Page 429.

cases of hyperpyrexia, where a rapid reduction of temperature is indicated, and it produces at first a certain amount of discomfort. The patient, however, soon becomes accustomed to the application of the cradle.

In 1787, **cold affusion** was proposed by Dr. Currie of Liverpool both for arresting and mitigating continued fevers. The patient was seated naked in an empty tub or bath and several buckets of water of a temperature of 40° to 50° F. were poured from a height of one to three feet, or more, over the head and chest. He was then hastily dried and placed in bed—the operation being repeated once or twice daily. The affusions were most successful when employed at the beginning of the fever, but even later this treatment reduced the pulse and temperature, relieved many of the distressing symptoms, and conducted the disease to a safer and speedier issue. Following the practice of Dr. Stokes at the Meath Hospital,[a] I have often used cold affusion, adopting the modified plan of pouring cold water from a jug over the head and face of a typhus-fever patient while still lying in bed crossways, his head and shoulders being supported over a bath placed alongside the bed. The stream of water should be directed upon different parts of the head from time to time, for otherwise pain may be caused. This last remark bears on the application of ice to the head also—a procedure of which Dr. Stokes speaks in terms of the highest approbation. He, however, observes that it is often most clumsily and ineffectually carried out. The proper method is to place a piece of ice, rubbed smooth with the hand, in a cup-shaped sponge of convenient size. Then, by inverting the sponge, the ice may be brought into contact with the shaven scalp and so passed round and round the head by a constant gentle motion. The sponge absorbs the water. When it is saturated it can be squeezed out, the ice replaced and the application resumed. In this way no pain is caused, the proceeding is grateful to the patient, and the whole head is uniformly and gradually cooled.[b]

[a] *Lectures on Fever.* By William Stokes, M.D., D.C.L. Oxon., F.R.S. London: Longmans, Green & Co. 1874. Page 411.

[b] *Loc. cit.* Page 412.

4. While we endeavour to sustain the vital powers of the fever patient by appropriate food and stimulants, we should avoid anything which would cause congestion or put an additional strain on organs already overtasked and with impaired functions. The food should be both nutritious and digestible, consisting of such articles as milk, eggs, beeftea, veal-broth, chicken-broth, mutton-broth (strained), meat-essences, meat-jellies, arrowroot, sago, bread and milk, custard, tea or coffee, well diluted with milk. If there is a tendency to diarrhœa, the milk should be boiled, or lime water should be given with it, in the proportion of one part in four; or vermicelli, gelatine (isinglass), or arrowroot should be added to the milk or broth. Acids should not be given with milk, which they coagulate, or cause to curdle. If the digestive powers are very weak, the food may with advantage be **peptonised**, the object of which process is to convert insoluble proteids or albuminoids into soluble peptones.

Food should be given to the fever patient at regular intervals—every three hours, every two hours, or even every hour; but, at the same time, the stomach should be allowed to rest for at least the last-named interval, else nausea, vomiting, flatulence, and diarrhœa may be caused through non-assimilation and decomposition of the food.

When a patient remains in a state of stupor he should be roused from time to time to take food; but, if he falls into a tranquil sleep after a period of wakefulness, nervous excitement, or delirium, he should not be aroused merely because the hour for food has come round. In delirious cases, or when the patient is unconscious, or unable to swallow, liquid nourishment should be introduced into the stomach by a long tube passed through the nares, or nutrient enemata should be administered. In the latter case the rectum should first be washed out by a *lavement* of warm water, and then an enema should be given of milk and brandy, or beef-tea, or egg-flip, at a suitable temperature (100° F.), and of moderate size (not exceeding 4 to 6 oz.).

It used to be the practice to half-starve the unhappy and ill-starred fever-patient; but Dr. Graves changed all that. Dr.

Stokes[a] narrates how his distinguished colleague was going round the Meath Hospital, when he began to expatiate on the healthy appearance of some convalescents from typhus. "This is all the effect of our good feeding," he exclaimed, "and lest, when I am gone, you may be at a loss for an epitaph for me, let me give you one in three words, 'He fed fevers.'" This is all very well, but there is reason to fear that the pendulum has swung rather too much in the other direction of late years, and that fever patients have been overfed, rather than underfed. Very few patients can digest more than one pint of animal broth, and from one and a half to two pints of milk, in the twenty-four hours, and these quantities seem to be a fair allowance, under all the circumstances, in the case of an adult.

The question of the **administration of alcoholic stimulants** in fever is an anxious one. Here, also, medical practice has been pushed to opposite extremes at different times. At present there is a distinct reaction from the liberal use of stimulants advocated and employed by Alison, Graves, Todd, and Stokes; and the result has, in my opinion, been satisfactory, although we seem to be in some danger of running into the other extreme of withholding wine or spirit when its use would really benefit the patient. Murchison[b] lays down excellent rules for our guidance in the use of stimulants. He considers that patients under twenty years of age do best as a rule without any alcohol, whereas most patients over forty are benefited by it after the first week of the fever. Intemperate persons require alcohol earlier and in greater quantity than others, and yet it does not always agree. The **chief indications for the use of stimulants** are derived from the state of the pulse, the heart, the tongue, and the brain, and from the presence of complications, and especially of the "typhoid state" (*i.e.*, stupor, low muttering delirium, tremor, subsultus, involuntary evacuations, coma vigil, &c.).

[a] *Studies in Physiology and Medicine.* By the late Robert James Graves, F.R.S. Edited by William Stokes, Regius Professor of Physic in the University of Dublin. London: John Churchill & Sons. 1863. Page lix. of the "Biographical Notice" by Dr. Stokes.

[b] *Loc. cit.* Pages 291 and 292.

Alcoholic stimulants are doing a fever patient good if, under their use—

1. The heart's action becomes stronger and less rapid, the impulse increasing in strength and the first sound becoming more distinct.
2. A soft, compressible, undulating, irregular, or intermitting pulse becomes fuller and stronger, and more regular in rhythm and volume.
3. A dry, brown tongue becomes clean and moist at the edges.
4. Delirium lessens, the patient becoming more tranquil, or even falling asleep.

Stimulants are most urgently required during the night and in the early morning, when the vital powers are wont to flag. In the forenoon they are less necessary.

In cases of extreme prostration, medicinal stimulants or tonics should be combined with wine or spirits. The preparations and drugs mostly employed under these circumstances are—carbonate of ammonium, the different ethers, camphor, musk, turpentine, bark, or quinine.

In addition to all these measures, the fever patient should husband his strength by taking to bed at the earliest opportunity and *remaining* there until convalescence has begun—in a word, the patient must be prevented in every way from exhausting his muscular and nervous powers.

5. Until its own specific remedy is discovered in the case of each of the infective febrile disorders, one of the main objects of treatment must be the relief of distressing symptoms—the treatment must be largely **symptomatic.**

Of these symptoms **headache** is one of the most common and earliest to appear. It is sometimes relieved by an emetic of ipecacuanha (20 grains) and antimony (1 grain), or of carbonate of ammonium (40 grains) (Murchison), or by an action of the bowels, or by the application of evaporating lotions or of warm fomentations to the forehead. Failing these measures, the hair should be cut close or the head shaved, and cold affusion tried, or

ice applied as described above. A blister or sinapism applied to the forehead or nape often does good; or, in the case of a young and robust patient, leeching the temples or mastoid process is very efficacious. A curious and beneficial effect of local depletion will be noticed in our account of smallpox.

Sleeplessness, nervous excitement, and **delirium** may often be controlled by the measures recommended for the relief of headache. Good nursing tells against these most exhausting symptoms. The proper alternation of day and night conduces to sleep, so the room should not be darkened in the daytime, unless photophobia is present. Whispering in the sick-room should be forbidden—far better to talk aloud, if it is necessary to talk at all. In private practice the plan of having two beds, and changing the patient from one to the other every twelve hours or so, often brings on sleep. Should all these measures fail, and if the patient has had little or no sleep for thirty-six hours, recourse must be had to **hypnotics.** It would be outside the scope of a Text-book to do more than direct attention to a very few selected drugs and formulæ. Foremost among hypnotics stands *opium*, which, unfortunately, has the disadvantage of tending to lock up the secretions. Nevertheless, to an adult, *whose kidneys are sound*, 15 to 20 minims of Battley's solution may be given; or 20 to 30 minims of the solutions of the hydrochlorate or acetate or bimeconate of morphin, or 5 grains of the compound soap-pill of the pharmacopœia, or 10 grains of the compound powder of ipecacuanha, or better still, a pill representing that dose and consisting of 1 grain each of extract of opium and of ipecacuanha, with 2 grains of extract of hyoscyamus. A hypodermic injection of morphin ($\frac{1}{8}$–$\frac{1}{4}$ grain) may be substituted for any of the foregoing, if preferred. Graves used to combine tartar emetic with opium, and with excellent effect in the persistent insomnia of typhus with delirium. His formula was a mixture containing 4 grains of tartar emetic and one drachm of laudanum in eight ounces of camphor water[a]—of this the dose usually given was two to four

[a] *Clinical Lectures on the Practice of Medicine.* By Robert James Graves, M.D., F.R.S. Edited by John Moore Neligan, M.D. Dublin: Fannin & Co. 1864. Page 177.

drachms every second hour until the patient slept. He varied the relative proportions of tartar emetic and laudanum in the mixture according to circumstances, and laid down as a rule that "*the practitioner must in all cases watch the effects of this medicine from hour to hour*, until he ascertain whether it agrees with the patient or not." The italics are in the original.

Murchison proposed that digitalis should be given in combination with the opium or morphin, and there is no doubt that by these combinations sleep will be induced in many cases where opium alone would fail or be contra-indicated. Murchison's prescription is as follows:—

℞. Liquor Opii Sedativi, ʒj ;
Tincturæ Digitalis, ʒj ;
Spt. Ætheris Nitrosi, ʒij ;
Aquæ Camphoræ, ad ℥vj ;

M. Sumat coch. mag. ij statim, et coch. mag. j secundâ quâque horâ usque ad somnum. (One fluid ounce at once, and half a fluid ounce every second hour until the patient sleeps.)

It should not be forgotten that **opium** in any form is **contra-indicated**:—(*a*), in infants ; (*b*), when there is evidence of extensive pulmonary engorgement ; (*c*), when the pupil is persistently contracted ; (*d*), when the urine is scanty, bloody, or highly albuminous ; and (*e*), when the patient, although sleepless, is in a state of profound nervous prostration (the *ataxic* or *typhoid condition*) and quite unconscious.

Murchison also recommends 20 grains of chloral with a drachm of syrup of orange-peel and seven drachms of peppermint water as a hypnotic for an adult fever patient, and I have found chloral very useful even in young children, given in this form—

℞. Syrup. Chloral (B. P.), ʒj ;
Syrup. Simplicis, ℥vij.

M. ft. mist. *Signa :* "A teaspoonful every hour until sleep sets in."

At the same time, and notwithstanding the *prestige* of Murchison's name, I have come to the conclusion that chloral is not a safe hypnotic in fever, and it must always be given *most cautiously* in typhus, or where there are any signs of failing heart. Other

sedatives sometimes employed are the bromides of sodium, potassium, and ammonium; belladonna, henbane, Indian hemp, chloroform, camphor and musk (the last especially in the ataxic state). A prescription of Graves may be quoted—it was a draught, to be taken every two hours, containing half a grain of tartarated antimony (tartar emetic), ten grains of musk, five grains of camphor, and tend rops of laudanum. After taking three such doses the patient fell into a quiet sleep, and awoke quite rational.[a] Dr. Daniel Lysons, of Bath, in the year 1771, prescribed the following camphor draught in fever with good effect:—

℞. Camphoræ, gr. 20;
Spt. Rectificati, min. vj;
Pulv. Gummi Acaciæ, gr. 20;
Potassii Nitratis, gr. 10;
Sacchari Albi, gr. 60;
Aquæ, ʒij.

M. ft. haustus.

When prescribing the bromides, it is well to order them to be freely diluted. They may be given in full doses, even up to 60 grains, at least two hours before sleep is expected.

In cases of profound **stupor**, attention should be directed to the **state of the bladder**. Timely use of the catheter may at once relieve retention of urine and cerebral oppression, and obviate a troublesome cystitis. An attempt should be made to rouse the patient by external stimulating applications—rapid blistering of the shaven scalp or forehead by painting with liquor epispasticus or acetum cantharidis, or by applying lint saturated with strong solution of ammonia under oiled silk for five or six minutes; the application of sinapisms to the inside of the thighs, the soles of the feet, the epigastrium, or the nape of the neck—are all efficient means. Personally, I have the greatest faith in wrapping the lower extremities and waist in flannels wrung out of mustard and hot water. The cold effusion or douche, as already described, is highly recommended by Dr. Armitage and Dr. Todd. Among drugs, valerian and phosphorus

[a] Graves. *Loc. cit.* Page 140.

have been highly spoken of for the coma of typhus, but Murchison "damns them with faint praise." Phosphorus, in particular, is lauded by Huss (1855) as a remedy in cases of extreme torpor and prostration. It may be given dissolved in almond oil in doses of one-twelfth of a grain every second or third hour.

Convulsions are often transitory at the beginning of a specific fever, but they constitute a very dangerous and intractable symptom in the later stages, when they are often of uræmic origin or usher in some complication. The bowels should be freely moved by a dose of calomel or croton oil (except in typhoid fever), the patient should be aroused from stupor as above recommended, the state of the bladder should be attended to, and congestion of the kidneys should be relieved by dry cupping and poulticing, the hot-air bath, or the hot-pack, while their action is promoted by copious draughts of water, saline diuretics, and digitalis.

Hyperæsthesia may be relieved by warm fomentations, or dry rubbing (shampooing), or by hypnotics and anodynes.

Rheumatoid and neuralgic pains may be treated in the same way as hyperæsthesia, or a few moderate (5-grain) doses of phenazone or of quinine may be given.

Thirst is best assuaged by draughts of cold water, if need be slightly acidulated or embittered with cascarilla or quassia (Murchison). According to the late Dr. R. D. Lyons, of Dublin, camphor is often a specific against thirst. It may be given in the form of camphor water.

Persistent vomiting is best treated by applying a sinapism to the pit of the stomach or nape of the neck, the administration of ice, lime-water, Schacht's solution of bismuth, magnesium, or effervescing draughts with dilute hydrocyanic acid. The bowels should be kept free.

Tympanites or **meteorism** (flatulent distension of the abdomen from inflation of the intestines with gas) and **hiccough** may be treated with antispasmodics internally—such as 30-minim doses of aromatic spirit of ammonium or of compound spirit of horse-radish, turpentine, peppermint with carbolic acid, &c.; or with enemata of turpentine, asafœtida, and rue; of creasote or carbolic acid. The

best external applications are hot fomentations, poultices, turpentine-epithems, and above all, ice-poultices, as recommended by Dr. Peter, of Paris. Dr. Cayley says the ice-poultice may be conveniently applied by putting small pieces of ice between two folds of flannel. This is especially useful in intestinal hæmorrhage in typhoid fever. Failing all these means, the long tube should be passed as far up the intestine as possible, and the colon even has been punctured to give vent to the pent-up gas.

The treatment of **diarrhœa** and of **intestinal hæmorrhage** will require special mention when we come to the subject of typhoid fever, in which disease they most frequently present themselves.

6. In the management of the various complications and sequelæ of the different infective febrile diseases "we must" (as Murchison says, when speaking of Typhus), "be guided by general principles and by the symptoms in the individual case, never forgetting that the primary disease" (and this is true in varying degrees of all the diseases we are considering) "has a tendency to induce great nervous prostration and depression of the heart's action, which forbid all depleting or lowering measures."[a]

The subject of **the treatment of complications and sequelæ** will be more conveniently discussed under the headings of the respective infective fevers.

[a] *Loc. cit.* 306.

PART II.

THE EXANTHEMATA, OR ERUPTIVE FEVERS.

PART II.—THE EXANTHEMATA, OR ERUPTIVE FEVERS.

CHAPTER VI.

General Considerations.

Cullen's classification of Diseases.—Four Orders of Pyrexiæ.—Definition of the Exanthemata.—Meaning of the term ἐξάνθημα.—Enumeration of the common acute Micro-parasitic Diseases which are called "Eruptive Fevers."—Smallpox, the "paradigm" or type.

So far as I can ascertain, the expression "**Exanthemata**, or **Eruptive Fevers**" was first introduced by Dr. William Cullen, Professor of the Practice of Physic in the University of Edinburgh, in his "Synopsis Nosologiæ Methodicæ," published in 1784.[a] He divided all diseases into four classes—*Pyrexiæ*, *Neuroses*, *Cachexiæ*, and *Locales*. His definition of the first is excellent, as far as it goes—"Post horrorem pulsus frequens, calor major, plures functiones læsæ, viribus præsertim artuum imminutis." ("After a shivering fit, a rapid pulse, increased body heat, impairment of many functions, diminished strength, especially of the limbs.") He arranged the Pyrexiæ in four orders—1. *Febres*, including the Intermittent and the Continued Fevers. 2. *Phlegmasiæ*, or Inflammations. 3. *Exanthemata*, or Eruptive Fevers. And 4. *Hæmorrhagiæ*, or Bleedings and Catarrhal Fluxes.

Cullen's definition of the Exanthemata is well worth quoting. It runs as follows:—"**Morbi contagiosi, semel tantum in decursu vitæ aliquem afficientes; cum febre incipientes; definito tempore apparent phlogoses, sæpe plures, exiguæ per cutem sparsæ.**" This may be translated freely: "Infectious, or taking, diseases attacking an individual (as a rule) once only in the course

[a] See *The Works of William Cullen, M.D.* Edited by John Thomson, M.D., F.R.S., L. & E. In two volumes. William Blackwood, Edinburgh. 1827. Vol. I., pages 243 and 276.

of his life, setting in with fever, and characterised by the appearance at a definite time of an inflammatory red rash, consisting of papules, often many in number, of small size, scattered over the skin."

It will be observed that Cullen was careful not to include typhus and typhoid fevers, which he called respectively "Typhus petechialis" and "Typhus mitior," or "Synochus" *pro parte* (Murchison), among the Exanthemata. It is true that some modern writers do not follow his example in this matter. For example, Dr. William Squire, in an otherwise excellent article on the "Exanthemata," in Quain's "Dictionary of Medicine," includes both these fevers among the *true* exanthemata, which he rightly says "are acute specific infectious diseases."

In the present work I intend to follow Cullen's example, which has been endorsed by such high authorities as Murchison, Strümpell, and, apparently, Trousseau.

It is indeed true that I look upon the rose spots of typhoid and the maculæ of typhus as true rashes, but their appearance is comparatively inconstant. The classification of these fevers with the Exanthemata would further have the awkward result of leaving Relapsing Fever in a class by itself, unaccompanied, as it is, by any rash yet running a definite course like typhus and typhoid, and certainly due, like them, to a specific cause.

The Greek term *ἐξάνθημα*, which means an **efflorescence**, or an eruption or rash, was used by Hippocrates in this sense. It is, therefore, classical. It is derived from the verb *ἐξανθέω*, *to put out flowers* (*ἄνθος*, *a blossom*, *a flower*).

The common acute micro-parasitic diseases, or infective fevers, which are characterised by the appearance at a definite period in their course of a rash upon the skin, are—(1) **Smallpox**, (2) **Chickenpox**, (3) **Measles**, (4) **Scarlet Fever**, (5) **Epidemic Rose-rash**, (6) **Erysipelas**. Of the entire group Smallpox is always taken as the paradigm,[a] or pattern, and we will accordingly begin with a description of this terrible disease.

[a] Gk. *παραδείγμα*, a *pattern*, *model*, *example*. Latin: *Exemplar*.

CHAPTER VII.

Variola, or Smallpox.

Nomenclature.—Definition.—Ætiology (historical sketch).—Exciting cause : contagion.—Predisposing causes : susceptibility, from non-protection, season, race.—Bacteriology of Smallpox.—Modes of dispersion of the morbid poison.—"Striking Distance" of the disease.—Chief Stages of Infectiveness.—Clinical History : Incubation, Invasion, Eruption (its five stages of development : Specks of Hyperæmia, Papules or Pimples, Vesicles, Pustules, Scabs or Crusts), Desiccation, Desquamation.—Variolous Exanthem upon the Mucous Membranes, but not upon the Serous Membranes.

Nomenclature—Smallpox.—*Synon.* :—Variola (from *Lat.* Varus, *a blotch, a pimple*); *Germ.* Blattern, Pocken; *Fr.* Petite Vérole, Picote,[a] Variole; *Ital.* Vaiuolo; *Span.* Viruelas; *Danish and Norwegian*, Kopper; *Swedish*, Smittkoppor.

"Pox" is one of the plural forms of "Pock," derived from the Anglo-Saxon *poc* or *pocc*, a bag or pouch. Chaucer has this line—

"Of *pokkes* and of scab and every sore."

The English term **Smallpox** was introduced at the end of the 15th century to distinguish the disease from Syphilis. **Varioloid** is *modified Smallpox*—the result of (*a*) a previous attack, (*b*) inoculation, (*c*) vaccination. In this variety of the disorder the febrile movement is usually moderate, and there is little or no secondary fever.

Definition.—An acute specific, infectious febrile disorder, setting in suddenly with chills, headache, sweating, vomiting, tenderness on pressure in the epigastrium,[b] and severe pain in the lumbar region; characterised by a certain typical range of temperature, a specific inflammation of the skin (dermatitis) and often of the mucous membranes also. The dermatitis is shown by the development, usually on the third day, of a papular or pimply rash, which quickly becomes vesicular and finally pustular; the appearance of the rash is followed by a fall of temperature, but a secondary fever

[a] A man marked with Smallpox is said to be *picoté*.

[b] "A Pain at the Cavity of the Breast beneath the Region of the Heart." (Sydenham).

accompanies the development of pustules at the end of the first week. These pustules finally dry up (desiccation) and form crusts, which are shed or cast off about the eighteenth day. The disease is not infrequently complicated with hæmorrhages into the skin (*Purpuric Smallpox*) and from the mucous membranes (*Hæmorrhagic Smallpox*).

Ætiology.—The origin of Smallpox is unknown. It was first described by the Arabian physician, Rhazes, who flourished in the tenth century. Rhazes speaks of the Smallpox as a disease generally known over the East, but the occurrence of the malady on European soil cannot be traced back with certainty beyond the Christian era. The native foci of smallpox may be looked for in India and in the countries of Central Africa; from these original habitats its diffusion over the earth's surface has been brought about by successive importations of the morbid poison. At the present time the dominion of smallpox extends over almost the whole inhabited globe, and only a few isolated regions still enjoy a complete immunity from it.[a] It is generally believed that the disease first appeared at Pelusium, in Egypt, A.D. 544 (Procopius: *de Bello Persico*, Lib. II. cap. 22). Its diffusion, eastward and westward, was probably effected by the Saracen armies, led forth to conquest by Mahomet at the era of the Hegira, A.D. 622. The name "**Variola**" occurs for the first time as a designation of the disease in a description by Marius, of Avenches, of an epidemic which was widely prevalent in France and Italy in the year 570.[b]

The researches of antiquarians lead to the belief that smallpox first appeared in England about the year 900. All authors concur in representing the dreadful mortality occasioned by this pestilence and the consequent terror which its visitations everywhere excited.[c] To Boerhaave, of Leyden, belongs the credit of assigning **contagion** as the **proper exciting cause** of the disease.

[a] Cf. *Handbook of Geographical and Historical Pathology.* By Dr. August Hirsch, New Sydenham Society, 1883. Vol. I., 123, *et seq.*

[b] "Chronicon" in Bouquet's *Collections des Historiens de France.* Paris. 1738. Vol. II., page 18. [Quoted by Hirsch, *loc. cit.* p. 126].

[c] *Elements of the Theory and Practice of Medicine.* By George Gregory, M.D. Fourth Edition. London: Baldwin and Cradock. 1835. Page 105.

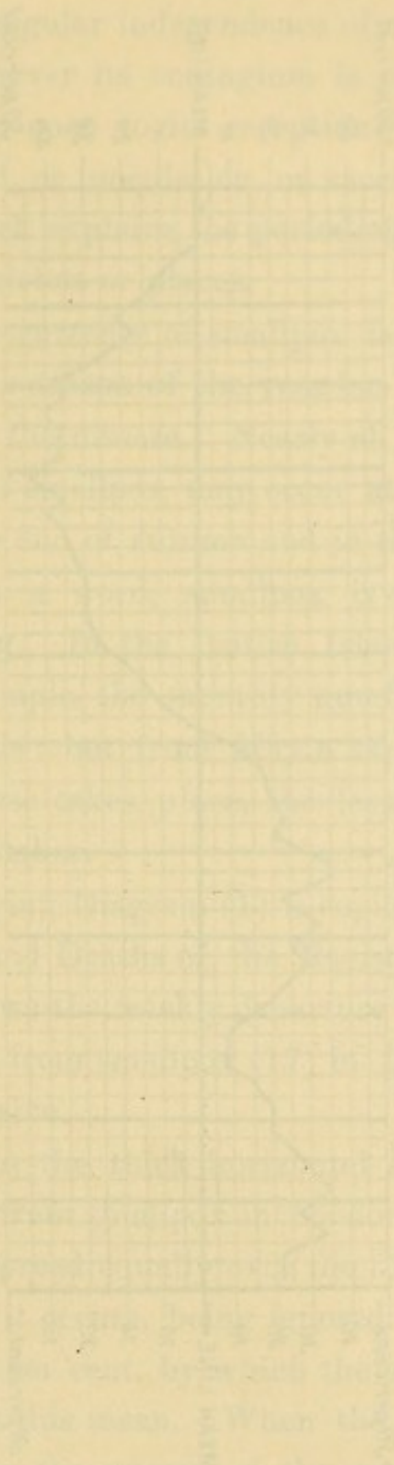
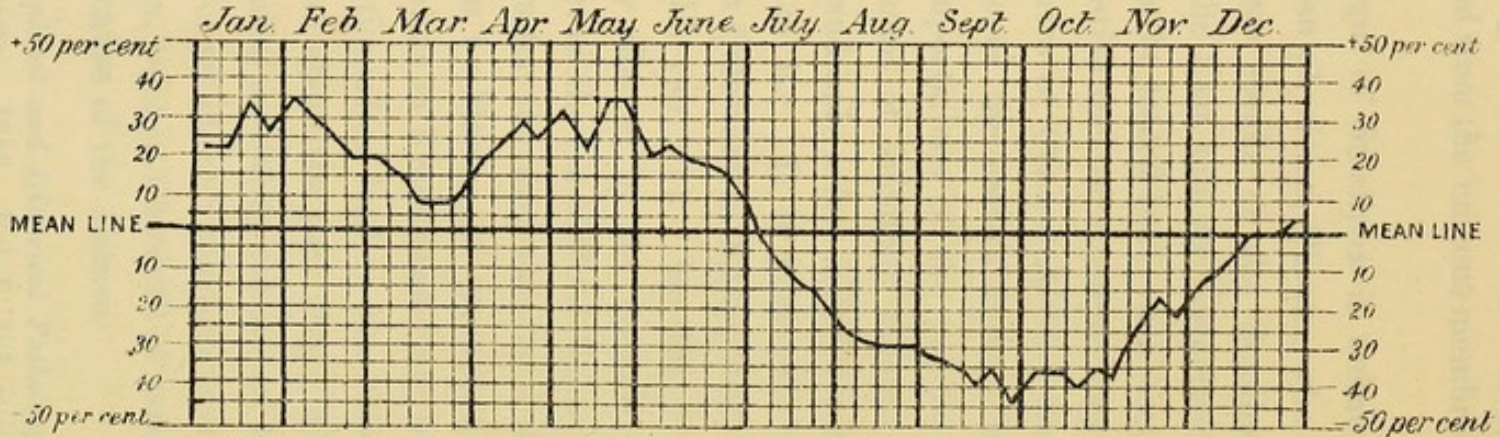


DIAGRAM I. — SMALLPOX.

(50 Years 1841-90)



The mean line represents an average weekly number of 17 deaths.

Hirsch is of opinion that there are two factors only which determine the recurrence of an epidemic of smallpox—on the one hand, the necessary number of persons susceptible of the morbid poison, and, on the other hand, the introduction of the virus itself. The disease shows a singular independence of climate and soil, thriving equally well wherever its contagium is carried, and wherever it finds a population open to its reception—being unprotected by a previous visitation, or inoculation, or vaccination. It is this latter circumstance which explains the **periodicity** of epidemics of smallpox in various districts or places.

Although the occurrence of smallpox is apparently **independent of climate**, yet the **season of the year** has a marked influence upon the prevalence of the disease. Nearly all writers are agreed that, while outbreaks of smallpox may occur at all seasons, they mostly begin towards the end of autumn and in the early spring, or in the cold season. In a word, smallpox is essentially a **disease of winter and spring.** In the British Islands, and Western Europe generally, for example, the monthly number of cases is high from November onwards; but from May a rapid decline in the prevalence of the disease takes place, the least number of cases being observed in September.

The accompanying Diagram (1) is copied from the Annual Summary of Births and Deaths of the Registrar-General for England for 1890. It shows the weekly departure from the average weekly number of deaths from smallpox (17) in London in the fifty years, 1841–1890, inclusive.

In this diagram the thick horizontal line represents the mean weekly mortality from smallpox in London, on the supposition that the mortality is spread equally over the 52 weeks of the year—the 53rd week, when it occurs, being ignored. The curved line represents the amount per cent. by which the average mortality in each week differs from this mean. When the percentage for any week is above the mean, the amount of the percentage excess is marked above the horizontal line representing the mean; and when the percentage is below the mean it is marked below the line.

It must be remembered that the data on which the curve is

formed are the deaths registered in each week, not the deaths which occurred in the week, and that the registration is usually a few days after the death; and, secondly, that the curve relates to deaths—that is, the final termination of the attack of illness, and not its commencement. So that, in estimating the effect of season in generating smallpox, allowance must be made for the average duration of this disease when fatal—that is, 11 or 12 days. It is, moreover, possible that the curve of mortality may, for another reason, not accurately represent the curve of prevalence. For it may be that an attack of smallpox is more likely to terminate fatally if it occurs at one season—for example, midwinter—than if it occurs at another, such as midsummer.

The diagram shows that at the beginning of February and in the second half of May the weekly number of deaths was 35 per cent. in excess of the average weekly number of 17 deaths represented by the mean line, whereas at the end of September there was a deficit of 43 per cent. in the weekly number of deaths as compared with the same average weekly number over the whole year.

In Dublin, during the autumn of 1871, the prevalence of, and mortality from smallpox increased with a fall of mean temperature below 50°, and the greatest severity of the epidemic was experienced in the first half of the following April, shortly after a period of intense cold for the time of year. With the rise of mean temperature to between 55° and 60° in the middle of June, 1872, the epidemic declined rapidly. Abundant rainfalls seemed to be followed by remissions in the severity of the epidemic, and the converse was also true.[a]

Individual susceptibility to the poison of smallpox extends to the whole of mankind, but experience shows that the coloured races, and particularly the negro race, are—other things being equal—in greater risk from smallpox than the whites.[b]

[a] *Manual of Public Health for Ireland.* Dublin: Fannin & Co., 1875. Page 298. See also Buchan's and Mitchell's Paper in the *Journal of the Scottish Meteorologial Society.* 1874.

[b] Pruner. *Die Krankheiten des Orients.* Erlangan. 1847. Page 120. [Quoted by Hirsch, *loc. cit.* Page 151.]

The **bacteriology** of Smallpox is still incomplete. According to Edgar M. Crookshank,[a] cocci, ·5 μ[b] in diameter, singly, in pairs, and in long or short chains, and colonies have been found by Cohn (Virchow's *Archiv*, 1872) in the fresh lymph of human and cowpox, and in the pustules of true smallpox. They are regarded as the active principle of vaccine lymph, since filtration deprives this of its infectious element (Chauveau. *Comptes Rendus*, 1868). Burdon Sanderson confirmed this observation. The lymphatics of the skin in the region of the pustule of both human and sheep-pox (*Variola ovina*) are filled with cocci (Weigert and Klein). Successful vaccination has been stated by Quist (*St. Petersburg med. Wochenschrift*, 1883) to result from artificial cultivations. These cocci have been called *Streptococcus*[c] *variolæ et vaccinæ*. Loeff and Pfeiffer (*Centralbl. für Bacteriologie und Parasitenkunde*, 1887) have discovered in the blood of smallpox patients certain protozoa, to which they attribute a pathological significance.[d]

"The *dispersion of the morbid poison*," writes Hirsch,[e] "takes place either by the smallpox patients themselves, or through the medium of other persons, or of articles to which it clings. It has been conclusively proved by very numerous and unambiguous observations that an atmosphere of smallpox poison develops around the sick, especially when they are crowded in close rooms; or, in other words, that the air may become a carrier of the contagion, so that the latter can be spread by the atmospheric currents within a small range. There is certainly no mathematical expression to be found for the extent of that range; at the utmost, it extends no farther than the immediate surroundings of the sick."

Sir Thomas Watson[f] mentions a remarkable instance of smallpox

[a] *Manual of Bacteriology.* London: H. K. Lewis, 1887. Page 203.

[b] The Greek letter μ represents one-thousandth of a millimetre ($\mu = 0{\cdot}001$), and is the sign of a micro-millimetre or micron.

[c] Gk. στρεπτός, *pliant.* Hence *chain-armour.* Streptococci are micrococci which arrange themselves in chains.

[d] *Clinical Diagnosis.* By Dr. Rudolph v. Jaksch. Translated by James Cagney, M.A., M.D. London: Charles Griffin & Co. 1890. Page 35.

[e] *Loc. cit.* Page 152.

[f] *Lectures on Practice of Medicine.* Second Edition. London, 1838. Vol. II., page 786.

being caught from the dead body. The corpse of a man who had died of the disease was brought into Mr. Cæsar Hawkins' dissecting-room in Windmill-street, London, with the result that four students took the disease, although only one had *touched* the body.

In a discussion on the mode of preventing the spread of epidemic disease from one country to another, in the Section of Preventive Medicine at the Seventh International Congress of Hygiene, London, 1891, Dr. Hewitt, of Minnesota, U.S.A., recorded a still more striking case, in which a female immigrant attended a smallpox patient on board ship while crossing the Atlantic from Liverpool to New York, then doffed and put by her clothes, which she again began to wear some time afterwards while attending a sick child in a distant part of the country, with the result that this child sickened of smallpox, the disease spreading until the *fatal* cases numbered 100. All the time the woman, whose clothes spread the disease, remained perfectly well and free from smallpox.

The experience of recent epidemics—those of 1871–73 and of 1876–79—in the British Islands proves that the **striking distance** of smallpox is considerable—certainly much greater than that of typhus fever.

In his "Manual of Public Health,"[a] Mr. A. Wynter Blyth observes: "The usual spread of smallpox is from person to person, but from inquiries which have taken place as to the influence of smallpox hospitals upon a surrounding population, and the experience of the Sheffield epidemic (of 1887–1888), it is certain that the infection can strike at a distance. Whether the contagious particles are conveyed by the air itself, or by the medium of the common household fly or other insects, the important fact remains that infection may travel far. The influence of the Sheffield Hospital could be distinctly traced for a circle of 4,000 feet; for instance, the following percentages of households attacked at successive distances from the hospital are given in the original

[a] London: Macmillan & Co. 1890. Page 369.

Report (by Dr. Barry, Inspector of the Local Government Board for England)[a]:—

0-1,000 ft.	1-2,000 ft.	2-3,000 ft.	3-4,000 ft.	Elsewhere.
1·75	·50	·14	·05	·02

This possibility of smallpox spreading by aerial infection increases greatly both the hospital difficulty and that of individual isolation."

There can be no doubt that the **chief stages of infectiveness** in smallpox are the earliest period of suppuration (Curschmann),[b] and the *Stadium Decrustationis* of Hebra—the stage of *desiccation*, or *scabbing*.

Dr. William Heberden, in his "Commentaries on the History and Cure of Diseases,"[c] says: "Many instances have occurred to me which show that one who has never had the smallpox may safely associate, and even lie in the same bed with, a variolous patient, for the two or three first days of the eruption, without any danger of receiving the infection. One woman continued to suckle her infant for two days after the smallpox had begun to appear upon her; and the child being then removed escaped the distemper for that time, but was unquestionably capable of being infected, because he *catched* it about a year and half after."

Clinical History.—For convenience of description we may divide the course of smallpox into five stages—(1.) Incubation; (2.) Invasion; (3.) Eruption; (4.) Secondary Fever; (5.) Desiccation and Desquamation.

I. Stage of Incubation.—This begins with the reception of the virus into the system and ends at the appearance of the earliest symptoms. Its average duration is 12 days, except in cases of inoculation, when it is only 8 days, or still shorter—48 hours, according to Curschmann. As a rule, there are no symptoms in this stage, but towards its close the patient probably feels unwell and out of sorts—what the French aptly call *malaise*.

[a] *Report of an Epidemic of Smallpox at Sheffield*, 1887-8. By Dr. Barry, Local Government Board. London, 1889.

[b] Cf. Von Ziemssen's *Cyclopædia of the Practice of Medicine*. Art. "Smallpox." By Heinrich Curschmann, of Giessen. London: Sampson Low, Marston, Low, & Searle. 1875. Vol. II.

[c] London: T. Payne. Second Edition. 1803. Page 437.

II. Stage of Invasion.—Smallpox sets in suddenly and with violence. As is usual in febrile disorders, the earliest symptoms are connected with the nervous system. They are *rigors*, varying from a mere feeling of chilliness to a downright shivering fit, with chattering teeth, pallor, blueness of the extremities, and "goose-skin" (*cutis anserina*), and, in young children, *convulsions*. More or less intense pain is complained of in the lumbar region, or in the centre of the sacrum (*rachialgia*), apparently due to hyperæmia of the spinal cord, for it is almost always relieved by hæmorrhage, natural or induced. *Rheumatoid pains* in the limbs are also often felt, so that the question of acute rheumatism has had to be taken into account in the diagnosis (Hilton Fagge). Severe *headache*, often *delirium*, and sometimes *partial paraplegia*, *numbness*, *retention* or *incontinence of urine* and *incontinence of fæces*, may be also present. In children, *diarrhœa*, *sleepiness* or *drowsiness* and *stupor* may occur.

On the first or second day the temperature rises to a considerable height (104° F.=40° C., seldom below this, sometimes above it, even to 105·8° F.=41° C.—Wunderlich). The maximal temperature (*fastigium* or *acme*) is usually reached shortly before the rash appears on the third day. This initial fever is called the *Prodromal Fever*, because it runs before, or precedes the appearance of the rash.[a] The other most prominent symptoms are also due to the influence of the poison on the nervous system; they are—*pain in the epigastrium*, or pit of the stomach, *nausea* and often *vomiting*, *constipation* (except in children) and constant profuse *sweating*. There are, besides, *loss of appetite* (*anorexia*), *thirst*, *furred tongue*, very *fœtid breath*, *full* and *rapid pulse*, and *prostration*. In women, *menstruation* nearly always comes on, whether the period is due or not, and it is generally profuse (*menorrhagia*). All these symptoms are, as a rule, more acute and prolonged in confluent than in discrete smallpox.

During this stage *accidental rashes* are apt to appear, causing much difficulty in diagnosis. They are usually erythematous in character—if diffuse, resembling scarlatina or erysipelas; if spotty

[a] *Prodromal*—i.e., *running before, preceding;* from Gk. *πρό, before; δρόμος, a course, running, race.*

or macular, resembling measles. Rayer long ago gave to these prodromal rashes the name of *Roseola variolosa*, and it was adopted in 1853 by Eimer. Dr. Wilks drew attention to these accidental rashes in "Guy's Hospital Reports" for 1857 and 1861.

These prodromal or initial erythematous rashes are very evanescent, they usually usher in an attack of varioloid or mild smallpox, and so have no little prognostic importance—but they are not pathognomonic of smallpox, as they show in other specific fevers also—for example, in typhoid fever and in measles. They probably depend on a reactive inhibition of the vaso-motor system of nerves, brought about by the fever poison. Hebra and Trousseau, as well as Curschmann, seem to have independently remarked that the parts affected by a *Roseola variolosa* afterwards remain free from the proper smallpox rash. This observation has been generally confirmed by subsequent writers (Hilton Fagge).

A more serious phenomenon is the development, even at this early stage, of *petechiæ*, or subcutaneous extravasations of dissolved hæmatin, varying in size from a pin's head to a pea or a bean. These *purpuric* rashes are commonly seen in the brachial and crural triangles of Th. Simon, of Hamburg[a]—the former embracing the lateral thoracic region, including the inner side of the arm, the axilla, and the pectoral region; the latter embracing the lower abdominal region, including the hypogastrium, the groins, and the inner aspect of the thighs. The prognosis in such cases is generally, but not necessarily, more grave.

The duration of the stage of invasion is on the average three days—as a rule, it is prolonged in the milder, shortened in the severer cases. Sydenham,[b] speaking of *confluent smallpox*, observes: "This kind of smallpox generally comes out on the third day, sometimes earlier, but scarce ever later, whereas the *distinct* appears on the fourth day inclusive from the beginning of the distemper, or later, but very rarely before, and the sooner the pustules come out

[a] Th. Simon, *Das Prodromalexanthem der Pocken. Arch. für Dermatologie und Syphilographie.* II. Jahrgang. Page 347, *et seq.*

[b] *The Entire Works of Dr. Thomas Sydenham.* By John Swan, M.D., London: F. Newbery. 1769. Page 105.

before the fourth day the more they run together." To the same effect, Boerhaave wrote[a]: "Most practitioners observe that the slower the smallpox come out, the milder they prove and the better they ripen. Those appearing on the first day of the illness are esteemed the worst kind, those on the second, milder, those on the third still more gentle, and on the fourth, the most favourable."

Trousseau and nearly all modern authorities concur in this view, but Curschmann thinks it is not warranted for all epidemics. It may, however, be accepted as a sound working proposition, although there is one striking exception to it. In consequence of great organic lesions—as Sydenham writes, *ob atrocius aliquod symptoma*—the eruption may be retarded till the sixth or seventh day both in distinct and confluent cases—"*malo semper omine.*" Trousseau[b], in illustration, mentions the case of a woman, aged 30, in whom the rash did not appear until the fifth day. At the beginning of her attack of smallpox she had all the symptoms of sporadic cholera—vomiting, purging, cramps, general coldness, blanching of the mucous membranes, dry, cold tongue, injection of the conjunctiva, and a dull appearance of the cornea. The choleraic symptoms ceased on the fourth day, and on the fifth the eruption of smallpox appeared. Again, a long experience in the wards of Cork-street Fever Hospital, Dublin, has led me to the conclusion that a purpuric or hæmorrhagic tendency early in smallpox postpones—it may be indefinitely—the appearance of the true variolous exanthem. To this subject it will be necessary to return.

III. **Stage of Eruption.**—The true exanthem, or rash, of smallpox appears first on the head, neck and face, and about the wrists, next on the trunk, lastly on the lower extremities. In severe cases, as has been already mentioned, it shows itself on the second or even on the first day; in mild cases its coming may be postponed until the fourth day. The usual day for its appearance is the *third, inclusive*, from the earliest symptoms. When the "pocks"

[a] Boerhaave. *Prax. med.* Vol. V., page 302. [Quoted by Sydenham, *loc. cit.*]

[b] *Clinique Médicale de l'Hôtel Dieu de Paris.* Paris: J. B. Baillière et Fils. 1865. Deuxième Édition. Tome premier, page 6.

fulfil their whole life-history, they are seen to pass through the following stages of development :—

1. **Specks of Hyperæmia**, like the fine pricks made with a needle, and sometimes like recent fleabites (first day of rash).

2. **Papules**, or **Pimples**, such as are met with in persons affected with lichen or prurigo (Trousseau), at first slightly raised, then conical, already hard or "*shotty*" to the touch, feeling like grains of *shot* underneath the skin (second and third days of the rash).

3. **Vesicles** filled with a clear, transparent fluid at first, but which quickly becomes lactescent or milk-like. "By the fourth or fifth day of the eruption (seventh or eighth of the disease)," continues Dr. Hilton Fagge, "the vesicle is generally as large as a split pea, hemispherical in form, and opaline in appearance."

4. **Pustules** formed by a further change in the contents of the vesicles in which young cells increase and multiply, causing them to assume a more and more opaque and yellow appearance, and to increase quickly in size (sixth and seventh days of the rash). About this time a central depression is found in these pustules. This is the so-called umbilicus, at the bottom of which the opening of a hair follicle or sweat gland is frequently observed (Curschmann).

This carries us on to the 10th day of the disease, when the rash has reached its fullest development. The pustule, when at its height, is often quite hemispherical, the umbilicus having disappeared in consequence of the rupture of the retinaculum which formed it. These pustules are really small abscesses. They become extremely painful, and the pain is accompanied by great swelling of the affected parts—greatest where the tissues are loosest or most relaxed, as in the eyelids and lips, and about the prepuce. In the confluent variety, about to be described, the face swells to a shapeless mass, rendering the patient absolutely unrecognisable.

Each pustule is now surrounded with an inflammatory zone or **areola**, called the **halo** of the pustule.

This period of fullest development of the rash is called the period of **maturation**, or **ripening**. It lasts about three days and is followed by—

5. The last stage in the life history of the eruption, that of **desiccation**—the drying-up of the pustules—and the formation of **crusts** or **scabs.**

IV. **Stage of Desiccation.**—Even before the eleventh day an exudation of a sticky fluid takes place, and a yellowish matter like thick honey oozes from the surface of the pustules. This, together with their other contents, speedily dries up, first in the centre, and brownish scabs are formed, which are at first adherent, but afterwards fall off in from 3 to 6 days, leaving elevations or projections of a violet red hue, like *a cold skin.*

On the trunk and extremities, where desiccation begins later than on the face, the pustules frequently burst and their purulent contents, soaking into the bed and body linen, under godecomposition upon the skin and in the clothing, causing an overwhelming **stench** about the 11th or 12th day of the disease. This occurs in the confluent, not in the discrete, form. With the drying-up of the pustules, the redness, swelling, and tenderness of the skin subside, the eyes reopen, the nostrils are cleared, and the features of the patient become once more recognisable.

V. **Stage of Desquamation.**—After this, successive scales of epidermis form and peel off—a process which is called **desquamation**—ultimately leaving a small white puckered scar (cicatrix) should the variolous inflammation of the skin (dermatitis) have dipped deep and involved the papillary portion, or *cutis vera.* When every scab has fallen off and desquamation has ceased, the patient may be considered free from infection.

About the same time that the rash of smallpox shows on the skin, a **true variolous exanthem** develops **upon the mucous membranes** in general. Of this we have ocular proof in the case of those parts of the mucous membranes which are visible and are most exposed to the air. Thus, the conjunctivæ, the mucous membranes of the nose, mouth, pharynx and adjacent parts, are nearly always affected. Thence the rash extends through the whole system of mucous membranes, invading the larynx, trachea, and bronchi in one direction, the œsophagus, stomach, and intestines in another. It is true that Curschmann states that it is very doubtful if

real pustules are ever formed in the stomach and intestines, although they have been described by the older authors (Robert, in his account of an epidemic in Marseilles). He adds that they are seen only in the lowest part of the rectum, close to the anus. Nevertheless, for many years I have had an opportunity of illustrating my lectures on Smallpox by exhibiting a series of beautiful original drawings, by the late Mr. J. Conolly, of the pathology of this disease. These drawings show the entire respiratory and digestive tracts thickly beset with a variolous eruption, presenting itself as whitish or pearly-gray elevations upon a reddened base, with abrasions or large irregular excoriations of the mucous membrane. During life the presence of such an eruption is evidenced by such symptoms as deafness, due to blocking of the Eustachian tubes (Wendt); hoarseness or aphonia; cough and dyspnœa; dysphagia, or difficulty of deglutition; diarrhœa.

According to Curschmann, **true pocks upon the serous membranes** are **fables** belonging to antiquity.

CHAPTER VIII.

SMALLPOX (*continued*).

CLASSIFICATION AND VARIETIES.

Classification of Smallpox based upon the distribution and amount of the Rash: VARIOLA DISCRETA, CONFLUENS, COHÆRENS, CORYMBOSA.—Symptoms of Confluent Smallpox: its mortality and sequelæ.—Meaning of the terms "Semi-confluent" or "Coherent," and "Corymbose," Smallpox.—The latter said to be a very fatal variety.

BEFORE describing the behaviour of the **temperature** during the eruptive and subsequent stages of Smallpox, we may refer to a **classification** of the varieties of the disease **based upon the distribution and amount of the rash**, which has been handed down from the time of Sydenham, and has received universal acceptance.

Under all circumstances, whether modified or unmodified, Smallpox appears under two principal forms—**Discrete** or **Distinct**, and **Confluent**. The first of these is generally free from danger; the latter is one of the most terrible and fatal of diseases. Of confluent smallpox two modified varieties are described—namely, (1) **Semi-confluent**, or **Coherent Smallpox**; and (2) **Corymbose Smallpox**.

Variola discreta, vel **distincta**, is the name given to those cases in which the rash is sparse or scanty, the several papules or pustules being more or less widely *separated* from each other. Hence the term *Discrete* (from Lat. *discerno, I separate*). In this form the initial symptoms are, as a rule, less acute and less persistent, and the rash not infrequently stops short of the pustular stage (*Variola crystallina*).

Variola confluens is the term applied to those cases in which the rash overruns the entire, or nearly the entire, surface of the body, and invades the mucous membranes also with great severity. The symptoms of the invasion stage are all intensified, and the rash appears as early as the second day. As additional and very characteristic symptoms, Trousseau mentions, (1) *persistent diarrhœa*, both in adults and in children; (2) *profuse salivation* in adults,

resulting either from parotitis, or, as a reflex symptom, from inflammation of the mucous membranes of the mouth—stomatitis (Curschmann); (3) *great tumefaction of the face and eyelids*, so that the latter sometimes burst or slough; and (4) *most painful swelling of the hands and feet.*

Salivation, or ptyalism, sets in early, and is extraordinarily profuse. According to Trousseau, from one to two litres (that is, from 35 to 70 fluid ounces) of clear, but ropy or viscid, saliva may flow from the patient's mouth within twenty-four hours, and this ptyalism is accompanied by a burning, unquenchable thirst.

To omit special mention of the **delirium** of confluent smallpox would be misleading. In the epidemics of 1871 and 1878, in Cork-street Fever Hospital, Dublin, the occurrence of delirium was one of the commonest and most striking features of the disease. It was often violent and noisy (*delirium ferox*); or busy, with extreme muscular agitation (*delirium tremens*); but in the later stages it assumed more of the low muttering type (*typhomania* of Galen). Both at the Meath Hospital and at Cork-street Hospital, attempts at suicide were made by delirious smallpox patients, and homicide has happened before now as a result of the same *delirium ferox.* Huxham, in his classical "Essay on Fevers,"[a] graphically describes the rash in these cases as consisting of pocks which are "pale, crude, pitted, and *sessile.*"

The face is covered with pustules, which run together so that the epidermis is raised by a milky sero-purulent secretion, and the face seems as if it were dipped in tallow or covered with a mask of parchment—"*Pergamenæ speciem visu horrendam* (*cutis faciei*) *exhibet*," as Morton said in his "Pyretologia."[b] It is right to mention that, in confluent smallpox, while the face and hands may be absolutely covered with pocks, in other parts of the body the eruption may be more or less discrete, the amount and intensity of the pustulation seemingly being in direct proportion to the vascularity and inflammatory state of the surface. This was pointed out several years ago in an admirable paper on the "Treatment of Smallpox" by the

[a] London: J. Hinton. 1764. Page 127.
[b] London. 1692.

late Dr. William Stokes,[a] Regius Professor of Physic in the University of Dublin. Of this fact we have two proofs—in the first place, portions of skin which have been subjected to mechanical or chemical irritation, either before infection or during the stage of incubation, invariably throw out a very abundant pustular, and frequently a confluent, eruption even in discrete cases of smallpox. Conversely, and secondly, where the vascularity of a part has been reduced by pressure, local depletion, or removal of irritation by poulticing, bathing, or other means, the eruption of smallpox is distinct even in confluent cases.[b]

The **mucous membranes**, like the skin, are the seat of a closely set rash in confluent smallpox, and very dangerous forms of secondary inflammation are apt to place the patient's life in imminent peril. *Glossitis variolosa*, or inflammation of the tongue; diphtheria, acute œdema of the glottis, intense and wide-spread bronchitis and pneumonia; violent, uncontrollable vomiting, retching, and diarrhœa, are among the evidences we have of the serious engagement of the mucous membranes.

Towards the close, should the patient survive, multiple pyæmic abscesses, erysipelas, and even gangrene may occur in those parts of the integument where the confluence is most pronounced.

The **mortality** is, of course, very great in this form of the disease, at any stage of which the patient may succumb. In some epidemics, according to Trousseau, half the patients die; in others, four-fifths, and in others, less fatal, one-third of those attacked perish. It is, therefore, the most deadly of all pestilences, yellow fever and cholera not excepted. The terrible feature of smallpox is that it kills not only in the acute stage, by dissolution of the blood or by the intensity of the fever, but also in the later stages and in convalescence. The most fatal epoch is about the eleventh or twelfth day, but even far on in the stage of desiccation death not infrequently results from exhaustion, or pyæmia, or some other complication. I will return to the subject of mortality later on.

[a] See the *Dublin Journal of Medical Science*, Vol. LIII., p. 9. Dublin: Fannin & Co. 1872.

[b] Cf. Stokes. *Loc. cit.*

Should confluent smallpox end in recovery, convalescence is very tedious, and is frequently interrupted by serious **sequelæ**, of which an "acute furuncular diathesis," as Trousseau calls it, is one of the commonest and most troublesome. It shows itself in the formation of successive crops of most painful boils, and of more or less deep-seated abscesses.

In the stage of desiccation large, ecthymatoid crusts form upon the ulcerated surface of the skin. These become detached, leaving the dermis scooped out. Successive layers of thinner and thinner crusts form, and are shed during two, three, or four weeks, the ulcerations finally cicatrising, leaving the rugged scars which seam the faces of those who have passed through confluent smallpox. To this disfigurement the term "pitting" has been applied. It is especially unsightly about the nose, the borders of the alæ nasi appearing indented, and the bridge and tip of the nose split and torn.

With the separation of the scabs, or sometimes later, the **hair commonly falls off**, in some cases in handfuls. If the variolous inflammation of the scalp has dipped deeply and involved the hair follicles to any great extent, the resulting alopecia, or baldness, may be permanent. More usually, the hair grows again, in a few cases more luxuriantly, if less smoothly, than before. It is not usual for the nails to fall off after smallpox, although the atrophic furrows across them, described and figured by A. Vogel, and mentioned by Murchison[a] as occurring in typhus fever, are not infrequently observed.

I have stated above that two modifications of confluent smallpox are recognised as regards the distribution of the rash. These are the semi-confluent and the corymbose varieties.

The term **Variola semi-confluens**, or **Coherent Smallpox**, is applied to those cases—(1) in which the pustules touch each other without coalescing, or (2) in which the eruption is confluent on and about the face, and more or less discrete elsewhere.

[a] *A Treatise on the Continued Fevers of Great Britain.* Third Edition. 1884. Page 136.

Variola corymbosa is a term applied to those cases where the pustules are confluent in patches, or *clusters* (*κόρυμβος, a cluster of fruit*), these being separated by intervals of unaffected skin. Vascular parts, like the axillæ, groins, and popliteal spaces, are often the seat of such an eruption. Hilton Fagge says that he never saw a case of corymbose smallpox, but there is a beautiful drawing of it in the collection of J. Conolly's illustrations of smallpox observed in the Hardwicke Fever Hospital, Dublin, to which I have already alluded. According to Marson, formerly of the London Smallpox Hospital, this is **a very fatal variety of smallpox**, the mortality reaching 41 per cent. Strangely enough, in the London Smallpox Hospital it was scarcely less destructive to vaccinated persons than to those who were unprotected (Hilton Fagge).

CHAPTER IX.

SMALLPOX (*continued*).

TEMPERATURE—VARIETIES—COMPLICATIONS—PATHOLOGY—DIAGNOSIS—PROGNOSIS.

TEMPERATURE : Two distinct Types of Fever in Smallpox—viz. 1. A brief continuous Fever ; 2. A relapsing Fever.—Prodromal or Initial Fever.—Secondary Fever, or Fever of Suppuration or of Maturation.—Hyperpyrexial Temperature in Fatal Cases.—VARIETIES OF SMALLPOX : Discrete, Confluent, Benign or Varioloid—1. Variola sine Exanthemate, 2. V. cornea (Hornpox), 3. V. verrucosa (Wartpox) ; Malignant (V. maligna)—1. Purpuric, 2. Hæmorrhagic (Purpura variolosa), 3. V. hæmorrhagica pustulosa of Curschmann, V. nigræ of Sydenham, V. cruentæ.—TABLE OF THE VARIETIES OF SMALLPOX. COMPLICATIONS AND SEQUELÆ, affecting the skin, eyes, ears, nose, tongue, larynx, respiratory organs, digestive organs, circulatory system, kidneys, nervous system, genitals, blood, joints.—PATHOLOGY : Morbid anatomy and histology.—DIAGNOSIS, PROGNOSIS, AND MORTALITY.

Temperature.—As pointed out by Wunderlich,[a] the fever in variola exhibits **two distinct types**, which closely correspond, however, at their commencement.

I. One of these types is a brief **continuous fever**, belonging in particular to the milder forms of variola discreta and to most cases of varioloid or modified smallpox, occurring chiefly although not exclusively in vaccinated or inoculated persons. This is the so-called **Prodromal** or **Initial Fever** of the stage of invasion. In the forms of the disease just mentioned, this continuous fever both begins and usually completes the febrile movement. The *maximal* temperature of the initial or prodromal fever is rarely less than 104° F. (40° C.) ; it generally exceeds this, reaching even 106° F. (41·1° C.). This great height is quickly attained, generally on the second day. Soon after the true rash of smallpox appears, the temperature falls more or less rapidly—usually from the fourth to the sixth day. The defervescence is either rapid and continuous,

[a] *A Manual of Medical Thermometry.* New Sydenham Society. 1871. P. 337.

or slower and interrupted by a moderate evening exacerbation. In cases of uncomplicated varioloid and of mild discrete smallpox this defervescence is complete and final.

The fall of temperature which occurs with the coming out of the rash of smallpox is **pathognomonic** of this disease, and is therefore of the first importance in diagnosis. It is exactly the *converse of the behaviour of the temperature in measles*, in which the fever is moderate up to the appearance of the rash, and then becomes more and more intense until the rash is most fully developed.

It should be borne in mind that the initial or prodromal fever is often very severe even in the mildest cases.

II. The other type of fever in smallpox is a **relapsing type**, which is characteristic of true smallpox in its severer and confluent forms. The falling temperature after the prodromal stage in this case either never reaches the normal line, remaining at subfebrile (99·5° to 100·4° F.), or even at febrile (points 100.4° to 102·2° F. in the morning, rising to 103° F. in the evening); or the normal temperature is reached, if at all, tediously, and defervescence is by lysis.

Then, with the beginning of the pustulation, or *ripening* (*maturation*) of the exanthem the temperature again begins to rise, ushering in a **Secondary Fever**—the **Fever of Suppuration** or of **Maturation** ("*Eiterungsfieber*" of the Germans) as it is called—which is of indefinite duration and varies in intensity according to the severity of the disease. In a sharp attack of discrete or semi-confluent smallpox, the temperature in this secondary fever rarely exceeds 103° or 104° F. There are morning remissions, and the duration is only a few days. In bad confluent smallpox, on the other hand, the fever runs very high, presenting sometimes a remittent course with marked exacerbations, sometimes a continuous range with occasional isolated elevations or *spikings* of temperature.

Repeated elevations of temperature above 104° F. (40° C.) during this fever of suppuration, or maturation, are a sign of great danger. In cases which tend towards recovery, defervescence takes place by an irregular lysis. In fatal cases, **hyperpyrexial temperatures** (107·6° F.=42° C.) are wont to occur before, at the moment of,

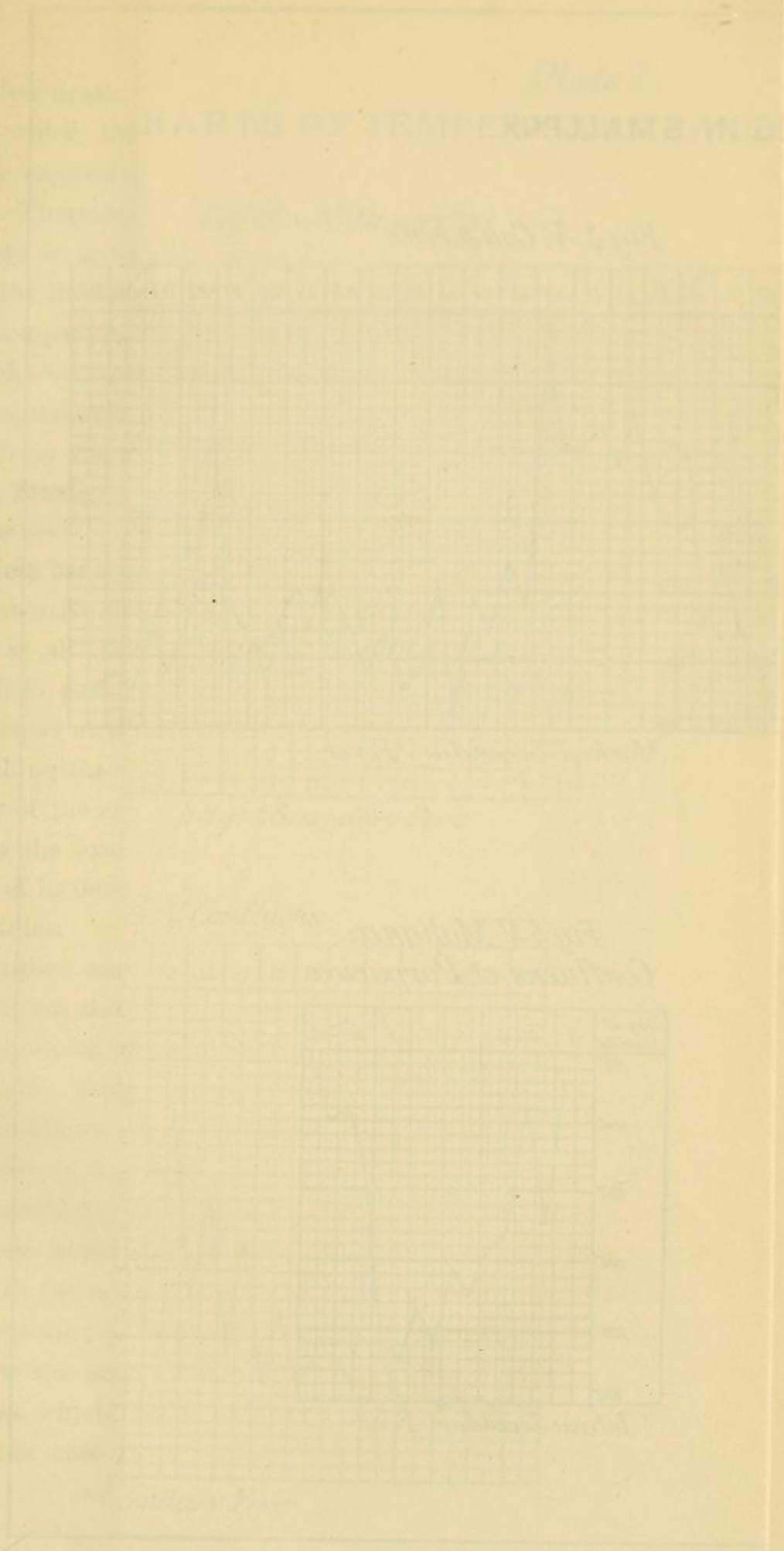
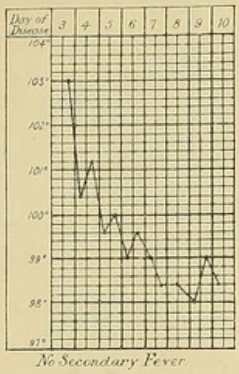


Plate I.

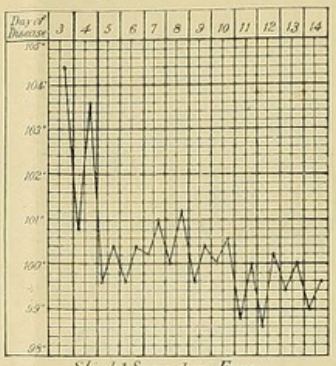
CHARTS OF TEMPERATURE RANGES IN SMALLPOX.

Fig. I – Variola Discreta.



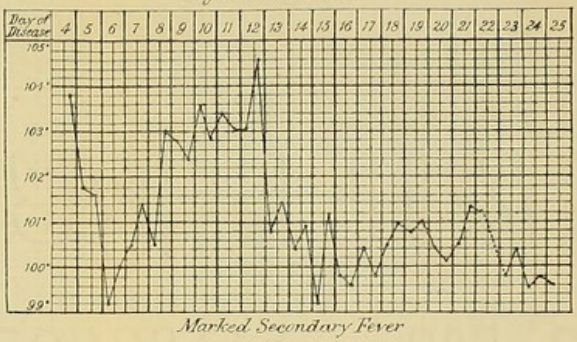
No Secondary Fever.

Fig. 2 – V. Discreta.



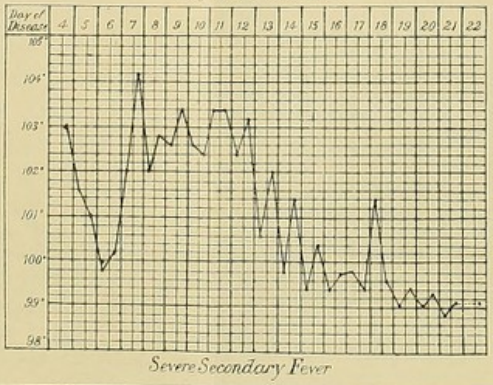
Slight Secondary Fever.

Fig. 3.-V. Cohærens.



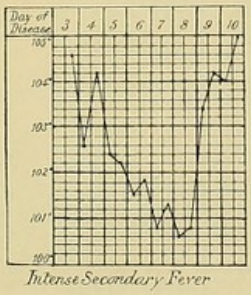
Marked Secondary Fever.

Fig. 4-V. Confluens.



Severe Secondary Fever.

Fig. 5-V. Maligna: Confluens et Purpurica.



Intense Secondary Fever.

or even after death. Th. Simon, of Hamburg, has published[a] two cases in which the temperature after death was 110·75° and 112·1° F. respectively. In Plate IV., illustrating his work on "Medical Thermometry," Wunderlich includes a chart of the temperature in a case of smallpox fatal in the suppurating stage, in which the thermometer marked 109·2° F. shortly before death.

The accompanying Temperature Charts are from cases observed by me in the wards of Cork-street Fever Hospital, Dublin, and the Meath Hospital and County Dublin Infirmary. [See Plate I.]

Apart from the classical and recognised forms of **Discrete** and **Confluent Smallpox**, we meet also with the following varieties of the disease:—

1. **Variola benigna** (**Varioloid**—Fr. *Variole modifiée*). This is a mild and abortive form of smallpox in which the pocks either fail to appear at all (**Variola sine exanthemate**, or **V. sine variolis**), or else fail to pass through the later stages of their development, stopping short at the papular stage (**Variola cornea** or **Hornpox**), or, if reaching the vesicular, drying up and shrivelling on the 5th or 6th day of the eruption (**Variola verrucosa**, or **Wartpox**). In other cases the exanthem passes rapidly and imperfectly through all phases of its development, producing more or less dwarfed forms of the pustules.

Some readers may be sceptical as to the existence of a *Variola sine variolis*, but the evidence is overwhelming that there is such a form of smallpox. After a well-marked initial stage, the disease aborts and the patient is well in three, four, or six days at the latest. Dr. Hilton Fagge refers to one instance of this kind, which was attended by a characteristic roseola. Another example of it is thus recorded by Marson. A lady walked with a person already affected with smallpox. Twelve days afterwards she was taken ill: she was for a few hours delirious, but her illness passed off without eruption; twelve days later still, her sister, who had not been out of the house for three months, was attacked with the same disease, which *ultimately assumed a confluent form*. Curschmann mentions the case of a woman who was seized with shivering,

[a] *Charité Annalen*, XIII. Band V.

fever, headache, and pain in the back, so that—as variola was epidemic at the time—she seemed without doubt to be passing through the initial stage. On the fourth day defervescence occurred, no rash could be detected, and by the tenth day she felt perfectly well. However, she gave birth to an infant, which was covered with an early eruption of smallpox. This afterwards suppurated and proved fatal.[a]

In the case of the so-called wartpox, also, the solid part of the pock remains for a long time, presenting the appearance of a wart—hence the name, "wart-pox" (Latin—*verruca, a wart*).

As Curschmann points out, **Varioloid** is nothing more than a form of true smallpox with a milder course and a shorter duration. Many individuals are attacked only by this form because of a naturally slight susceptibility to the contagium of the disease. Again, when the immunity gained by a previous attack of smallpox, or from a previous inoculation, or from vaccination, has become impaired through lapse of time, then exposure to the poison may induce an attack of *modified smallpox* or varioloid. There can be no question that this mild form of so dreadful a disease as smallpox has become relatively much more frequent since vaccination was introduced about a century ago.

As stated above, Hebra, Trousseau, and Curschmann, all consider that, as a rule, the development of the pocks is less the more extensive is the initial erythematous eruption. Viewed in this light the purely erythematous rashes of the invasion stage come to be of decided value in prognosis. I have already said that these rashes are to be carefully distinguished from the petechiæ which form in the earliest stage of some cases of the terrible purpuric or hæmorrhagic smallpox which I will now describe.

2. **Variola maligna—Variola purpurica** vel **hæmorrhagica.**—Observations of some three thousand cases of smallpox in two hospitals and two epidemics has led me to the conclusion that, apart from confluent smallpox in which the patient's life is endangered by the amount of suppuration and the intensity of the

[a] *The Principles and Practice of Medicine.* By the late Charles Hilton Fagge, M.D., F.R.C.P. London: J. & A. Churchill. 1886. Vol. I., page 225.

secondary or suppurative fever, **Malignant Smallpox** is to be recognised under two forms—(1) **purpuric**, and (2) **hæmorrhagic**. These forms differ merely in degree—in both the blood is profoundly altered, and devitalised to such an extent that it is incapable of throwing out or developing the characteristic eruption of smallpox. In the purpuric variety the dissolution of the blood leads to the formation of petechiæ, vibices or purple streaks or blotches, and ecchymoses—appearances connected with the skin which are sufficiently well known and do not require further definition.

In hæmorrhagic smallpox the dissolution of the blood is carried still further, so that a condition of acute hæmophilia is produced—the ill-fated patient bleeds from every pore and orifice of the body. There is chemosis—he may even weep tears of blood. There is epistaxis—he bleeds from the lips and gums. He spits or coughs up blood, he vomits blood. The motions from the bowels are tarry. Blood pours from the kidneys, and in the female from the genital organs. The tongue looks as if it was parboiled, and there is an unquenchable thirst. Under these circumstances, unless the hæmorrhage is staunched by turpentine and ergot, or ferric chloride, or pyrogallic, gallic, and tannic acids, or other means, death speedily ensues—too often indeed in spite of all that human skill and care can do.

One of the most extraordinary as well as the most painful features of this deadly malady is the clearness of mind which often remains with the unhappy patient almost up to the time when he draws his latest breath. There is in some instances no delirium, no stupor, no dulling of the intellect whatever—the victim literally looks death in the face in full possession of his senses. It has happened to me to be asked by a patient at 11 a.m. how long he had to live, and that patient lay dead four hours afterwards.

The cessation of the bleeding may bring back hæmorrhagic smallpox to the purpuric form, and in the case of the latter variety of malignant smallpox the restoration of the blood, evidenced by the brightening of the purpuric spots, may be followed by the tardy development of a copious eruption of either aborted pustules (papules)—in which case the patient happily recovers speedily, and

without suffering from a fever of maturation—or true and fully-formed pustules, when the patient has still to run the gauntlet of a severe attack of confluent smallpox, with its secondary fever, complications, and sequelæ. This is the idea of malignant smallpox and its varieties which I have formed from a lengthened experience.

Strong muscular men and pregnant or recently delivered women are said to be particularly liable to fatal hæmorrhagic smallpox, and this was certainly the case in the Dublin epidemics of 1871 and 1878. Curschmann, however, states that he has often seen this variety of the disease in delicate persons and in drunkards also.

According to Marson the blood in this form of smallpox is "poisoned from the very first and is rendered very fluid and watery."

In the fact that the true eruption does not develop, the two extremes of smallpox as regards gravity meet—the most benignant form, described by Sydenham, Peter Frank, and others, as **Febris variolosa sine exanthemate**—what de Haën called **Variolæ sine variolis**—and the so-called **Purpura variolosa**, the most malignant form, leading, as we have seen, to almost certain death. Under this latter title Curschmann includes those cases in which the process designated "hæmorrhagic diathesis" (which I prefer to call "acute hæmophilia") imprints its frightful stamp upon the disease even in its initial stage, or during the eruptive stage, or at the close of the latter.

We have just stated that hæmorrhagic or purpuric symptoms may occur not only in the stage of invasion (when they constitute ordinary Purpuric or Hæmorrhagic Smallpox—*Purpura variolosa*)—but also at almost any time in the stage of eruption. In order to distinguish these latter cases, as a matter of convenience, Curschmann employs the term **Variola hæmorrhagica pustulosa.** In this form of purpuric smallpox, the true exanthem may become the seat of hæmorrhage in the papular, the vesicular, or the pustular stage—most commonly, the pocks are about the size of a lentil when the bleeding into them begins, and the characteristic appearance is seen first on the lower extremities.

This variety of smallpox corresponds exactly to the "anomalous" or "irregular" smallpox of 1670, so described by Sydenham, who also speaks of the malady as "this dangerous **black smallpox**" (**Variolæ nigræ**), in which the eruptions "were more inflamed, and in the declension after their suppuration, frequently looked black."[a] Another name for this variety is **Variolæ cruentæ** (Bloody smallpox.)

From the foregoing description we may compile a Table of the varieties of Smallpox, which will prove useful for reference. It will be observed that *Variola discreta* finds a place in both portions of the Table:—

I. Variola vera—Natural Smallpox.

1. *Variola discreta*, vel *distincta.*
2. *Variola confluens*—
   - (1.) *V. confluens vera.*
   - (2.) *V. semi-confluens* (*Syn.*: "*Coherent Smallpox.*")
   - (3.) *V. corymbosa.*
3. *Variola maligna*—
   - (1.) *V. purpurica.*
   - (2.) *V. hæmorrhagica.*

   } *Variolæ nigræ.*
   - (3.) *V. hæmorrhagica pustulosa* (Curschmann). (*Syn.*: *Variolæ nigræ*, vel *cruentæ.*)

II. Variola modificata vel **mitigata—Varioloid (Variole modifiée)—Modified Smallpox.**

1. *Variola benigna*—
   - (1.) *V. sine variolis* (*Syn.*: "*Variolous Fever*").
   - (2.) *V. cornea*, vel *verrucosa* (*Syn.*: "*Hornpox*," or "*Wart-pox*").
   - (3.) *V. discreta*, vel *distincta.*

In drawing attention to this Table, I wish emphatically to enforce the view that all these various forms are merely modifications of one and the same disease—namely, **Smallpox.** We classify these varieties simply as a matter of convenience, and with Curschmann

[a] *The Entire Works of Dr. Thomas Sydenham.* By John Swan, M.D. London: F. Newbery. 1769. Pages 179, *et seq.*

we should hold that "none of these forms are sharply defined amidst the great group of variolous affections; but there is rather a gradual transition from one into the other, so that general outlines are to be associated with the most customary designations rather than sharply circumscribed features."[a]

Complications and Sequelæ.

1. The **Skin** may be the seat of—
   - (1.) Multiple Abscesses.
   - (2.) Phlegmonoid Erysipelas.
   - (3.) Boils—the "Furuncular Diathesis" of Trousseau.
   - (4.) Bedsores.
   - (5.) Gangrene—very rarely and only in the scrotum.
   - (6.) Brownish Discolorations (pigment spots) and *Acne pustulosa* on the face.
2. The **Eyes** may be affected in various ways—
   - (1.) Conjunctivitis, from pustulation or from irritation by retained secretions owing to œdema of the eyelids.
   - (2.) Abscesses and Sloughing of the Eyelids, caused, it may be, by the presence of even two or three pustules.
   - (3.) Keratitis, or Inflammation of the Cornea from pustulation or from atrophy leading to ulceration—"Atrophic Keratitis."
   - (4.) Iritis, and Suppuration of the Globe (Panophthalmitis), causing loss of sight.
   - (5.) Hæmorrhages into the Retina in hæmorrhagic smallpox, leading to Blindness.

Hebra's experience on this point must be unique, for he asserts positively that, of more than 5,000 cases of smallpox, pustulation occurred on the conjunctiva in only *one* per cent. He holds that eye-sight is lost in smallpox only as a result of metastatic abscesses occurring as a sequela.[b]

[a] Von Ziemssen's *Cyclopædia of the Practice of Medicine.* Art. "Smallpox." Vol. II., page 366.

[b] *Diseases of the Skin.* New Sydenham Society. 1866. Vol. I., page 254.

3. The **Hearing** often suffers. Thus we may have—
   (1.) Chronic Suppurative Otitis.
   (2.) Caries of the Bones of the Ear, with partial or complete deafness.
   (3.) Suppurative Thrombosis of Cerebral Sinuses.
4. The **Nose** is sometimes the seat of ulceration.
5. The **Tongue** may be enormously swollen (*glossitis*), leading to dysphagia.
6. **Œdema of the Glottis**, ushered in by aphonia, often causes death about the eighth day.
7. Secondary affections of the **Respiratory Organs** such as—
   (1.) Laryngitis (Perichondritis laryngea), diphtheritic or catarrhal—aphonia is an ominous symptom in this complication.
   (2.) Tracheitis.
   (3.) Bronchitis, nearly always present in greater or less degree.
   (4.) Pneumonia, usually lobular or catarrhal.
   (5.) Pleuritis, with purulent effusion from the outset.
8. Secondary affections of the **Digestive Organs** are uncommon in Smallpox. They are—
   (1.) Diarrhœa, which may cause death, particularly in young children.
   (2.) Hæmatemesis or Melæna.
9. Secondary affections of the **Circulatory Organs**—
   (1.) Pericarditis—very rare in smallpox.
   (2.) Hæmorrhages—(α.) cutaneous; (β.) from mucous membranes; (γ.) from serous Membranes.
10. Secondary affections of the **Renal Organs**—
   (1.) Albuminuria.
   (2.) Hæmaturia.
   (3.) Acute Nephritis.
11. Secondary affections of the **Nervous System**—
   (1.) Delirium, with or without Meningitis.
   (2.) Acute Mania (Trousseau).
   (3.) Various Paralyses.

12. **Phimosis**, from œdema of the prepuce.
13. **Pyæmia**, or **Septicæmia**.
14. **Joint Disease**, with painful swellings, effusions of serum or pus, inflammation of cartilages and of bones, may occur.

Pathology.—The morbid anatomy of the **skin** and **mucous membranes** must first be considered.

"Unlike the papule of measles and of most other exanthemata," writes Dr. Hilton Fagge, "the papule of smallpox depends upon a definite change in the superficial and middle cells of the rete mucosum, which from the very commencement of the morbid process are swollen and opaque, and in their midst exudation quickly takes place, so that by the end of two days the horny layer of the epidermis is raised to form a minute conical vesicle." Subsequently, the pustule forms as a result of cell proliferation, and a central depression—the so-called umbilicus—develops, at the bottom of which the opening of a hair follicle or sweat gland is, according to Curschmann, frequently found. This fact suggests an explanation of the formation of the umbilicus, so characteristic of the smallpox pustule. Either of these structures—the hair follicle or the sweat gland—may form a *retinaculum*, tying down the roof of the vesicle in the middle.

This explanation, however, is not always applicable, "since," says Dr. Hilton Fagge, "the pock does not necessarily bear a definite relation to any of the canals which traverse the cuticle. In all probability a similar function is then discharged by one of many other bands which cross the upper part of every vesicle in a direction more or less vertical, dividing it into a number of separate chambers" (or *loculi*). "This *loculated* character of the pock attracted notice long before its nature was understood; it affords the reason why only a small part of the fluid is evacuated when a needle is introduced into the roof at a single spot. . . . Auspitz and Basch showed several years ago that all the septa in question are in reality formed out of the original cells of the rete mucosum, small bundles of which adhere together, and become stretched out into filaments and bands, as the exuda-

tion accumulates around them. In this fluid leucocytes are present in small numbers from the very first; they go on increasing, and it gradually passes from transparent serum into opaque pus; the change is complete in about six or seven days from the first appearance of the papule—that is, in the earliest part of the eruption, by the ninth or tenth day of the disease."

The morbid processes in the skin are by no means confined solely to the epidermis. The papillary layer of the derma is also the seat of a variolous inflammation which may have very important and serious consequences. According to von Bärensprung, a hyperæmia extends down through the whole thickness of the skin. The exudation which fills the vesicle and afterwards the pustule is derived from these vascular (hyperæmic) tissues. Some of the papillæ are, according to Curschmann, flattened by pressure, becoming in consequence permanently atrophied. Others, however, are infiltrated by leucocytes to such an extent as to obliterate their nutrient blood-vessels and so to destroy their structure, converting them into a white or ash-gray substance—the *diphtheritic* pock of the German histologists. To prove this, Rindfleisch gives a drawing from an injected preparation, in which the affected area had failed to receive any of the colouring matter (Hilton Fagge).

The **liver**, **kidneys**, and **spleen** undergo important morbid changes. The spleen is much swollen, its pulp soft and of a light red colour, in those who die early in the disease. It subsequently resumes its normal appearance except in purpuric smallpox, when it may be found small, hard, of a dirty dark red colour, sometimes with white or yellowish follicles (Ponfick).[a] The liver and kidneys are the seat sometimes of cloudy swelling (granular degeneration), sometimes of acute fatty degeneration resembling that produced by poisoning with phosphorus. Fatty degeneration is the more advanced condition in which granular swelling may terminate. These organs may be found normal when death occurs either too soon to permit of degenerative changes, or so late that they have

[a] *Ueber die anat. Veränd. der innern. Organ. bei hämorrh. und pust. Variol. Berl. klin. Wochenschrift.* 1872. No. 42.

returned to their normal condition. The bile is generally pale and thin in confluent smallpox.

The **heart-muscle** may undergo degenerative changes like those observed in the liver and kidneys.

In Hæmorrhagic Smallpox, large or small **hæmorrhages** may be found in nearly all the viscera, *ecchymoses* in the serous membranes, and *extravasations of blood* in almost all the mucous membranes.

Diagnosis of Smallpox.—The pain in the back may simulate **lumbago**, which is, however, apyrexial.

The prodromal fever may be mistaken for **simple continued fever**, which has no rash.

The initial rash and vomiting may be mistaken for **scarlatina**, but the marked sore throat of this disease is wanting.

The initial macular rash, and the papular stage of the true rash, may be taken for **measles**, the marked coryzal symptoms of which are absent; in measles too the fever *increases* as the rash comes out, while in smallpox it *decreases*. At the onset of a papular eruption it is often difficult to decide whether the case is one of measles or smallpox. The following method, called the "**Grisolle sign**," is a certain means of diagnosis. If upon stretching an affected portion of the skin the papule becomes impalpable to the touch, the eruption is caused by measles; if, on the contrary, the papule is still felt when the skin is drawn out, the eruption is the result of smallpox.[a]

The pain in the back is wanting in measles, which usually attacks young children, whereas smallpox commonly occurs amongst adults.

The onset of smallpox and that of **typhus** often closely resemble each other, but the fever continues after the rash of typhus appears and the rash is macular rather than papular.

Strange as it may appear, **pustular syphilides** have been confounded with smallpox and *vice versâ;* but the clinical history should solve the question without much trouble.

The differential diagnosis of smallpox and **chickenpox (varicella)**

[a] *Sacramento Medical Times.* February, 1889. See Sajous' *Annual of the Univ. Med. Sciences.* 1889. Vol. I. H.-77.

had best be postponed until the latter disease has been described (see Chapter XII., page 125).

The vast and unsightly swelling of the face in confluent smallpox is very like **erysipelas**, but a careful physical examination would set the question of diagnosis at rest.

Dr. Robert Liveing[a] says that, of all diseases, perhaps **glanders** in an early stage is the one most likely to be mistaken for smallpox. The febrile symptoms are like those of smallpox, and the rash consists of hard infiltrations in the skin and mucous membrane, which quickly suppurate and form deep and inflamed ulcers. When these infiltrations are small and scattered, and before ulceration has begun, the difficulty of a diagnosis is by no means slight. Glanders is a rare disease and usually occurs in grooms and stablemen. The rash comes out in *successive* crops and ulcerates rapidly.

Lastly, in both recent epidemics of smallpox in Dublin, male patients were sent into Cork-street Fever Hospital as suffering from smallpox, but on examination it turned out that the suspicious rashes, the presence of which caused them to be sent to hospital, were due to the fact that they were taking *cubebs* or *copaiba*—the rashes were **medicinal rashes.**

In all cases, account should be taken of any prevailing epidemic; we should also inquire whether the patient has been exposed to any infection and whether he has already suffered from any of the eruptive or continued fevers, which might be mistaken for smallpox, or, conversely, smallpox for them.

Prognosis.—The mortality from smallpox depends on—(1) the patient's state as regards **previous protection** by an attack of natural smallpox, by inoculation, or by vaccination; (2) the **virulence** of the disease itself—the hæmorrhagic form being the most deadly, next the purpuric form, then the confluent form; (3) the **general hygienic condition**, or otherwise, of the patient's surroundings; (4) the presence, or otherwise, of **complications.**

Smallpox is most fatal to unvaccinated children under 5 years of age and to unvaccinated adults over 30 years. It is estimated

[a] *Diseases of the Skin.* Fifth Edition. London: Longmans, Green & Co. 1887. Page 51.

that 50 per cent. of the confluent cases, and 100 per cent. of the malignant cases, perish. The influence of vaccination for good is unquestionable—the mortality being 50 per cent. among the unvaccinated in general, 26 per cent. among the badly vaccinated, and only 2·3 per cent. among the efficiently vaccinated.

In Sheffield, in the outbreak of 1887–88, of 4,703 cases, 474 proved fatal, or 10 per cent.; of 4,151 vaccinated patients, 200 died, or 4·8 per cent.; of 552 unvaccinated patients, 274 died, or 49·6 per cent.[a]

Hæmorrhagic, or malignant, smallpox may kill in four, five, or six days from the earliest symptoms. In confluent smallpox, on the contrary, the patient seldom dies before the eleventh day, and, in general, according to Trousseau, the most fatal epochs are the twelfth, thirteenth, and fourteenth days.

In the case of a disease like smallpox, in which the blood is poisoned and destroyed from the outset, it goes without saying that defective sanitary surroundings—such as overcrowding, want of ventilation, bad house drainage, and so on—must enormously increase the patient's risk.

Again, the complications and sequelæ of the disease often slay the unfortunate individual who had escaped from the perils which beset the earlier part of his passage through smallpox. Œdema of the glottis, perichondritis laryngea (inflammation of the cartilages of the larynx), bronchitis (especially in winter), and diarrhœa may kill straight off; while pyæmia, septicæmia, and the furuncular diathesis, may exhaust the patient's strength after weeks or even months of suffering.

In his "Medical Report" of Cork-street Fever Hospital, Dublin, for the year ending March 31, 1880, Dr. Reuben J. Harvey included a Table showing how protracted was the stay in hospital of many of the non-vaccinated patients who ultimately recovered from smallpox. The high death-rate among the unvaccinated cases, fearful as it is, is not the only calamity the victims of a smallpox epidemic have to encounter. In the year named, 50 out

[a] *Report of an Epidemic of Smallpox at Sheffield*, 1887–88. By Dr. Barry, Inspector of the Local Government Board for England. London. 1889.

of 74 unvaccinated patients died in the hospital. Of the 24 who recovered, one-half were detained in hospital for a period of from one to three months; and boils, abscesses, and eye-affections occurred in these cases with a frequency and severity altogether out of proportion either to their numbers or even the apparent severity of the primary attack.

In the "Medical Report" of the hospital for the following year (ending March 31, 1881), I gave a similar Table, which illustrated the tediousness of the recovery of several non-vaccinated patients, who indeed escaped with their lives, but were fated to pass through weeks or months of suffering before they were fully convalescent. In one case 68 days, in another 77 days, and in a third 106 days were spent in hospital by these victims of non-vaccination smallpox. Even this was surpassed in the case of a man who was discharged after a sojourn of *nine months and nine days*. This unfortunate sufferer had as many as 42 large abscesses on his body as a sequel to the smallpox, but at length he happily recovered under antiseptic treatment.

CHAPTER X.

The Preventive Treatment of Smallpox.

Smallpox communicable; but one attack confers immunity from a second — Preventive Measures: 1. Isolation; 2. Inoculation; 3. Vaccination.— Inoculation illegal. — History of Inoculation. — Clavelisation.—History of Vaccination.—Value of Vaccination in controlling prevalence and mortality of Smallpox. Marson's views as to the use of Multiple Vaccinal Cicatrices.— Periodical Revaccination.—Circumstances which conduce to success of Vaccination.—Jenner's "Golden Rule."—Bovine and Humanised Lymph.— Performance of Vaccination.—Bryce's Test.—Vaccina: its local and constitutional symptoms.—Vaccino-syphilis.

This subject of the Treatment of Smallpox naturally falls under the two headings—**Preventive Treatment**, or **Prophylaxis**, and **Curative Treatment.**

Preventive Treatment—The principles of the Prophylaxis of Smallpox are based upon two facts in the natural history of the disease—namely (1). Smallpox is eminently communicable. (2). One attack usually protects an individual from a second attack—confers immunity upon him.

The preventive measures which call for remark are—(1). **Isolation of the Sick**; (2). **Inoculation**; (3). **Vaccination.**

1. Of these topics, the first has already been discussed in the chapter on the Preventive Treatment of the Acute Specific Fevers (see pages 30 *et seq.*). It will be sufficient, therefore, to repeat that **isolation** consists in the removal of sick to suitable epidemic hospitals, the providing of refuges for the inhabitants of infected tenement houses or other dwellings, efficient disinfection, and the establishment of convalescent homes.

2. As regards **inoculation,** the intention was to engraft a mild form of smallpox on a healthy individual, whose *receptivity* or susceptibility might be supposed to be slight or low in consequence of his existing good health. The disadvantages of this procedure was that it gave smallpox to many who would otherwise have, perhaps, escaped the disease altogether, while it was impossible to guarantee that the resulting attack of smallpox would be mild.

In 1840 an Act of Parliament was passed rendering **smallpox inoculation unlawful** in England (3 and 4 Vict., cap. 29).

Inoculation was **declared to be illegal** in Ireland by the fourth section of the "Vaccination Amendment (Ireland) Act" of 1868 (31 & 32 Vict., cap. 87).

The operation had been practised from time immemorial in China and Persia. The Chinese plan of giving smallpox to persons in health consisted in inserting into the nostrils tents of charpie covered with the dried crusts of the variolous eruption. They called the procedure "sowing the smallpox." From China and Persia inoculation or "engrafting" was introduced into Georgia, Circassia, Turkey, and Greece. In 1717, a very clever Englishwoman, by name Lady Mary Wortley Montague, wife of the British Ambassador at the Ottoman Court, wrote home glowing accounts of the marvellous results of inoculation as practised at Adrianople, amongst other persons, upon her own son, a boy of six years of age. Her influence led to the open adoption of the procedure in England in April, 1721, and two years later, in 1723, Dr. Bryan Robinson, sometime President of the King and Queen's College of Physicians, first performed the operation in Ireland. Many years elapsed before it obtained a footing in France, where at first it had been rigorously prohibited by law; but in 1756 the children of the Duke of Orleans were inoculated, and in 1758 the practice was introduced into most of the large towns.

A favourite analogous procedure, practised since the last century by Continental veterinary surgeons and farmers, was called **clavelisation**—a term derived from **clavelée**, the French name for smallpox of sheep (*Variolæ ovinæ*), a disease popularly known in England as "**tag-sore**," or "**sheep-rot**;" in Italy as "**vaccuolo.**"

The object of clavelisation was by repeated inoculations to **attenuate** the virus of sheep-pox. And exactly in the same way it was sought, by employing virus from a discrete case of smallpox which had been modified by antecedent inoculation, to communicate a very mild variola.

The operation consisted in raising the epidermis by means of a lancet charged with such an attenuated virus or **lymph.** A mere

prick was sufficient. The resulting symptoms were, first *local*, then *constitutional*. Thus, on the second day after inoculation, a small red *pimple* appeared at the site of puncture; by the fifth day this had become a conical *vesicle* (sometimes umbilicated); on the seventh day, the vesicle had developed into a *pustule*, surrounded by a slightly red inflammatory areola, which gradually increased up to the ninth or tenth day, when a ring of secondary small pustules, true satellites of the first, formed upon the inflammatory areola. This pustule of inoculation resembled a kind of large pock which is sometimes found in natural smallpox, and to which Van Swieten, of Vienna, gave the name of **Meisterpocken, master-pock**, or what Trousseau called **le maître bouton.**

On the ninth or tenth day after inoculation, the constitutional symptoms used to make their appearance. They were, in a word, all the primary or prodromal symptoms of smallpox. Finally, about the eleventh, twelfth, or thirteenth day, the specific eruption was seen—in general but slightly confluent, following the course of ordinary or sometimes that of modified smallpox.

I have given Trousseau's account of the phenomena attending inoculation at this length, because they are exactly analogous to the features presented after successful vaccination.

3. **Vaccination** (Germ. **Kuhpockenimpfung**).—About the middle of the eighteenth century the opinion gained ground in England that **inoculation with cowpox lymph protected from smallpox.** As Dr. E. Crookshank, in his elaborate "History and Pathology of Vaccination," says:—"In some parts of the country a belief existed among those who had the care of cattle that a disease of cows, which they called 'cowpox,' when communicated to the milkers, afforded them protection from smallpox." It is necessary to explain that various domestic animals are liable to a disease which is practically smallpox. Thus, a variety of the *grease* (Fr. *eaux aux jambes*) of horses—in which an eruption appears usually on the foot-joints—is properly called "*Variolæ equinæ.*" The "tag-sore" (Fr. *clavelée*) of sheep is "*Variolæ ovinæ;*" and the well-known "cowpox," in which the eruption is almost exclusively observed upon the udder and teats of the cow is technically called "*Variolæ vaccinæ*"

Fr. *picote*), or, shortly, "**Vaccina**" (the form "*Vaccinia*" being etymologically incorrect).

At a time when the majority of the people were deeply pitted with smallpox, the immunity enjoyed by the comely milkmaids of Gloucestershire and Devonshire from the unsightly scars left by the disease could not fail to attract attention. It was noticed that the dairymaids and farm-labourers were subject to an attack of sores on their hands, which seemed to arise from contact with pustules on the udders of milch cows. Those who suffered from this apparently local malady of sore-hands were observed to escape smallpox. At length, in 1774, Benjamin Jesty, a Gloucestershire farmer, who, however, had been born at Yetminster, in Dorsetshire, became so convinced of the protective efficacy of cowpox against smallpox that he inoculated his wife and two sons with cowpox—thus performing **vaccination** for the first time, and anticipating Edward Jenner by no fewer than twenty-two years.[a] On May 14, 1796, Jenner vaccinated a peasant lad, whom he failed to inoculate with smallpox two months afterwards. The crucial test was in this way applied to the efficacy of vaccination as a preventive measure.

It would be foreign to my present purpose to linger over the history of vaccination; nor is it necessary at this time of day to take much trouble to refute the views of the fanatical "anti-vaccination" party. Before the introduction of vaccination, the annual mortality from smallpox in England and Wales alone was at the rate of 3,000 deaths in every million of the population—this, according to the Census of 1891, would correspond to a loss of some 87,000 lives per annum—the population of England and Wales being 29,081,047 in April of this year. What are the actual facts? In 1890, smallpox caused only 15 deaths in England; and the average annual number of deaths from this disease in the ten years 1881–90 inclusive, was 1,227·8—that is, one-seventieth part only of the death-rate of pre-vaccination times.

But vaccination has not only diminished the number of cases of and deaths from smallpox—it is also found to influence the death-rate among those attacked to a very remarkable extent. In Sheffield,

[a] See *The Lancet*, September 13, 1862. Page 291.

in the outbreak of 1887–88 already referred to, of 4,151 vaccinated patients, 200 died, or 4·8 per cent.; of 552 unvaccinated patients, 274 died, or 49·6 per cent.

The following Tables are taken from a Report on the Epidemic of 1871–72, as observed in Cork-street Fever Hospital, Dublin, by Dr. T. W. Grimshaw, now Registrar-General for Ireland and Consulting Physician to the Hospital[a]:—

TABLE II.

Hospitals	Mortality per Cent.		
	Vaccinated	Unvaccinated	General
Cork-street - -	10·8	71·8	21·6
Hardwicke - -	11·2	78·57	20·0
Cork - - -	5·5	58·0	22·5
London Smallpox -	14·9	66·2	18·8
Hampstead (London) -	11·4	51·2	19·36
Homerton - -	5·9	37·7	16·3

TABLE III.

—	Discrete			Confluent			Malignant			Total		
	Total	Died	Per cent. Mortality	Total	Died	Per cent. Mortality	Total	Died	Per cent. Mortality	Total	Died	Per cent. Mortality
Vaccinated -	443	1	0·2	143	46	32·3	25	18	71·8	611	65	10·8
Unvaccinated	17	6	35·1	94	67	71 2	24	24	100·0	135	97	71·8
Total -	460	7	1·6	237	113	47·6	49	42	85·7	746	162	21·6
Per cent. vaccinated in each class	96·3			60·4			51·9			81·8		

Of the vaccinated cases in the discrete variety, the mortality

[a] See *Manual of Public Health for Ireland.* Dublin: Fannin & Co. 1875. Pages 172 & 173. Also *Special Report on the Smallpox Epidemic*, 1871-73, *as observed in Cork-street Fever Hospital, Dublin.* By T. W. Grimshaw, M.D. Being an Appendix to the Medical Report of that Hospital for the year ending March 31, 1873. Dublin: John Falconer. 1875.

was practically nothing (0·2 per cent.), only one patient having died. In that case the patient had inflammation of the lungs, probably quite independent of the smallpox. Of the unvaccinated patients, however, in this class, 35·1 per cent. died. In the confluent cases the mortality among the vaccinated patients was 32·3 per cent., while among the unvaccinated it was as high as 71·2 per cent. In the malignant or purpuric variety, the mortality among the vaccinated patients was 71·8 per cent., or about the same as among the unvaccinated confluent cases, while in this variety *not one* unvaccinated case recovered. It may be merely a coincidence of percentage mortality; but it is a remarkable fact that in the cases under consideration vaccination reduced the mortality of confluent cases to that of discrete unvaccinated cases, and that of malignant to that of confluent unvaccinated cases. The proportion of vaccinated to unvaccinated cases in each variety is considerably greater, except in the malignant form, where the proportions are nearly equal. The difference is most remarkable in the discrete variety, where the number of unvaccinated cases is very small—in other words, vaccination *prevented* a large number of these cases from being confluent.

In my "Medical Report of the Fever Hospital and House of Recovery, Cork-street, Dublin," for the year ending March 31, 1879, I give an account of the still more terrible epidemic of 1876–1879, proceeding on very much the same lines as Dr. Grimshaw's Report. The number of cases with which the Report deals is so large—namely, 1,804 from April 1, 1876, to March 31, 1879—that the results may be fairly considered conclusive as to the modifying influence of vaccination over (1) the type of the disease, and (2) its fatality. Of 1,003 patients who suffered from discrete smallpox, 943 were vaccinated, and only 60 were unvaccinated—the percentage of those vaccinated being 94·0. There were 10 deaths—the mortality being *one* per cent. Among these 10 deaths was that of an infant of 9 months, who was not *vaccinated*. Seven patients succumbed to the complications of discrete smallpox, dying *in* or *after*, not *of* this disease.

Of 604 patients suffering from confluent smallpox, 377 were

vaccinated and 227 were unvaccinated—that is, 62·4 only of every 100 patients were vaccinated. There were 243 deaths, the rate of mortality being 40·2 per cent. But of the vaccinated only 22 per cent. died, compared with 70·5 per cent. of the unvaccinated.

Of 197 malignant cases, 110 were vaccinated, or 55·8 per cent., and 77 were unvaccinated. The deaths numbered 151, the mortality being 76·6 per cent. Of the vaccinated 67·2 per cent. died, but of the unvaccinated 88·5 died.

Of the 1,804 patients in all, who were admitted, 1,430, or 79·2 per cent., were vaccinated, and 374 were not vaccinated. There were 404 deaths, or a mortality of 22·3 per cent. Among the vaccinated 166 deaths occurred, the mortality falling to 11·6 per cent. Among the unvaccinated 238 deaths occurred, the mortality rising to 63·6 per cent.

In very few instances, indeed, had the patients been revaccinated, probably not in more than 5 of the whole 1,804 cases.

Many years ago Mr. Marson, of the Smallpox Hospital, London, showed as a result of his examination of 5,000 cases, between 1836 and 1855, that the **number of vaccinal cicatrices** seem to influence the mortality from smallpox. The death-rate among patients having only one cicatrix was 7·73 per cent.; that among those who had two cicatrices was 4·7; that in the presence of three cicatrices was 1·95, and that with four or more cicatrices was only 0·55 per cent.

There can be no doubt that the protective efficacy of vaccination wears out gradually with the lapse of time. **Periodical revaccination** every seven or ten years is, therefore, necessary if smallpox is to be completely prevented, any case where revaccination at stated intervals has failed being always of a most peculiar nature.

That vaccination has in rare instances done harm cannot be disputed. Unsuitable subjects have been vaccinated, or the lymph has been taken from improper sources;[a] but such mishaps afford no valid argument against the practice of vaccination, however much they may lead us to call in question the skill and care of the operator in each untoward case.

[a] Grimshaw. *Loc. cit.*

The **circumstances which conduce to the success of the operation** are briefly the following :—

1. The subject to be vaccinated should be healthy. Special provision is made in the Vaccination Act for postponing the operation if the individual is not quite well. The presence of skin diseases and the propinquity of scarlet fever or erysipelas forbid the operation.

The periods of teething and of weaning should be avoided for the performance of vaccination.

2. The vaccinifer should also be healthy, vaccinated for the first time, and, above all, free from any syphilitic taint. The best age is from 5 months to over a year old; but vaccination may be, and—in epidemic times—should be, performed much earlier. In 1878, smallpox raging at the time, I vaccinated one of my own children at the age of three weeks successfully and without any untoward result.
3. The lymph should be taken between the fifth and eighth days. As a French medical poet, Casimir Delavigne by name, quoted by Trousseau, sings :—

> "Puisez le germe heureux dans sa fraîcheur première
> Quand le soleil cinq fois a fourni sa carrière."
>
> "Draw forth the auspicious germ in its first freshness,
> When the sun has five times completed his course."

The eighth day, inclusive, is generally selected in the United Kingdom as that of inspection.

4. The incisions, or scarifications, should not penetrate to the subcutaneous areolar tissue. They should be made with a scrupulously clean instrument.
5. Bleeding should be avoided as much as possible, lest the lymph should be washed away from the site of inoculation. Blood should not be drawn when piercing the vesicle to obtain the lymph.
6. Jenner's "**golden rule**" should always be observed, and that is—not to use lymph from a vesicle which has already showed the "areola," or inflammatory ring which forms around the vaccine vesicle at the beginning of the second week.

7. A thin, serous, readily-flowing lymph should not be used. Good lymph is perfectly limpid and viscid or sticky.

Whenever practicable, the **lymph**, which may be **bovine** or **humanised**,[a] should be carried directly from arm to arm. This, however, is often impossible, and so the lymph must be preserved for future use. The old-fashioned method was to allow the lymph to dry in a thin film on flat ivory or bone points. A much better plan is to preserve the lymph in hermetically sealed capillary glass tubes. Müller, of Berlin, usually mixes the lymph carefully with two parts of glycerine and two parts of distilled water (by means of a small brush) in a watch glass, and preserves this diluted vaccine lymph in air-tight capillary tubes. The activity of the lymph is not impaired by this procedure, and the precious virus is economised.

Vaccination is generally done upon the outside of the left arm—in girls, at the place where the scars will be as far as possible hidden by the future evening dress. At least two sets of minute punctures or scarifications are made with a vaccinating needle or lancet, moistened or "armed" with the lymph, at each side of the deltoid muscle as it passes down to its insertion on the outer aspect of the humerus. A little of the lymph should afterwards be rubbed into the punctures.

Various instruments for vaccination have been designed, one of the neatest and best being Rose's "**vaccinator.**" This ingenious little instrument consists of a lancet-blade at one end, and a series of prodding or "tattooing" needles, arranged like the five of dice, at the other. A rotatory movement projects the needles from the case, causing them to puncture the skin, and the lymph can then be rubbed in from the surface of the lancet.

According to Hebra, the most satisfactory instrument is a **lancet** having one surface convex, the other hollowed and presenting a groove to which a drop of the lymph adheres.

Another instrument, called by the Germans **Impffeder**, or **Vaccine-pen**, resembles a drawing pen, and consists of two parallel limbs with cutting extremities, between which the lymph is taken

[a] That is, derived from the heifer or from a human being already vaccinated.

up by capillary attraction. In employing this instrument the vaccine matter is introduced beneath the cuticle by a horizontal or vertical incision (Hebra).

Sir Thomas Watson[a] says that a very ingenious **test**, free from all ambiguity, by which we determine whether the cowpox is running its proper course or not, was devised by Mr. Bryce—hence called "**Bryce's test.**" His plan was this. He vaccinated the other arm, or some other part of the body four or five days after the first vaccination. If the system had been properly affected by the first operation, the inflammation of the second vesicle would proceed so much more rapidly than usual that it would be at its height and would decline and disappear as early as that of the first: only the vesicle and its areola would be smaller. One of the earliest disciples of Dr. Jenner, Mr. Hicks, used in a doubtful case to repeat vaccination in a few days after the first operation, and he remarked that the second vesicle made "immense strides to overtake the first."

Vaccina.—The **symptoms** of this affection are first **local** and then **constitutional**—the latter in most cases being very slightly marked. On the third day (inclusive) a patch of redness at the site of vaccination appears and rapidly develops into a papule or pimple, which in its turn, about the fifth day, becomes surmounted with a pearly vesicle, multi-locular, oval or circular in outline, with raised margin and depressed centre. This vesicle enlarges, while its contents also increase—remaining, however, clear as crystal—until the eighth day, when it attains its perfect growth. An inflammatory red zone, called the "**areola,**" now develops, beginning round the base of the vesicle and thence spreading out, perhaps to a distance of two or three inches. After the tenth day the areola fades, and the vesicle begins to shrink and dry up in the centre. The contained lymph becomes opaque and thickens. By the fourteenth or fifteenth day a hard, dry, brown scab forms, which finally separates and falls off about the twenty-first day. A circular, slightly depressed, foveate (or pitted) cicatrix remains, which—except in rare instances—is permanent through after-life.

[a] *Loc. cit.*, page 792.

During the earlier stages, the vaccine vesicles should be relieved from any pressure or friction, which might increase inflammatory action. In all cases, the vaccinated person should be seen on the eighth day—the day week—from the operation, when a certificate of successful vaccination may be given if the vesicle is well-formed and running a natural course.

The constitutional symptoms are—slight pyrexia from the fourth day, becoming more marked from the eighth to the tenth days; often derangement of the stomach and bowels during the stage of areola, with restlessness. The axillary glands may also swell, and rashes may show upon the skin—either a blush (*Vaccinal Roseola*), or a crop of papules (*Vaccinal Lichen*), or a vesicular rash (*Vaccinal Herpes*). In normal cases all these symptoms subside in a few days, or they may fail to appear at all.

Vaccino-Syphilis.—The inoculation of the syphilitic virus along with vaccine at the time of vaccination is, in the words of Dr. C. E. Shelly, the writer on the subject in Fowler's *Dictionary of Practical Medicine*, "the most lamentable accident by which carelessness or misfortune can prejudice the performance of vaccination." Since the incubation period of syphilis—namely, from three to five weeks—is much longer than that of vaccina, the latter affection usually runs its course before the local inflammation which constitutes a specific chancre begins. This is attended by the usual glandular enlargement (*axillary bubo*) and is followed in due course by the recognised secondary symptoms of constitutional syphilitic infection. If the precautions in the performance of vaccination described in the foregoing pages are strictly carried out, it is simply impossible that the disgraceful accident of simultaneously inoculating syphilis can occur.

CHAPTER XI.

The Curative Treatment of Smallpox.

No specific for Smallpox—two great Principles of Treatment: (1) to guide the essential disorder, (2) to combat secondary affections.—"Hot Regimen" treatment of olden times.—Thomas Sydenham's "Cooling Regimen."—Treatment of Discrete, Confluent, and Hæmorrhagic Smallpox.—Two dangers in Confluent Smallpox: general blood-poisoning, and exhaustion.—Antiseptic Treatment.—Prevention of "Pitting."—Dr. Stokes's views.—Three Indications for Treatment: (1) to exclude air, (2) to keep the surface in a permanently moist state. (3) to lessen the local irritation.—Hebra's Treatment by the Warm Bath.—Dr. Stokes's account of this method.—Hebra's apparatus for the Continual Bath.—Treatment of Affections of the Skin in Smallpox, and of the various Local Affections.—Turpentine and Ergot in Hæmorrhage.—Transfusion of Blood.

The superscription of this Chapter is in one sense somewhat misleading. No physician has ever yet *cured* a case of smallpox. **No specific for the disease** or for its many complications and sequelæ has as yet been discovered. Accordingly we must be content to set before us two great principles of treatment—first, **to guide the essential disorder** to a favourable termination; and, in the next place, **to combat secondary affections** as they arise.

There can be no doubt that the mortality from smallpox was enormously increased during the Middle Ages by mistaken and ignorant treatment. The old physicians did all they could to protect the patient from cold and to promote a copious eruption, adopting the vulgar maxim that "it was better out than in." In the fourteenth century flourished John of Gaddesden, author of a curious book entitled *Rosa Anglica*, court physician of the day, but "a very sad knave," as Sir Thomas Watson calls him. This representative physician of the Age not only put the unhappy smallpox patient on a "**hot regimen**," administering wine and cordials, piling on bed-clothes, and jealously excluding every breath of fresh air from the sick-room, but surrounded the half-suffocated victim with red curtains, red walls, and red furniture of all kinds—for in this colour there was, he pretended, a peculiar virtue.

To the celebrated Thomas Sydenham, who lived in the seventeenth century, belongs the credit of substituting for this barbarous and disastrous system of treatment the opposite or "**cooling regimen**" in smallpox, and this practice is pursued to the present day with the happiest results.

Premising that the reader has mastered the general principles of treatment of the specific fevers laid down in Chapters IV. and V., I will proceed to consider what further measures should be adopted in the case of Smallpox under its three chief forms—Discrete, Confluent, and Malignant or Hæmorrhagic.

Discrete.—The patient, having been placed in bed in a large, airy and well-ventilated, but warm room, should be carefully and skilfully nursed: his hair should be cut close, his hands and face should be washed daily, or twice a day, with warm carbolised water, from 1 to 2 per cent. in strength, or with a weak solution of corrosive sublimate (1–2000). Warm baths are very useful and refreshing. The water may, with advantage, be tinged with permanganate of potassium solution, bearing in mind, however, that this is decolourised at once and rendered inert by soap.

Confluent Smallpox.—The pain in the back may be relieved by dry cupping, or by the application of an India-rubber bag filled with hot water, or by a hypodermic injection of ergotin. An ice-bag applied to the head will often control headache. In all the severer forms of the disease, the patient should not be allowed to assume the upright position for fear of syncope, which is especially likely to come on after a free evacuation of the bowels.

In confluent smallpox the patient is beset by two dangers in particular—general blood-poisoning from the intense and widespread suppuration, and exhaustion from pain, sleeplessness, delirium and long-continued fever.

To check the development of a copious eruption **antiseptics** have been recommended and tried, but with no striking or even satisfactory results. One of the best and fullest accounts of the antiseptic treatment of smallpox will be found in a paper by my colleague, Dr. Arthur Wynne Foot, Physician to the Meath Hospital, in Vol. LIII. of *The Dublin Journal of Medical Science*, pages 242,

et seq. The way in which he endeavoured to carry out this treatment in the wards of the Meath Hospital during the epidemic of 1871–72 was by giving carbolic acid internally in the shape of the sulpho-carbolate of sodium (in doses of from 7 grains occasionally to 60 grains in water every third hour), or—when more suitable—the sulpho-carbolate of iron; by giving the sulphurous acid of the British Pharmacopœia, diluted with water—one drachm in a wineglassful or two of iced water—as the usual drink; by spraying the larynx with it, and washing the nares and upper surface of the palate with solutions of sulphurous or of carbolic acid; by keeping carbolised oil to the face; by washing the body with solutions of sulphurous acid or of vinegar and water; by throwing pure sulphurous acid about the bed and bed-clothes of the patient; and by burning sulphur in the sick-room.

My own experience is that in **quinine** and in **perchloride of iron** we possess the two most valuable antiseptics for internal use so far as smallpox is concerned. Quinine may be given in 5-grain doses thrice daily or oftener. This dose may be administered in water with just enough dilute hydrochloric acid to dissolve it, or with dilute hydrobromic acid and water, or mixed with fresh milk—when three grains of powdered camphor may with advantage be added to each dose. Either the tincture or the solution of ferric chloride may be given in 20 to 30 minim doses, with glycerine (℥ ss.) and water (℥ vii.), and perhaps a few minims of liquor strychninæ hydrochloratis.

If we possessed any certain means of checking the development of a confluent rash in smallpox, much distress and even danger to life in the first instance, and later on permanent disfigurement would be avoided. In a characteristic paper entitled "Some Notes on the Treatment of Smallpox," to which allusion has already been made,[a] Dr. William Stokes pointed out that the virulence of the pustulation and the tendency to pitting are directly as the cutaneous vascularity and heat of surface. In proof of this Stokes instanced the case of a strong and healthy young woman, who was admitted to hospital with symptoms of fever, including intense headache.

[a] See Chapter VIII., page 84.

For the relief of this symptom she was leeched freely on the temples. The attack proved to be one of confluent smallpox, but on the face not more than two or three small aborting pustules made their appearance. "Who can doubt," asks Stokes, "that in this instance the depletion of the face influenced the local progress of the disease." Again, he quotes a case commented on by Dr. Graves—that of a man who contracted smallpox while under treatment in one of the surgical wards for a chronic affection of the knee-joint. The affected joint was strapped with mercurial plaister, which exerted such pressure on the neighbouring cutaneous capillaries as absolutely to prevent the development of the eruption over the part. In a third case of severe confluent smallpox there was great tumefaction, accompanied with extraordinary heat of the face, and, in the hope of saving the eyes, poultices were applied over them. The patient recovered, but with deep and permanent pitting. There was, however, no pitting on the eyelids or in their immediate neighbourhood. From the date of this last case (1849) Stokes adopted as a routine practice the application of light poultices over the entire face, or of a mask of lint steeped in glycerine and water and covered with a corresponding mask of oiled silk. He found that, with but one exception, pitting was effectually prevented. In that case the patient was delirious, and could not be kept from tearing the poultices off his face.

As the outcome of his observations, Stokes came to the conclusion that, if from an early period we protect the surface from the air and keep it in a permanently moist condition, marking will seldom occur. There are then three important indications of treatment:—

1. **The exclusion of air.**
2. **The keeping of the parts in a permanently moist state,** so as to prevent the hardening of the scabs.
3. **The lessening of the local irritation.**

All the authorities are agreed that these indications should be as far as possible fulfilled. Dr. Charles West recommends strapping with mercurial plaister or sponging with a solution of corrosive sublimate, apparently attributing a specific action to the mercury in

each case. But for this there seems to be no warrant. Dr. Alfred Hudson used to smear the face with glycerine. Sir John Banks, K.C.B., many years ago suggested the application of an ointment composed of lapis calaminaris (native impure carbonate of zinc) and glycerine. Dr. Foot applied to the face carbolised oil—varying in strength from 1 in 4 to 1 in 8 parts; he also recommended the application of flexible collodion to the papules as early as possible, for its late application only does mischief, forcing the pus to burrow backwards into the cutis, so increasing the dermatitis and insuring pitting. Mr. Marson, of the London Smallpox Hospital, waited until the pustules had burst and the discharge had begun to dry. He then applied the best olive oil, or a mixture of glycerine and rose water in the proportion of 1 in 3. He also recommended "cold cream," or oxide of zinc ointment, or olive oil and lime water (*linimentum calcis*, or "Carron oil") or calamine mixed with olive oil. Curschmann speaks highly of the value of cold—even iced—compresses frequently renewed for the relief of pain, swelling and redness of the skin. The American practice to prevent pitting is exclusion from the room of the solar light and the application of a solution of boric acid (1 drachm to 1 pint of water) by means of compresses, frequently changed, or covered with oiled silk. In Germany a paste—composed of carbolic acid, 4 to 10 parts; olive oil, 40 parts; and prepared chalk, 60 parts—is spread on linen and applied to the parts where the eruption is apt to be worst. This application should be changed every twelve hours.[a]

Dr. Lewentauer[b] suggests the application to the face by means of a mask, and also to the other parts on which the eruption is marked, of an ointment consisting of salicylic acid, 3 parts; starch, 30 parts; and glycerine, 70 parts.

Bertrand[c] recommends the application, with a brush, as soon as

[a] *Outlines of Medical Treatment.* By Samuel Fenwick and W. Soltau Fenwick. Third Edition. London: J. & A. Churchill. 1891. Page 449.

[b] *Bulletin Gén. de Therapeutique.* No. 32. 1889.

[c] *Gazette des Hôpitaux.* Paris. July 15 and 17, 1890, and Sajous' *Annual of the Universal Medical Sciences.* 1891. Vol. I. H.—72.

the eruption appears either on the face or in the pharynx of a mixture of 4 grammes (1 drachm) of boric acid to 50 grammes (1½ ounces) of glycerine. The eyes, meanwhile, should be bathed with a tepid saturated solution of boric acid.

Talamon[a] applies ethereal solutions of various antiseptics by means of a spray apparatus. Salol does well only when the rash is slight and scanty; in all cases corrosive sublimate is to be preferred. He sprays the part for a minute 3 or 4 times a day—until desiccation takes place—with a solution consisting of corrosive sublimate and citric acid, of each one gramme (15·432 grains), alcohol (90 per cent.) 5 cubic centimetres (80 minims), and ether, sufficient to make 50 cubic centimetres (1½ ounces). The eyes should be guarded during the application.

Skoda prefers compresses moistened with solution of corrosive sublimate (gr. 2–4 to water ℥vj.). Hebra applies only cold water compresses.

My own plan is to apply over the face a light mask of lint thoroughly soaked in a mixture of iced water and glycerine (a teaspoonful in an ounce of water) and covered with oiled silk.

Closely akin to these various measures for lessening the irritation of the surface, keeping the eruption moist, and excluding air, is the treatment of smallpox by the **warm** or **tepid bath.**

In his classical "Essay on Fevers,"[b] Dr. John Huxham advises bathing not only the legs and feet but the arms and hands—"nay, and even the trunk of the body also," in certain cases of smallpox. His object, it is true, was to bring out the eruption well. He adds: "This is not altogether a new method: for Rhazes[c] advises the patient to be kept in a kind of *Balneum Vaporis*, to facilitate the eruption."

Hebra appears to have had his attention drawn to the treatment of smallpox by the warm bath through observing its efficacy in the management of burns. In his practice in the Vienna General

[a] *La Médecine Moderne.* Paris. April 17, 1890. And Sajous' *Annual of the Universal Medical Sciences*, 1891. Vol. 1. H.—71.

[b] London: J. Hinton. 1764. Pages 137–138.

[c] *Vide* Rhazes *de Variolis et Morbillis.* Cap. vi. Ex editione Mead.

Hospital patients suffering from extensive burns have been kept in the warm bath continuously for *one hundred days* with good effect. "It is clear," says Dr. Stokes, "that in the case of the continued warm bath we have the conditions just mentioned completely fulfilled, and that, too, as regards the entire person of the patient."

In the paper from which I have so largely quoted, Dr. Stokes details a case in illustration of the use of the warm-bath treatment of bad smallpox. So graphic and striking as a word-picture is this clinical record that I know I shall be excused for transcribing it at length:—"Not many years since," wrote Dr. Stokes (in 1872), "one of our students, a very large and robust man, was attacked with smallpox, which soon showed itself in its worst characters. The fever at first was very high, and the head-swelling and vascularity of the face intense. The *eruption was universal*, while the pustules on the face became confluent at an early period.

"Delirium set in, and the patient tore off the dressings from his face so often that we desisted from their further application. After the tenth day the condition of the patient was most appalling. The delirium continued, the circulation became every day weaker and more rapid, notwithstanding the free use of stimulants; the crusts were not only black, but on the legs where here and there there was less confluence, the blackness of the worst purpura appeared—a condition held by Hebra to be always fatal. The body was one universal ulcerous sore, and the agonies of the patient from the adhesion of the surface to the bed-clothes were not to be described. In addition to the usual fœtor of smallpox in the stage of decrustation, which was present in the highest degree, there was an odour of a still more intensely pungent and offensive character, which seemed to pass through the bystander like a sword. I never before or since experienced anything similar. Stimulants alone, freely and constantly employed, seemed to preserve the patient alive; the pulse was rapid, weak, and intermittent, and for several days we despaired of his life.

"At this juncture I happened to describe the case to my colleague, Mr. Smyly, who suggested the trial of the warm bath, with the view of relieving the terrible suffering. A bath in which he

could recline was speedily procured, and, pillows being adjusted in it, we lifted the sufferer in and placed him in the recumbent position. The effect was instantaneous and marvellous. The delirium ceased as if by magic; it was the delirium of pain, and the patient exclaimed, 'Thank God! thank God! I am in Heaven! I am in Heaven! why didn't you do this before?' The fœtor immediately and completely disappeared, so that on entering the ward no one could suppose that there was a case of smallpox in it. He was kept at least seven hours in the bath, during which time brandy was freely administered, and omitted only when he showed symptoms of its disagreeing with the brain. He was then removed to bed. The surface was clean, and in many places the sores looked healthy and white. The bath was repeated next day, after which he fell, for the first time, into a tranquil slumber. From this time his recovery was progressive, delayed only by the formation of abscesses and the great soreness of the feet.

"That this gentleman's life would have been sacrificed but for the timely use of the bath, few who have had any experience in prognosis can reasonably doubt. He was in the condition of a patient every portion of whose skin had been burned and ulcerated. The pustulation was almost universally confluent; the purulent matter highly putrescent; the hæmorrhagic state developed, and the nervous system suffering—in fact, he had every symptom of the worst putrid absorption.

"This case," adds Stokes, "and its singular result, in addition to the experience of Hebra, justifies the recommendation of the use of the bath."

In a discussion on the treatment of smallpox at the Medical Society of the College of Physicians of Ireland, on March 20, 1872, Dr. Hawtrey Benson, Physician to the City of Dublin Hospital, detailed a very similar case to the foregoing.[a] The patient was kept in a slipper-bath, at a temperature of 98° F. for five hours and a half. He was then put to bed perfectly free from delirium, and, with the help of 15 grains of chloral (of which 60 grains had previously had no effect), he slept uninterruptedly for

[a] *Dublin Journal of Med. Science.* Vol. LIII., page 325. 1872.

eight hours. The case progressed from that time forward without the slightest check.

Professor Hebra's **apparatus for the continual bath** was exhibited in the London International Exhibition of 1862. It was also fully described, with an illustrative plate, in the *Wiener allgemeine med. Zeitung*, No. 43, 1861, as well as in the inventor's work on "Diseases of the Skin," translated for the New Sydenham Society in 1866 by the late Dr. Hilton Fagge (Vol. I., page 320). The apparatus consists of a bath, six feet long by three feet broad, made of wood and lined with copper or zinc. Exactly fitting its interior is an iron frame to which are fastened transverse bands of webbing, as in an ordinary bed. About two feet from one end of this frame is attached a head-support, which moves on a hinge and can be fixed at any angle by a simple piece of rack-work. The frame is covered with a blanket and is also provided with a horse-hair pillow; it does not rest on fixed supports, but is suspended in the bath by cords attached to it at each end. These cords pass over two small rollers, placed one at the head, one at the foot of the apparatus and provided with handles, so that the whole bed can easily be raised or lowered within the bath. At the head of the bath, but at a higher level, is a vessel made of copper, which can be heated so that the water may be supplied at any required temperature. The supply pipe enters the bottom of the bath, the escape-pipe opening into it at the water-level. When the apparatus is in use water is kept flowing constantly through it, so that all impurities are rapidly washed away. To enable the face to be kept continually wet, or to be specially irrigated, additional small tubes, each provided with a rose, are connected with the copper vessel or reservoir.

Before the patient is placed in the bath, it is filled with warm water, at a temperature of 90° to 100° Fahr., according to his inclination. A wooden cover, upon which a blanket is spread, is put over the lower part of the apparatus while the patient is in the bath. If it is desired that the head also should be covered, this is easily managed by roofing in the head of the bath by means of hoops upon which blankets are placed.

Four of these baths were put up in the General Hospital of Vienna under Prof. Hebra's supervision.

The treatment of such **affections of the skin** in smallpox as bed-sores, abscesses, boils, erysipelas, and gangrene, consists largely in scrupulous cleanliness and efficient nursing. The body linen should be frequently changed. The patient should lie on a water-bed, or a woven-wire mattress, since the introduction of which into our hospital wards bed-sores have become much less common than before. The intense pain which attends the formation of pustules upon the soles of the feet and the palms of the hands is due to the thickness of the epidermis, which is with difficulty raised by the exudation and thus causes a counter-pressure on the cutis. Our object then should be to keep these parts moist and therefore soft, and this is effected by wrapping the feet and hands in wet cloths covered with oiled silk or gutta percha tissue as recommended by Hebra.[a] If this is done, no disagreeable sensations are felt. Writing in 1764, Huxham said: "I would recommend also bathing the feet and legs in warm water, or milk and water, for a few minutes, two or three times a day before and at the eruption, and would likewise have cataplasms of *milk and bread*, *boiled turnips*, or the like, applied to the feet."[b] He did this "to make a very powerful revulsion from the head and breast."

The **eyelids** should be poulticed to reduce **œdema**, or kept covered with cold compresses. For **atrophic keratitis** cod-liver oil, iron, wine, and good food are indicated.

Affections of the **mouth, tongue, and pharynx** are best treated with ice, antiseptic sprays of sulphurous acid, chlorinated soda solution, corrosive sublimate solution, or Condy's fluid well diluted, antiseptic gargles of quinine, chlorate of potassium, boric or lactic acid, carbolic acid, and so on; linctuses of glycerine of tannin or of carbolic acid, boric acid, &c.

In cases of **laryngitis** the internal use of ice is invaluable. Hot poultices should be slung round the neck and kept smeared with glycerine of carbolic acid or a $2\frac{1}{2}$ per cent. carbolised oil.

[a] *Diseases of the Skin.* New Syd. Soc. 1866. Vol. I., page 266.

[b] *Essay on Fevers.* Page 136.

In the early stage leeches may be applied to the angle of the jaw. In like cases in scarlatina Graves recommended the application of relays of sponges wrung out of hot water to the front of the neck for 15 or 20 minutes at a time. Above all, the steam kettle should be kept going and the patient should be placed in a croup-tent and well supported by food and stimulants. In **acute œdema of the glottis**, Curschmann advises that an emetic should be given, if the patient is strong enough, or local scarifications or tracheotomy may be employed.

The same measures as those advised for laryngitis may be adopted in **bronchitis** or other affections of the respiratory tract, in addition to dry-cupping (if the rash is not thick-set) and poulticing.

Diarrhœa is often controlled by a starchy diet and brandy or port wine, and by poulticing the abdomen. If not, solution of pernitrate of iron may be prescribed, or pills of acetate of lead and opium, or—in the case of children—aromatic chalk powders.

In cases of **sleeplesssness and delirium**, the hair should be cut close or the head shaved, ice may be applied as recommended by Stokes, unless there is much depression, and stimulants often agree. The drugs mentioned in Chapter V. may be prescribed according to circumstances. Attendants upon smallpox patients should always be on their guard against homicidal or suicidal attempts.

Curschmann recommends that chloral hydrate should be administered by the rectum, in an enema containing from one and a half to two drachms, with eight ounces each of water and of mucilage. He says that variolous affections of the pharynx and larynx may be dangerously intensified by this drug.

Lastly, while only too often it happens that all our efforts to combat **hæmorrhagic smallpox** are in vain, we yet may save life by the administration of the solution or tincture of ferric chloride in full doses—30 minims every third hour, or of gallic or tannic acid in 5 to 10 grain doses, or of pyrogallic acid, in one grain doses, or of ergot (ounce-doses of the infusion or 3 grains of ergotin dissolved in glycerine and water, the latter dose being given hypodermically if need be), or—best of all—of **turpentine and ergot.**

The formula for the last combination which we used at Cork-street Hospital was as follows:—

℞. Extract. Ergotæ Liquidi, ʒiii;
Olei Terebinthinæ, ʒiii;
Spt. Ætheris Nitrosi, ʒii;
Spt. Rectificati, ʒi;
Ovi Vitellum;
Aquæ Menth. Piperitæ, ad ℥viii.

Signa: One eighth part every 3rd, 4th, or 6th hour, as required.

At Cork-street Hospital, also, in **menorrhagia** and **metrorrhagia**, cold to the vulva was of use, also slapping the buttocks with cloths dipped in ice-cold water. In several cases, hot water injected into the vagina, as recommended by Dr. Emmett of New York—the temperature of the water being from 98° to 110° F.—and cordially approved by Dr. Lombe Atthill, seemed to do good.

In these awful cases, stimulants are imperatively called for—brandy, whisky, or wine according to circumstances, and especially "egg-flip" mixture and "turpentine punch." Curschmann recommends a further trial of **transfusion of blood**, which, he admits, has so far disappointed expectation.

CHAPTER XII.

Varicella, or Chickenpox.

Nomenclature.—Derivation of the term "Chickenpox."—Definition.—Ætiology (historical sketch).—Clinical History: Incubation, Invasion, Eruption, Desiccation.—No secondary Fever.—Not a fatal Disease.—Complications and Sequelæ: Varicella gangrænosa (Jonathan Hutchinson).—Dermatitis gangrænosa—"Varicella-prurigo" (J. Hutchinson).—Diagnosis from Lichen, Herpes, Pemphigus, and Varioloid.—Prognosis and Treatment.

Nomenclature.—Chickenpox.—*Synon.*:—Varicella, Crystalli. *Germ.* die Wasserpocken, Windblattern, or die fliegende Blatter; *Fr.* Petite vérole volante, *La Vérolette* (Hatté), or Varicelle; *Ital.* Ravaglione; *Eng.* Waterpock or Glasspock. The term Chickenpox is supposed by Dr. Hilton Fagge to be a corruption of *Chick-pease*, the French *Pois chiches*, dwarf-peas, or briefly *Chiche* from the Latin *Cicer*, the chick-pea.

Definition.—An acute specific and highly infectious febrile disease, especially of infancy and early childhood, not dangerous to life, characterised by the appearance on the skin of *successive crops* of clear, colourless, watery vesicles. It is a separate and distinct disease from smallpox—a disease *sui generis*. The accompanying fever is moderate as a rule and of a remittent type, increasing and abating with the coming and going of the vesicular rash.

Ætiology.—Varicella is essentially a **disease of childhood**, and usually occurs before the first dentition is completed. Even sucklings may be attacked, but among children over ten years of age the disease is infrequent. Thomas,[a] of Leipzig, never saw an adult suffering from varicella. Baader,[b] of Bâle, carefully noted 584 cases—382 occurred in children aged from 1 to 5 years; 191, from 6 to 10; 7, from 11 to 15; 2, from 16 to 20; and 2 (?), from 20 to 40. Two years ago, a friend of mine, aged 25 years, himself a member of the medical profession, had in Dublin a well-marked attack of varicella.

[a] Von Ziemssen's *Cyclopædia of the Practice of Medicine*. Vol. II. *Acute Infectious Diseases*. Art. "Varicella."

[b] *Jahrbuch für Kinderheilkunde*. XVII., page 104.

Varicella shows itself **sporadically** (in isolated cases) or in **moderate** and **often-repeated local epidemics** *quite independently of smallpox.* In Germany, Thomas says it appears yearly or even twice a year with great regularity shortly after the opening of the "Kinder-gärten," or Infant-schools. Its appearance is not determined by season, but it often follows in the wake of other specific fevers—scarlatina especially.

It is supposed that the virus is generally inhaled. Its tenacity of life does not seem to be great: hence, perhaps, the limited spread of its epidemics. The contents of well-marked varicella vesicles have been **inoculated with negative results** by Heim, Vetter, Czakert, Fleischmann, and Thomas, in Germany; Boyce, in Edinburgh; and J. Lewis Smith in America. Hesse, of Leipzig, occasionally succeeded in inoculating varicella, and Steiner was also successful in 8 out of 10 cases reported in the *Wiener med. Wochenschrift*, No. 16, for 1875. In marked contrast with its practical non-inoculability stands the facility of its dissemination among little children by contagion. As a rule chickenpox does not recur, but Trousseau says that second attacks are not uncommon, and Gerhardt is said to have seen it recur for the third time.

The nature of the specific virus of varicella is as yet unknown. Dr. A. Tschamer[a] claims to have isolated a hitherto unknown micrococcus, which he considers stands in a causal relation to the disease. But no definite conclusions have so far been arrived at.

The earliest authentic descriptions of varicella date from the middle of the sixteenth century. About the year 1550 two medical writers, Vidus Vidius and Ingrassias of Naples described the disease under the name "**Crystalli,**" in reference, no doubt, to the clear, crystal-like contents of the chickenpox vesicles. The Italian synonym was **Ravaglione.** The malady was first fully described and differentiated in England by Heberden in 1766 under the name of "**Variolæ pusillæ.**" His paper was published in the first volume of the "Medical Transactions of the Royal College of Physicians," 1767. In it he pointed out the chief reason which made the recognition of chickenpox a matter of importance—

[a] *Archiv. f. Kinderheilkunde.* Band II. Heft 3.

namely, that those who had it might otherwise be deceived into a false security "which might prevent them either from keeping out of the way of the smallpox or from being inoculated" (Hilton Fagge). Two years previously, in 1764, Vogel is said to have introduced the name of "**Varicella**," which, like "Variola," is a diminutive of the Latin *varus*, a pimple.

Clinical History.

I. **Stage of Incubation.**—The stage of latency is believed to be on an average about as long as that of smallpox—namely, **12 days.** Makuna[a] states that it varies from 8 to 17 days. Bristowe says[b] that in some cases it lasts exactly a week, but perhaps more commonly a fortnight. According to Thomas it varies from 13 to 17 days, while Trousseau extends its duration to from 15 to 27 days. On the other hand, Gregory limited it to from 4 to 7 days. The discrepancy, in Dr. Hilton Fagge's opinion, arises from the fact that the length of the incubation has in general been calculated upon the very precarious basis of the interval between the dates at which different children of the same family have been successively attacked. Towards the close of this stage there is, according to Thomas, in some cases a slight rise of temperature.

II. **Stage of Invasion.**—This stage, also called that of the "**Prodromal Fever,**" is badly marked in varicella. It often happens that the child feels perfectly well until the rash appears. In other cases, however, *malaise*, loss of appetite, a feeling of sickness, headache, chilliness, and muscular pains precede the eruption by a few hours, or one to even three days. The "Prodromal Fever" is usually very slight, but occasionally a sudden rise of temperature to 101°, or—in severe cases—even to 104° takes place just before the rash appears. Once Thomas observed a transitory general erythema with a temperature above 106° F., but such an occurrence is quite exceptional.

III. **Stage of Eruption.**—This may be ushered in by a roseolar scarlatiniform rash, but the true eruption consists of papules or measles, like the rose spots of typhoid (Trousseau), and deleble on

[a] *Lancet*, Vol. II., page 350. 1875.

[b] *Theory and Practice of Medicine.* Seventh Edition. 1890. P. 186.

pressure (Gee), which rapidly change into vesicles. These do not, under ordinary circumstances, become pustular and are unaccompanied by an inflammatory areola. The vesicles appear first on the trunk, especially the chest, then on the face and scalp, finally on the limbs. They increase in size up to the third or fourth day, when they are about as large as split peas. They become acuminated or conoidal, and finally burst, shrivel, and dry up. They are not markedly umbilicated and are said to be not divided into compartments (*loculi*) like the pustules of smallpox. According to their shape, they were described by Willan and Bateman, in their work on Skin Diseases, published in 1806, as *lenticular*, *conoidal*, or *globate*. They contain a clear, watery, but afterwards straw-coloured lymph. Dr. Hilton Fagge describes the vesicles as sometimes having a red base, but sometimes being seated upon a perfectly colourless surface, so that the patient looks exactly as if he had been sprinkled with drops of clear water.

When air exists under the roof of the vesicle, it has always entered through some aperture from without (**Varicella ventosa, emphysematosa; Windpox**).

The visible mucous membranes, as well as the skin, are the seat of an eruption. Thus, vesicles are not uncommonly seen on the buccal mucous membrane, the hard and soft palates, the throat, lips and tongue, the conjunctivæ and gums (Henoch), and the genital mucous membrane in girls (Thomas).

The febrile movement is not acute—indeed in some very mild cases there is a complete absence of fever. Usually, however, the fever is remittent, increasing at night (Trousseau), and in proportion to the amount of the rash, which may continue to come out in successive crops for even 10 or 12 days. In Willan and Bateman's *Delineations of Cutaneous Diseases* there are two very good Plates illustrative of these appearances. Defervescence, when it occurs, is usually rapid (Hilton Fagge).

IV. **Stage of Desiccation.**—This stage, or process, varies in duration owing to the development of the eruption in successive crops of vesicles. In the case of individual vesicles, desiccation takes place rapidly, the vesicular contents being partly absorbed

and partly extravasated through bursting of the vesicle. A pit, or scar, may be left when the vesicle has become a pustule. A good example of this is represented in Plate XXXII. of the New Sydenham Society's *Atlas of Skin Diseases*.

Unless in very rare instances, there is **no secondary fever**, and the disease—if uncomplicated—**seldom or never destroys life.**

According to Trousseau and Canstatt, relapses in varicella are frequent, but Thomas never saw a *true relapse*—that is, the renewed appearance of the disease in its totality, and he rightly looks upon cases of so-called relapse as being really examples of recurrent crops of the rash.

Complications and Sequelæ.—These may almost be said not to exist, but in a communication to the Royal Medical and Chirurgical Society on Tuesday, October 25, 1881, Mr. Jonathan Hutchinson, F.R.S., drew attention to a formidable and very dangerous, though happily rare, variety of the disease, to which he gave the name of "**Varicella gangrænosa.**" This malady, according to him, was well-known in Ireland in past times under the names of the "**White Blisters,**" the "**Eating Hive,**" and the "**Burnt Holes.**" In proof of this, reference was made to a paper written by Dr. Whitley Stokes, of Dublin, in 1807, in which he proposed for the disease the name of "**Pemphigus gangrænosus.**"[a]

"This dangerous form of the disease," writes Dr. Eustace Smith,[b] "is not confined to weakly, ill-nourished children, but is most common in them. It is, no doubt, connected with the curious tendency to spontaneous gangrene sometimes met with in children." According to several observers, this condition often attends **acute miliary tuberculosis.**

"In gangrenous varicella," proceeds Dr. Eustace Smith, "the vesicles, instead of drying up in the ordinary way, become black and get larger, so that a number of rounded black scabs, with a diameter of half an inch to an inch, are scattered over the surface of the body. If a scab be removed, it is seen to cover a deep

[a] *The Dublin Medical and Physical Essays.* Dublin: Gilbert & Hodges. Vol. I. 1808. Pp. 146, *et seq.*

[b] *Disease in Children.* New York. 1884. Page 49.

ulcer. Around it the skin is of a dusky red colour. All the vesicles do not take on the gangrenous action, so that we find many varicellous scabs of ordinary appearance mixed up with the blackened crusts. The gangrenous process often penetrates deeply through the skin to the muscles, but under some of the scabs the ulceration is more shallow. These cases are very fatal. Mr. Warrington Howard has reported the case of a weakly baby twelve months old, who weighed only six pounds and a half. The child was attacked with gangrenous varicella and died in a few days of pyæmia with secondary abscesses in the lungs."

Dr. Radcliffe Crocker points out (*Lancet*, May 30, 1885) that this gangrenous eruption may occur in parts not the seat of the varicellous rash, and it is—in fact—well known and described as **Dermatitis gangrænosa** by various writers. I have myself seen a most remarkable case of the affection in a young lady, who for years suffered from urgent gastric symptoms, apparently of neurotic origin. Hutchinson, in his paper, states that loss of sight may occur from purulent irido-choroiditis in the course of gangrenous varicella.

According to Hilton Fagge, it is a question whether the early stage of **vesicular strophulus** is to be regarded as distinct from chickenpox. If not, the favourable prognosis which is allowed by all writers must to some extent be modified, for whereas they speak of varicella as always ending in recovery, one of Hutchinson's cases of "**Varicella-prurigo**" terminated fatally. This is a remarkable skin affection which Mr. Hutchinson thinks arises out of varicella, but which Hilton Fagge believes to be an exaggerated form of Strophulus, or Red Gum.

Diagnosis.—Chickenpox may be confounded with **lichen, herpes, pemphigus,** and **varioloid.** The vesicles may be somewhat clustered together, as in herpes. Trousseau speaks of an epidemic in the Necker Hospital, Paris, in which, during from 15 to 40 days, blebs or *bullæ* like those of pemphigus kept appearing on different parts of the patients' bodies, leaving ulcerations which lasted for six weeks or two months. From the three first-named skin affections chickenpox is sufficiently distinguished by the age of the patients, their previous history, and the course of the disease. It is of the

first importance to correctly diagnosticate varicella from smallpox. In cases of doubt, it will be better for the physician to act as if the disease were really varioloid, in order to protect the community.

At this time of day, it seems hardly necessary to insist that varicella is a disease of its own kind (*sui generis*), absolutely distinct from smallpox in all respects. But the glamour which attaches to the name of the great dermatologist of Vienna—Hebra, and to that of his son-in-law, Kaposi—both of whom refuse to recognise the separate identity of chickenpox, render imperative a statement of the grounds upon which a **differential diagnosis** is based. Of these the principal are here given after Trousseau's account, contained in his masterly *Clinique Médicale de l'Hôtel Dieu:*—

1. Chickenpox has often prevailed epidemically without smallpox. Möhl, for example, states that from 1809 to 1823 chickenpox was annually observed at Copenhagen, from which smallpox was absent. On the other hand, varioloid has never been prevalent without coincident smallpox.
2. As to age, very young children are attacked by chickenpox, whereas smallpox in a population protected by vaccination usually shows itself in adults.
3. Chickenpox had been described and known long before the introduction of vaccination, previously to which date varioloid was rarely met with.
4. Vaccinated children readily take chickenpox, not so smallpox, even in the form of varioloid.
5. Children who have had chickenpox may contract smallpox even soon afterwards. In the *Lancet* for 1877 a case is recorded of an unvaccinated child, who was admitted into St. Thomas's Hospital for chickenpox, but who was placed on the floor containing the smallpox wards because the diagnosis was at first uncertain. Two days afterwards vaccination was performed, which succeeded. Eight days later still the child fell ill with modified smallpox (Hilton Fagge).
6. The two diseases may co-exist. In 1845, Dr. Delpech published a case of this kind.

7. The virus of chickenpox never gives rise to smallpox, and the converse of this proposition is believed to be equally true.
8. Chickenpox is generally held to be non-inoculable, whereas smallpox is notoriously inoculable.
9. Second attacks of chickenpox are not uncommon, while smallpox rarely occurs twice in the same person.
10. The eruption of chickenpox may set in after 24 hours, that of varioloid is postponed to the fourth or fifth day.
11. The febrile movement in chickenpox continues after the spots appear, that in varioloid subsides.
12. In chickenpox the spots come out in successive crops and the fever is slight and remittent.
13. The characters of the spots are essentially different in the two diseases.
14. Chickenpox is not a deadly disease, whereas even the mildest form of smallpox may prove fatal.

The interested reader will find an excellent statement by Dr. Samuel Jones Gee of the arguments in favour of the non-identity of varicella and variola in Vol. I. of Reynolds's *System of Medicine.*

Prognosis and Treatment.—The simplest measures suffice in the management of a disease which is generally so harmless that Thomas thinks that any prophylactic isolation of the patient is quite needless. In passing, I may state that I, for one, do not concur in this view. Dr. Douglas Powell[a] considers that eczematous eruptions after varicella, with concurrent swollen glands, may lead to the development of phthisis. All that is necessary in a case of ordinary chickenpox, however, is to keep the child indoors, prescribe a milk and broth diet, avoiding strong animal foods, and regulate the bowels by gentle aperients. Sir William Aitken recommends stewed prunes or baked apples to fulfil this indication. We should further give mild diaphoretics, if the skin is dry and itchy and the fever acute. Lastly, it is most desirable to protect the vesicles as far as possible from injury by rubbing or scratching, lest a secondary and more severe dermatitis, resulting in scarring, should be set up.

[a] *On Diseases of the Lungs and Pleuræ, including Consumption.* London: H. K. Lewis. Third Edition. 1886. Page 413.

CHAPTER XIII.

Morbilli, or Measles.

Nomenclature.—Definition.—Ætiology.—Bacteriology.—Chief Stages of Infectiveness.—Epidemics in Faroe Islands and in Figi.—Seasonal Prevalence: Measles a disease of Spring and Autumn.—Clinical History: Incubation, Invasion, Eruption, Desquamation.—Furfuraceous Desquamation.—Convalescence complete on the Eighteenth Day.

Nomenclature.—Measles (originally Mesles or Maseles),[a] corresponding to the German *Maal*, marks or moles—and *Masern*, spots (the Sanscrit *Masura*, spots). *Ital.* Morbilli ("The little plagues," being diminutive of *Il Morbo*, the plague); *Lat.* Rubeola (so called by De Sauvages, 1760); *Fr.* Rougeole; *Dutch*, Mazelen; *Danish* and *Norwegian*, Mæslinger; *Swed.* Mässling; *Span.* Sarampion; *Ital.* Rosolia, or Roselia; *Arabic*, Hhasbah (Rhazes and Avicenna).

Definition.—A highly infectious, acute, febrile disorder, usually setting in with, and throughout accompanied by, catarrh of the mucous membranes, especially those of the eyes, nose, and respiratory passages; characterised by the appearance on the fourth day of a deep rose-red, or crimson inclining to purple, eruption of soft papules or pimples, which spreads over the whole body in the course of thirty-six hours, and is preceded and accompanied by sharp fever. This terminates by crisis between the sixth and eighth days, coincidently with the fading of the rash. Convalescence is apt to be complicated with affections of the glandular system and respiratory organs.

Ætiology.—The question of the *native seat* of measles baffles all research (Hirsch). The disease was probably widely diffused over Europe and Asia in the middle ages, and at the present day the area of its distribution is practically conterminous with the entire habitable globe. Its separate identity was first shadowed forth by Forestus (1563), but it is to Sydenham (1676) that we owe the full differential diagnosis between measles and smallpox.

[a] Joh. Anglicus. *Praxis Med.* Aug. Vindel. 1595. Page 1,041.

Age does not affect the occurrence of measles as much as is generally supposed. It is a disease of all ages, although Thomas rightly declares that children under six months old enjoy a considerable immunity, or rather are less susceptible to the disease. In communities unprotected by a previous outbreak, measles attacks individuals of all ages.

The **bacteriology** of measles is scanty—the authorities on the subject up to the present being Salisbury (*American Journal of the Medical Sciences*, 1862[a]), Keating (*Phil. Med. Times*, 1882), and Cornil and Babès (*Les Bactéries*, 1885). According to Edgar M. Crookshank,[b] round cocci and diplococci have been observed in the catarrhal exudations, in the papules and in the capillary vessels of the skin, as well as in the blood of patients attacked by the disease. The *materies morbi*, therefore, may be considered to exist in the expectoration and in the cutaneous *débris*. There is no doubt that **measles is "taking,"** or infectious, **from the first sneeze or cough**—that is, from the very beginning of the initial or prodromal stage (invasion). Hence, the chief difficulty in checking its spread amongst the members of an attacked household. It is **most infectious**, however, **in the eruptive stage**, and probably **not very infectious in the stage of desquamation.** Its "striking distance" is believed to be considerable, but the contagium is less persistent than that of scarlatina (Hilton Fagge). One attack usually, but not necessarily, protects from a second—acquired immunity is not so constant after measles as it is after smallpox. Measles is a highly infectious and a very deadly disease when it attacks an unprotected community—that is, a community not leavened with the virus and in consequence not *immune*. Two notable illustrations of this have become

[a] Dr. Salisbury, of Newark, Ohio, United States, many years ago demonstrated his ability to produce a disease indistinguishable from measles by means of *musty* straw. Dr. Henry Kennedy, of Dublin, reported in the *Dubl. Journ. of Med. Science* (Vol. XXXV., 1863, p. 60), a case in which a disease like measles arose in connection with *musty* linseed meal. Thomas, of Leipzig, disposes of Salisbury's statement that the rashes produced by decaying straw afford protection against measles, by saying that it "is not to be believed for a moment."

[b] *Manual of Bacteriology.* Second Edition. London: Lewis. 1887. Page 214.

matters of medical history within recent times. They are as follow:—

In the spring of 1846, measles was accidentally imported from Copenhagen into the **Faroe Islands**, which had enjoyed an immunity from the malady for sixty-five years (from 1781). On March 20 of that year a workman left Copenhagen, being then unwittingly in the incubation stage of measles. He landed in the islands on March 28, and developed symptoms of measles on April 1. His two most intimate friends were in due time attacked, and the malady spread until at last 6,000 out of a total population of 7,782 contracted the disease. *Age* afforded no protection—old men and women fell victims as readily as young children. This outbreak is of special interest, as it enabled Dr. Panum, of Copenhagen, who was its historian, to make a series of most valuable investigations into the length of the incubation period in measles.

In 1875 the **Fiji Islands**, in the Pacific Ocean, were annexed to the British Crown. From the *Medical Times and Gazette* of June 12 in that year, we learn that "the first advantage (?) derived by annexation"—so the Fiji papers of March 20, 1875, describe it—"is the introduction of measles, and for this the islanders are indebted to H.M.S. '*Dido*,' which came down and discharged her diseased passengers, utterly regardless of any consequences that might ensue." The disease made great ravages throughout the whole of the islands. Tui Levuka and other chiefs succumbed to it, and even the hardy mountaineers in the interior had considerable havoc made in their ranks. The germs of the disorder were taken into the mountains by the chiefs who had been brought over to Levuka—the capital—and entertained on board the *Dido*. The disease, which was almost always followed by dysentery (morbillous colitis), assumed the form of a plague.

Seasonal Prevalence.—Although like smallpox, apparently **independent of climate**—for it is met with alike amidst Arctic snows, in temperate latitudes, and under the tropical sun—measles prevails especially in the **spring** and **autumnal quarters** of the year. An analysis of the weekly returns of deaths from measles in the Dublin Registration District, published by the Registrar-

General for Ireland, long since led me to the conclusion that a mean temperature above 58·6° was not favourable to the spread of this disease, and that a mean temperature below 42·0° was equally inimical to its prevalence.[a] These results are in strict accord with those arrived at by Dr. Edward Ballard, who says[b] that the only condition concerned in the arrest of the spread of measles in summer is the rise of the temperature of the air above a mean of 60° F., while towards winter a fall below 42° F. also distinctly tends to check the disease.

The accompanying Diagram (2), copied from the Annual Summary of Births, Deaths, and Causes of Death in London and other great towns for 1890, by the Registrar-General for England, is based upon the weekly returns of deaths from measles in London for the fifty years, 1841–1890, inclusive. In it, the mean line represents an average weekly number of 34 deaths from the disease under discussion, and the weekly curve shows a double maximum and a double minimum, the larger maximum falling in November, December, and January, with an extreme excess of 50 per cent. in the fourth week of December, and the smaller in May and June, with an extreme excess of 25 per cent. in the first week of June. The larger minimum falls in August, September, and October—extreme deficit being 45 per cent. below average in the last week of September, and the smaller minimum in February and March—extreme deficit, 30 per cent. below average in the third week of February.

Clinical History.—A typical case of measles may be considered to run through four stages, which we will now study in detail—they are, in chronological order, the stages of incubation, invasion, eruption, and desquamation.

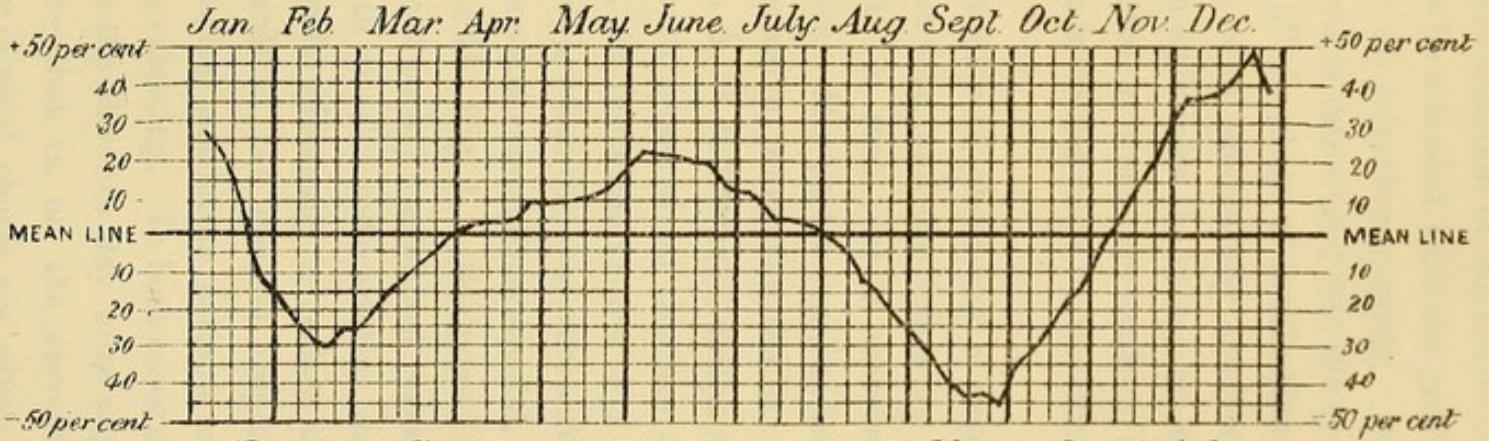
1. **Stage of Incubation.**—Ten days, as a rule, elapse between the reception of the poison into the system and the manifestation of the earliest febrile and catarrhal symptoms, or fourteen days between infection and the appearance of the rash. These are

[a] *Manual of Public Health for Ireland.* 1875. Pages 300, 301.

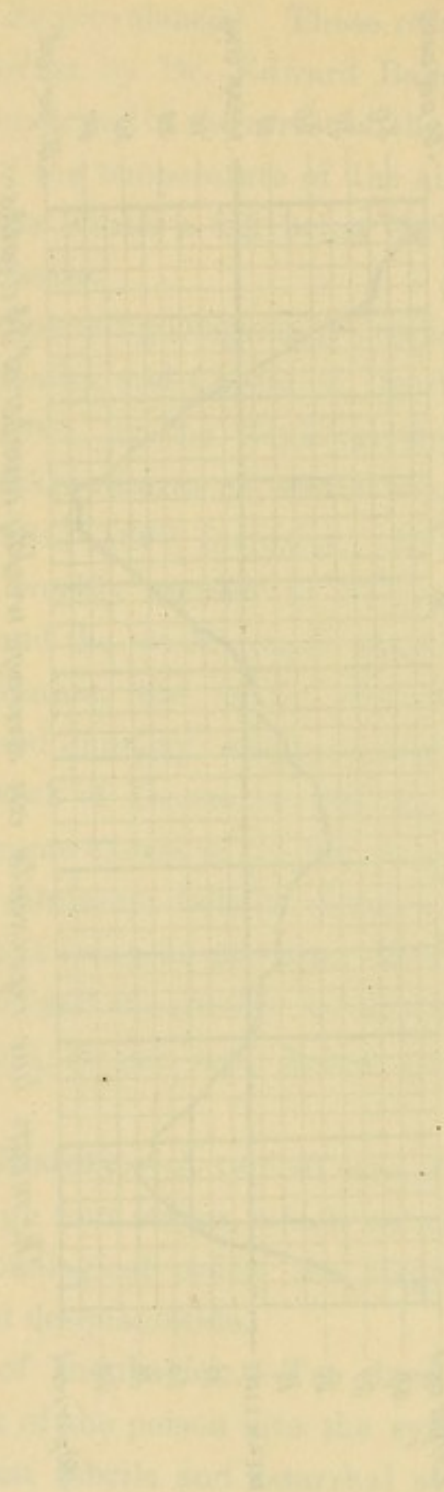
[b] *Eleventh Report of the Medical Officer of the Privy Council.* 1868. No. 3. Pages 54–62.

DIAGRAM 2. - MEASLES.

(50 Years 1841-90)



The mean line represents an average weekly number of 34 deaths.



Panum's estimates, based on his observations in a *newly-infected* community, the inhabitants of the Faroe Islands in 1846. Under other circumstances, the determination of the length of the incubative period of measles must be attended with the greatest difficulty owing to the multiplicity of infecting centres. As commonly happens when a specific virus is introduced into the system by what may be called "a short cut," the **stage of incubation is shortened after inoculation.** Hebra, who successfully inoculated with the nasal mucous, found that in such cases the symptoms showed themselves in 8 days, the difference in time representing the effect of the resistance offered to the invading virus by the skin and mucous membrane.

In 1876 I had an opportunity of observing a case of measles even from the stage of incubation, and the following facts will be of interest:—On March 26 in that year, a married lady, aged twenty-one, fell ill of measles. Her parlourmaid had sickened with the same disease *ten days* (inclusive) previously, that is, on March 17. At 3 p.m. of the 26th, Mrs. A. B. felt thirsty, chilly, and fatigued. Her appetite failed. The rash appeared on the face on the afternoon of the 30th (5th day), the fastigium (103·4° F.) was reached at 1 p.m. of April 1 (7th day), when the eyes were suffused and the rash fully out. Defervescence was complete on April 3. The clinical chart will be found in Plate II., Fig. 1. (See page 146.) In this case the duration of the latent period was almost certainly 10 days.

In the spring of 1877 both measles and scarlatina were prevalent in Dublin, and it was difficult effectually to isolate the incoming cases in the Epidemic Wards of the general hospitals. In two instances at the Meath Hospital scarlatina patients unfortunately contracted measles while convalescing. In both cases the symptoms of measles were detected on the eleventh day after exposure to the contagium of that disease—that is, on the eleventh day after admission to hospital. The clinical charts are given in Plate II., Figs. 2 and 3. (See page 146).

Few or no symptoms attend this preliminary stage. Towards its close, fatigue, lassitude, and nausea may be felt, and the pulse beats faster than in health. At this time, also, an ephemeral

fever, followed by defervescence, has been noticed by Thomas, of Leipzig.

2. **Stage of Invasion.**—The initial stage is longer in measles than in any other of the exanthemata. It lasts 4 or 5 days. The disease begins suddenly with the usual nervous symptoms of an acute fever, and, in addition, a remarkable group of symptoms connected with the mucous membranes. The patient, "sickening for measles," is seized with shivering; or, if a child, perhaps has a fit of convulsions. He complains of lassitude, headache, and pains in the joints. The skin becomes hot and dry, the pulse beats quickly, and there is a rapid rise of temperature to 102·5° or even 104° F. by the evening of the first day. This "**initial fever**," as Wunderlich calls it, is usually followed by an equally sudden fall of temperature, so that by the morning of the 3rd day the thermometer readings are normal (98·4°) or only slightly febrile (99° or 100°). As the eruptive stage approaches, a fresh rise of temperature begins. The physician should be on his guard against mistaking the initial or prodromal fever of measles for a mere febricula or ephemeral fever of non-specific origin.

But it is the **catarrhal symptoms** which are so eminently characteristic as to be pathognomonic of measles. From the outset the patient sneezes, the eyelids itch and swell, and become very red on the inside; the eyes water (*lacrymation*), look bloodshot and shun the light (*photophobia.*) A flow of acrid tenacious mucus takes place from the nostrils, the nose seems stopped up and then often bleeds (*epistaxis*), and, in a word, the individual seems to be suffering from a "bad cold in the head" (*coryza*). At the same time, the throat feels raw and sore, the voice becomes husky, and a hoarse, brassy cough sets in, occurring in paroxysms or "*kinks.*" Simultaneously there is a *catarrhal diarrhœa* in many cases, the motions being green and unhealthy-looking. On the second day an **efflorescence** may spread over the skin, leading to a wrong diagnosis of scarlatina. This **accidental rash** may also simulate urticaria because of the itchiness which accompanies it. It is, doubtless, erythematous in character. About this time, further, according to Dr. Hilton Fagge, an eruption of scattered points and spots may be

seen over the mucous membrane of the soft palate. Thomas, of Leipzig, also says that indications of the cutaneous rash may sometimes be seen on the face during this stage, in the form of minute puncta, around which the characteristic papules afterwards develop.

3. **Stage of Eruption.**—Towards the close of the fourth day—rarely earlier, very rarely later—the appearance of the true rash of measles is heralded by a renewed rise of temperature and an exacerbation of all the symptoms connected with the mucous membranes. In many cases there is a profuse serous diarrhœa; and sometimes the eruptions from the bowels become glairy and bloody, indicating the presence of morbillous colitis. The eruption appears on the face first, next on the back of the wrists, afterwards on the trunk, lastly on the limbs. It consists of **small red specks, slightly elevated and velvety** to the touch, on the forehead and face, closely resembling flea-bites (Sydenham and Hilton Fagge). It is to be remembered, however, that the face generally escapes the infliction of flea-bites. These specks become grouped in **crescentic** patches, leaving interstices of skin of normal colour. The rash, therefore, is in general discrete rather than confluent, although in some cases it is really confluent. It is of a deep rose or crimson colour, inclining to purple (Hilton Fagge). There is some swelling of the face during this stage. On the trunk and limbs the papules are less prominent than on the face. Sometimes the true eruption is interspersed with a crop of miliary vesicles with an inflamed red base. Occasionally—to use Sydenham's words—"the eruptions turn livid, and then black; but this happens only in *grown persons*, who are irrecoverably lost upon the first appearance of the blackness, unless they be immediately relieved by bleeding and a cooler regimen." These **varieties of the rash** give rise to the terms *Morbilli læves*, *Morbilli papulosi* (the French *Rougeole boutonneuse*), *Morbilli miliares*, *Morbilli confluentes*, *Morbilli hæmorrhagici* (Mayr and Hebra). During the eruptive stage the perspiration has a peculiar heavy odour, which Niemeyer compared to the smell of a freshly plucked goose.

The eruption reaches its fullest development in thirty-six hours from the time of its first appearance—that is, on the sixth day. It is then dusky, and the skin begins to look rough and

dirty. As the rash comes out, the coryzal symptoms increase, and the temperature rises to its *fastigium* or *acme* of 104° to 105° F. on the evening of the 5th day, or on the 6th day—the fever being most intense when the eruption is at its height. In this behaviour of the temperature in the **Eruptive Fever** of measles we have a **diagnostic of the first importance** between smallpox and this disease.

There is an old tradition that a sudden fading of the rash in measles is an ominous sign; but its truth is disputed by Thomas of Leipzig. At the same time there can be no doubt that the rash develops badly or not at all in the presence of a complication. This occurrence is to be distinguished from those rare instances in which we have measles without a rash (*Morbilli sine morbillis*).

With the fading of the rash, **defervescence** begins, and is almost completed within 48 hours, being so rapid and sudden as to be **diagnostic** of measles. Here we have another marked contrast between the behaviour of the temperature in smallpox and in measles respectively—apyrexia in measles taking the place of the secondary fever in smallpox. The rash fades after the sixth day, slowly and in the order of its appearance, leaving yellowish red stains, which may persist for several days. In the early stage of its subsidence the eruption of measles exactly resembles a profuse maculo-petechial rash in typhus fever, which, indeed, is often called the "*measly* rash of typhus." The morbillous catarrh—both respiratory and intestinal—frequently persists at this stage, and a nummular expectoration indicates that the bronchioles share in the affection. Now and then it happens that the catarrhal symptoms of measles are wanting, or nearly so. To this aberrant but mild form of the disease Dr. Willan gave the name of *Morbilli sine catarrho*.

4. **Stage of Desquamation.**—This commonly begins about the eighth day and ends about the eighteenth day, counting from the first symptoms. The skin peels off in small bran-like scales, hence the term **furfuraceous** desquamation (Lat. *furfur*, scurf or bran). Trousseau says that these tiny branny flakes are seldom seen, because they adhere to the dress if the patient perspires, and in this opinion

Hebra concurs. But a careful search will hardly fail to reveal this branny desquamation taking place across the bridge and along the sides of the nose, as well as about the mouth and on the neck.

In uncomplicated measles, **convalescence** may be said to be **complete on the eighteenth day** from the earliest symptoms.

In rare cases a **relapse** of the eruption has been known to occur, associated with a return of the fever movement also. In these cases, according to Thomas, the spots appear on parts of the skin previously uninvaded by the exanthem. These relapses are of short duration. Lewin[a] mentions an anomalous course of measles in two boys. The rash disappeared suddenly, without causing any perceptible injury to the patients, but only to return after two weeks. In one case, the renewed attack ran a normal course; in the other, this occurred only after a second equally harmless interruption of several days. "Such a course," says Thomas, "is perhaps a parallel one to many of those relapses appearing after the first eruption, also to Trojanowsky's **recurrent form** of measles."[b]

[a] *Jahrbuch für Kinderheilkunde.* Vol. 42, page 95.

[b] See *Dorp. med. Zeitung.* 1873.

CHAPTER XIV.

Measles (*continued*).

Classification—Complications—Temperature—Pathology—Diagnosis—Prognosis.

Classification of Measles: Niemeyer's.—Benign and Malignant.—Benign: Morbilli sine catarrho; Morbilli sine morbillis. Malignant; Purpuric Measles; Asthenic or Adynamic Measles; Complicated Measles.—Causes of Complications (Hebra).—Complications of the stage of invasion: Convulsions, spasmodic catarrhal laryngitis (false-croup), suffocative catarrh, epistaxis, otitis, diarrhœa, colitis; of the stage of eruption: morbillous diarrhœa, capillary bronchitis, pneumonia, diphtheria (true-croup); of the stage of desquamation: glandular enlargements, otitis, cancrum oris (noma), gangrene of the vulva, acute desquamative nephritis, acute miliary tuberculosis, herpes, eczema, etc., pleuritis, chronic ophthalmia, atrophic keratitis.—Temperature in measles.—Pathology.—Diagnosis: from epidemic rose-rash, scarlatina, smallpox, Grisolle sign, varicella, simple rose-rashes, typhus.—Prognosis.

Classification of Measles.—Niemeyer describes three varieties of the disease under the headings—1. *Morbilli vulgares, simplices,* vel *erethici*;[a] 2. *Inflammatory* or *synochal Measles*; 3. *Asthenic, typhous,* or *septic Measles.* Another convenient arrangement of the cases is into the two groups of **benign** and **malignant.** In the former are included two species—1. *Morbilli sine catarrho*; and 2. *Morbilli sine morbillis.* In the latter we have three species—1. *Purpuric Measles*; 2. *Asthenic* or *Adynamic Measles*; and 3. *Complicated Measles.*

According to Thomas the form of benign measles in which there is **rash, but no catarrh** (*Morbilli sine catarrho*) is especially apt to occur in very young infants, and is attended with little or no fever. Probably many cases of Rötheln were formerly classified as non-catarrhal measles. The occurrence of **measles without the rash** (*Morbilli sine morbillis*) is probably a rare clinical experience.

Malignant Measles.—1. Of this form of the disease the **purpuric** variety is very infrequent. Trousseau in his vast experience met with but two cases. It is observed chiefly in young and sickly children

[a] Gk. ἐρεθιστικός, *irritative,* or *provocative,* from ἐρεθίζω, *to rouse to anger, to excite.* Hence, *rubefacient* (or *causing redness*).

(Hilton Fagge). The rash is profuse and dark, the skin is dotted over with petechiæ and vibices, while blood oozes from the mucous membranes in all parts of the body. Death generally takes place in a few days. In Willan and Bateman's Atlas of Skin Diseases[a] will be found an illustration of the appearances presented by the skin in a form of the disease which Dr. Willan called *Rubeola nigra*. This, however, must be quite different from Purpuric Measles, for he says the dark purple or black colour of the rash continued from the eighth day for ten days or longer "*without materially disturbing the functions of the patient.*" Such a description could never apply to the "Black Measles" of the older writers—the *Morbilli nigri*, vel *petechiales*—which has been just described. The ostensible frequence of this form long ago is, according to Thomas, to be explained in part "by the preposterous treatment of old times," partly by the fact that measles and scarlatina were confounded together and malignant forms of scarlet fever were described as measles. I agree with Hilton Fagge also that no doubt cases of hæmorrhagic smallpox were sometimes classed as "Black Measles."

2. In **asthenic** or **adynamic measles**, the symptoms are severe and persistent—the fever is intense so that the patient is soon exhausted, the pulse becomes rapid and feeble, delirium gives place to somnolence, and at last the "typhoid" or "ataxic" state ensues and terminates too often in early death. The term "**ataxic state**" is applied to a group of symptoms which indicate extreme nervous prostration, namely, sleeplessness, delirium, small rapid pulse, quick shallow breathing, agitation and restlessness, tremors, plucking at the bedclothes, dilatation of the pupils, and finally deepening coma and insensibility. I will return to this subject later on.

3. According to Hebra, the **cause of complications** must be sought for—(α) in individual peculiarities of the patient, (β) in the conditions under which he lives, and (γ) in the special characters of the prevailing epidemic—to use Sydenham's classical phrase, the "**epidemic constitution**" of the time or place.

[a] *Delineations of Cutaneous Diseases.* London: Longmans, Hurst, Rees, Orme, and Brown. 1817.

It will be convenient to consider the **complications of measles** as they present themselves in the stages of onset or invasion, of the rash, and of convalescence respectively.

Stage of Invasion.—Measles is not uncommonly ushered in by an attack of **convulsions** in young or nervous children. Such an attack or "**fit**" is simply an exaggerated rigor with cerebral phenomena. It is not of very serious import unless it persists for over two days. Trousseau mentions a very remarkable case in which the tonic stage of the convulsive fit lasted for two minutes and a half and death occurred.

The mucous membrane of the larynx is often the seat of ulcerations and erosions, which occasion severe laryngeal symptoms, constituting the condition known as "**false-croup**," or **spasmodic catarrhal laryngitis**. The cough is dry and harsh, frequent and spasmodic. The voice is hoarse, every movement of the larynx is painful, and and inspiration is laboured, accompanied with wheezing, whistling sounds like those of asthma. At times, especially after a fit of coughing or when drinking, suffocative spasms and a painful sense of oppression across the chest occur (Mertens [a]).

Suffocative catarrh may occur during any of the stages of measles. Its symptoms are: high fever, oppression of the chest, dyspnœa, and moist cough. Children, as a rule, do not spit out, but swallow the expectoration—hence it cannot conveniently be examined; but in adolescents and adults the sputa consist at first of thin, limpid mucus; afterwards they become copious and muco-purulent in character, occasionally rusty or prune-juice, if the so-called croupous pneumonia supervenes. Three stages or degrees of bronchitis—the most common and the most perilous of the complications of measles—are recognised: ordinary tracheo-bronchial catarrh, capillary bronchitis or bronchiolitis, and broncho-pneumonia—also called catarrhal pneumonia, or lobular pneumonia. In young children this affection is dangerous from purely mechanical causes—suffocation being brought about by œdema of the bronchial mucous membrane and accumulation of secretion. Of the three forms of bronchial affection just described, tracheo-bronchial

[a] *Canstatt's Jahresbericht.* 1852. IV. Page 210.

catarrh is most common in the stage of invasion; the other and severer forms in that of eruption.

Epistaxis, or **nose-bleeding**, may be so severe as to endanger life or permanently injure health.

Otitis is hard to diagnosticate in an infant, but cries and delirium from pain should suggest the presence of ear-trouble, and suppuration leading to otorrhœa usually solves the difficulty in 36 or 48 hours.

Diarrhœa is dangerous if continuous to the time when the rash is due to appear, if the rash does not "come out" well, and if the eyes look sunken with dark rings around their lids. The intestinal morbillous catarrh often attacks the colon, causing **colitis** with glairy, bloody stools, and tenesmus. This accident usually occurs in the eruptive stage.

Stage of Eruption.—In this stage, in addition to morbillous diarrhœa, **capillary bronchitis** and **pneumonia** are frequent and most dangerous complications of measles. Their approach is indicated by, perhaps, an attack of convulsions—a most fatal occurrence in this stage of the disease—by persistently high temperature after the sixth day, and by early and rapid fading of the rash. Bronchitis is a very obstinate complication, running on, it may be, to twenty or thirty days should the patient survive so long.

Diphtheria of the pharynx and of the larynx, **membranous laryngitis**, or **true croup**, is another dangerous, though rare complication of the eruptive stage. The diagnosis is based on the detection of false membranes and the presence of signs of laryngeal stenosis, or narrowing, due to mechanical obstruction.

Stage of Desquamation.—During this stage we meet with, as sequelæ and in addition to broncho-pneumonia, **glandular enlargements** in the neck and in the thorax (bronchial glands). These may go on to suppuration and cicatrisation, or the glands may become the seat of tubercular deposits or infiltrations.

Otitis in this stage may involve the middle ear, and affect the brain and dura mater through the mastoid cells; or it may give rise to purulent infection by inducing septic phlebitis and thrombosis of the cerebral sinuses. Trousseau, in his *Clinical Medicine*,

reports a remarkable case in point, which was seen in consultation and diagnosticated by Dr. Peter.[a]

Cancrum oris or **noma**, and **gangrene of the vulva**, may occur in delicate, badly-fed and badly-clothed children, sometimes speedily destroying life, but in non-fatal cases leaving disfiguring losses of tissue.

Acute desquamative nephritis is much less common in measles than it is in scarlatina.

Acute miliary tuberculosis often supervenes upon an attack of measles, the tubercles invading specially the lungs and the membranes of the brain.

Herpes and **eczema** often follow measles, attacking the upper lip, nasal fossæ, posterior nares, and Eustachian tube.

Pleuritis was observed as a sequela of measles by the late Professor Fleetwood Churchill, of Dublin, and by Professor Seidl.

Chronic ophthalmia ("Exanthematous ophthalmia" of Wardrop), with "granular lids" and distressing photophobia, and **atrophic** or **secondary keratitis**, must be included among the many eye affections which follow measles.

In considering the subject of the complications and sequelæ of this disease, it should never be forgotten that an attack of measles often sows the seed of even a fatal constitutional delicacy.

Temperature.— According to Wunderlich, the characteristic features in the temperature movement in measles are —

1. In the stage of incubation, a short preliminary fever course in the form of **an ephemera**, in which the thermometer may mark 102·8° to 103·6°. This is followed by a pause or interval of several days quite free from fever.
2. An **initial** or **prodromal fever** occurring in the stage of invasion, the temperature rising from the time of the first symptoms to 102·4°, and even 104°, on the evening of the first day. Next day a decided fall, so that on the third day the temperature is either normal or subfebrile. This

[a] *Lectures on Clinical Medicine delivered at the Hôtel Dieu, Paris.* New Syd. Soc. Translation. 1869. Vol. II., page 231.

Plate II.

CHARTS OF TEMPERATURE RANGES IN MEASLES.

Fig. 1.—Simple Measles.

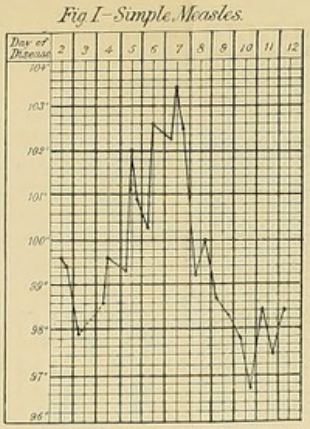


Fig. 2.—Measles. (after Scarlatina)

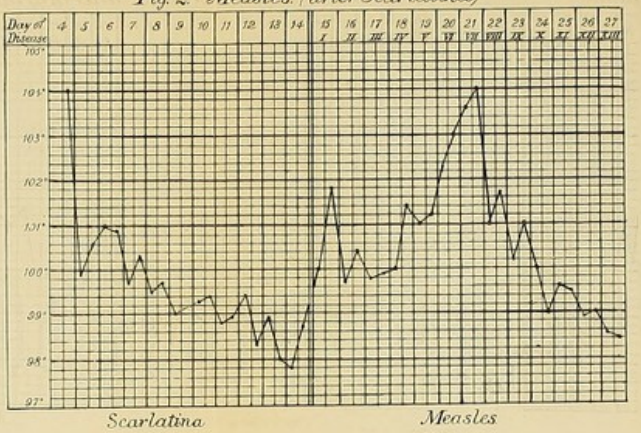


Fig. 3.—Measles, (after Scarlatina.)

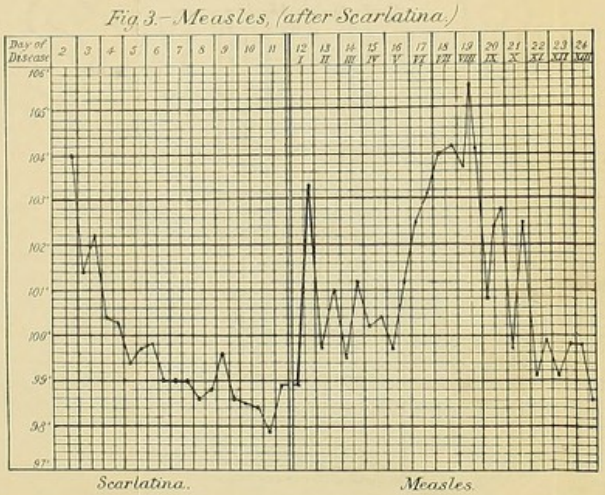
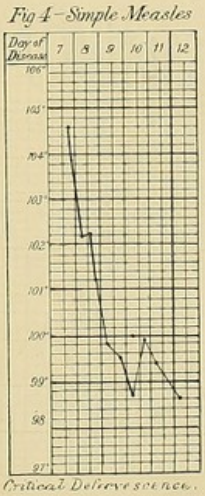


Fig 4—Simple Measles



pyrexial movement may easily be confounded with an ephemeral fever.

3. The **true eruptive fever**, setting in shortly before the rash is due, and consisting of (*a*) a moderately febrile stage which lasts from 36 to 48 hours, and (*β*) the stage of fastigium which corresponds in point of time with the fullest development and extension of the exanthem, and lasts from one and a-half to two and a-half days.
4. Decided **defervescence** in uncomplicated cases begins according to rule in the night, and generally runs a rapid course, being completed—after one or two evening exacerbations—by the eighth or ninth day of the attack.

The appended charts will illustrate the behaviour of the temperature in measles. (See Plate II.)

Pathology.—Among the pathological changes produced by measles, Mayr and Hebra mention, in the first place, a **catarrhal inflammation** of the mucous membrane of the respiratory tract; but assuredly the intestinal mucous membrane is also the seat of a specific catarrh.

It is true that Thomas, of Leipzig, while stating that, as a rule, the mucous membranes of the nose, the throat, the upper air-passages, and the conjunctivæ are attacked, and often much sooner than the outer skin, cannot admit any similarity of this affection of the mucous membranes with that of the skin, and so does not recognise the perfect justness of the term "exanthem of the mucous membranes."

But the appearances described by Thomas himself, by Hilton Fagge, and by others in connection with the visible mucous membranes, as well as the catarrhal symptoms, justify us in concluding that the mucous membranes are really the seat of an exanthem, or, more correctly, an *enanthem*—a view which the analogy of smallpox altogether supports.

The **nasal mucus**, according to Mayr and Hebra, is at first transparent, afterwards opaque; its reaction is always alkaline, never acid.

The same authorities assert that the **efflorescence** consists essentially in the pouring out of exudation around the mouths

of the hair-sacs. This view is disputed by Thomas, who says that, if the hair-follicles specially participated in the inflammatory process in morbillous dermatitis, the scalp would necessarily be the proper focus of the exanthem, and the palms of the hands and the soles of the feet would remain free—neither of which is the case. G. Simon also, in 1848, found no change in the hair-sacs, sebaceous glands, or cutaneous papillæ, but described the epidermis as being still in immediate contact with the corium, although slightly swollen at those points where papules exist.

Diagnosis.—The recognition of measles depends, first on **the exanthem**; secondly, upon **the mucous membrane symptoms** and the **characters of the fever** (the behaviour of the temperature); thirdly, upon a consideration of the **existing epidemic** and the exposure of the patient to the virus of measles.

Measles must be distinguished from—Rötheln, or Epidemic Rose Rash (Roseola); Scarlet Fever, Variola, Varicella, simple Roseolar Rashes, and Typhus Fever.

In **epidemic rose rash**, the stage of invasion is only 1 to 3 days, the pyrexia is much less than in measles, the catarrhal symptoms are slight, and the rash is less typical. The lymph-glands also are more swollen than in measles.

In **scarlet fever** there is a short invasion, only 24 hours; vomiting is constantly observed; catarrhal symptoms are absent, while there is early and severe sore throat with marked swelling of the lymph-glands. The tongue presents the appearance known as "strawberry-tongue." The fever runs a continuously severe course and drops gradually by lysis, with morning remissions and evening exacerbations. The rash is commonly confluent, consisting of bright red or scarlet points with a diffuse hyperæmic blush. The kidney affection of scarlatina is wanting in measles.

The most common error of diagnosis is between measles and **smallpox**, but the development of the pustular rash of smallpox sets the question at rest. At the beginning, too, confluent smallpox presents an eruption on the *second* day compared with the *fourth* or *fifth* day in measles. The papules of smallpox are hard and shotty; those of measles are soft and velvety. The application of the test called the "Grisolle sign" is also to be remembered (see p. 98).

Absence of catarrhal symptoms, a short invasion-stage, its slight pyrexia, and the life-history of the vesicles, serve to distinguish **varicella** from measles.

Simple rose rashes are distinguished by absence of fever and of catarrh, and by their inconstant and evanescent nature.

Typhus in children may be mistaken for measles, although my experience is that young children have typhus as a rule lightly, with only a scanty rash or without any rash at all. The catarrh of measles is wanting, its rash is brighter than that of typhus, and does not pass through the macular and petechial stages of the typhus eruption. The diagnosis may be assisted, as Murchison says, by examining other members of the same family who may be affected at the same time. Measles usually attacks children; typhus rarely attacks children before the adult members of a family.

Prognosis.—This in primary and uncomplicated measles is **thoroughly favourable**, death from the severity of the infection alone being extremely rare (Thomas.) The mortality of measles varies greatly from time to time according, as Sydenham would say, to the "**Epidemic Constitution**" of the time and place. Sometimes only 2 or 3 per cent. of the patients die; occasionally, the death-rate reaches the alarming figure of 50 per cent. In 1845 and 1846 Trousseau says he lost 22 out of 24 children with broncho-pneumonia in the Necker Hospital, Paris.[a] Measles is a mild disease in sucklings under six months old; it becomes severe at the first dentition, and is sometimes very severe in adults. In pregnancy it is a dangerous malady. It is most fatal among persons who are ill-nourished, rachitic, or tuberculous (scrofulous.) It kills from 6,000 to 14,000 children yearly in England, 90·5 per cent. of the victims being under five years of age.

Unfavourable symptoms in a case are: great weakness and excitement from the outset; a hot, dry skin; a hard and rapid pulse; quick, laboured respiration with a short cough; early fading of the rash, or a change of colour in it; or a persistent (petechial) rash. By far the most fatal complication of measles is bronchitis in its severer forms.

[a] *Clinique médicale de l'Hôtel Dieu de Paris.* 1865. Tome I., page 145.

CHAPTER XV.

The Treatment of Measles.

Prophylaxis: quarantine, isolation, hygienic measures.—Curative Treatment: no specific for measles—treatment is symptomatic and hygienic. Treatment of Complications: Malignant Measles—cool baths (Dieulafoy).—Initial convulsions, false and true croup, epistaxis, otitis, diarrhœa, ophthalmia, glandular enlargements, noma, gangrene of the vulva, acute tuberculosis.

I. Prophylaxis.—Owing to the fact that measles is infectious from the outset, and that the first case in a household may readily be mistaken for an ordinary cold, the attempt to stay its spread by adopting preventive measures, and especially isolation, is practically futile, Hence the extreme difficulty of arresting an outbreak of this disease in a family or a community.

Should an individual have been exposed to the infection of measles, at least sixteen days' quarantine will be necessary before he can be pronounced safe. A patient, again, who has passed through an attack of measles, should not be declared free from infection until at least three, and preferably four, weeks have elapsed from the first symptoms. It will be remembered that the stage of desquamation generally lasts until the eighteenth day.

With the view of lessening the susceptibility to measles, the greatest attention should be paid to the sanitary surroundings of a community.

II. Curative Treatment.—Dr. Hilton Fagge aptly points out that "the general plan of treatment in measles and in scarlatina is the same, for in neither of these maladies have we any specific method of dealing with the malady itself."

Bearing in mind the tendency to **catarrh of the respiratory mucous membranes** which exists in measles, a mild, equable atmosphere is essential (60° to 65° F.). In winter and spring a steam kettle should play in the sick room, at all events at night. Ventilation must be effected with caution, all draughts or undue lowering of temperature

being carefully avoided. Children should be closely watched at night lest in their feverish restlessness the bed-clothes should be thrown off, and they in consequence should be exposed to cold. The hands and face should be washed daily. The patients should be allowed to drink water freely, or bland mucilaginous drinks like linseed tea, barley water, and toast-water. The diet should consist of milk and animal broths, the latter to be thickened with rice, arrowroot, or gelatine in cases of diarrhœa. On the other hand, in constipation (which, in measles, is rare), oranges, stewed prunes, baked apples, and so on, may be given. During the stage of desquamation, the patient should be kept as far as possible in bed. Warm baths are specially useful, as they relieve troublesome itching, remove the *débris* from sweat and desquamation, and generally soothe the patient. The surface of the skin may afterwards be oiled with advantage—liniment of camphor, a weak carbolised oil or soft paraffin, or "hazeline cream," being used for the purpose. All exposure to cold should be avoided until the catarrhal symptoms have entirely disappeared. In convalescence, fresh air, driving in the open country, and change of air to the seaside or some sheltered inland health-resort, are most desirable.

Among **drugs** suitable for administration to convalescents from measles, mention may be made of quinine, saccharated carbonate of iron, cod-liver oil with saccharated solution of lime, and chloride of calcium.

The skill of the physician is often taxed to the uttermost in the treatment of the **complications** of measles.

In **malignant measles**, Dieulafoy, of Paris, recommends[a] a bath at 26° C. (78·8° F.) for twelve minutes, with cold affusion on the head. These measures will reduce temperature (102·9°) and the respirations (70). A second bath at five o'clock in the afternoon; a third at 9 p.m., a fourth at 2 a.m., a fifth at 5 a.m. These will probably be followed by marked improvement, reduction of temperature, and sleep. A sixth bath, at 6 p.m.

The cold bath re-establishes the secretion of urine, the skin

[a] *La Pratique Journalière des Hôpitaux de Paris.* Par le Professeur Paul Lefort. Paris J. B. Baillière et Fils. 1891. Page 312.

becomes soft, and temperature falls to 101·1° F. As regards the rash, it becomes pale but runs its course.

In a case of **initial convulsions** Trousseau's advice is excellent—"Wait—avoid boisterous practice." Find out if the patient is subject to fits. If the attack persists, compression of the carotids may be practised in the way recommended by Trousseau—namely, by pressing with the thumb on the common carotid opposite to the affected side of the body, or alternately on each side of the neck, keeping up the pressure for some fifteen minutes,

In **false and true croup** the patient should be placed in a croup-tent, and a simple plan recommended by Graves and heartily approved by Trousseau should be followed. This is an application during twenty or twenty-five minutes of relays of sponges soaked in hot water to the neck and throat.

These same measures will prove useful in suffocative catarrh also, in which hot poultices and the application of a cuirass of cotton wool or French wadding to the chest wall are very effective. Trousseau recommends stinging the chest with nettles (urtication) as an effectual means of producing counter-irritation.

In **epistaxis**, the application of ice to the forehead and that of a cold key to the nape of the neck are household remedies not to be despised. Either iced water or *very hot* water may also be injected into the nostrils. In extreme cases it becomes necessary to plug the posterior nares.

In **otitis**, the external meatus should be gently syringed, or a chamomile flower soaked in warm almond or olive oil, or in vaseline, may be inserted every few hours, taking care to cleanse the passage each time. Sometimes the application of a single leech to the mastoid process allays pain and affords immediate relief.

While **diarrhœa** is often checked by good nursing and suitable dieting, it may be necessary, as recommended by Trousseau, to give minute doses of opium in lime-water (one-thirtieth of a grain in 24 hours), or to administer albuminous enemata, if **colitis** occurs. Alterative doses of perchloride of mercury may also be of use. Dr. Charles West recommends decoction of logwood with white sugar and port-wine. This mixture stains linen or cotton fabrics.

For **ophthalmia**, fomentations with strained decoction of poppies or infusion of chamomile, or both, or with cold tea—which contains tannin—often give relief. When a sequel of measles, ophthalmia requires constitutional treatment—iron and quinine, cod-liver oil, and wine or eggflip.

Glandular enlargements are best combated by a generous, wholesome diet, with milk, cod liver oil, iron, iodide of iron, quinine, chloride of calcium, and arsenic. Change of air is especially desirable in the management of this troublesome sequela.

Fresh air and perfect cleanliness are the best preventives of **noma, gangrene of the vulva**, and such like complications. The patient's strength must be supported by nutritious food and wine. Caustics may be used in certain cases—namely, hydrochloric acid, nitrate of silver, sulphate of copper, or the actual cautery.

The prevention of **acute tuberculosis** may be attempted by change of air, wholesome surroundings, a nutritious diet with goat's milk and koumiss (mares' milk fermented), syrup of the chloride of calcium in milk, cod liver oil, syrup of the iodide of iron, compound syrup of the hypophosphites or of the phosphates, and syrup of the lacto-phosphate of lime. A favourite combination with me is embodied in the following prescription:—

℞. Liquor. Calcii Chloridi, ʒvj;
Acid. Hydrochloric. dil., ʒj;
Liquor. Strychninæ Hydrochlor., ʒj;
Acid. Hydrocyanic. dil., ʒss;
Liquor. Arsenic. Hydrochlorici, ʒss;
Aquæ Chloroformi, ad ℥viij.
M. fiat. Mistura.

The dose of this mixture for an adult is a tablespoonful by measure in water twice or thrice daily after food. A child aged 10 or 12 years may take half this dose.

CHAPTER XVI.

Scarlatina, or Scarlet Fever.

Nomenclature.—Definition.—Ætiology (historical sketch).—Area of Diffusion.—Epidemic and sporadic outbreaks —Rate of Mortality most variable.—Predisposing Causes: Climatic influences; season.—Exciting Cause: Specific poison—"Contagium Vivum."—Professor Klein's Researches: *Streptococcus scarlatinæ*.—Hendon Cow Disease.—Clinical History: Scarlatina simplex, anginosa, and maligna.—Varieties of Scarlatina simplex.—Stages of Incubation, Invasion, Eruption, Desquamation.—Prominent Symptoms: Vomiting, sore-throat, *tache scarlatinale*, "strawberry-tongue," albuminuria.

Nomenclature.—"Febris scarlatina" (*Ital. Scarlatto*, scarlet) was the name by which Sydenham (1675) and afterwards Withering (1778) designated this disease. Hence the synonyms "Scarlatina" (Boissier de Sauvages, Vogel, and Cullen) and "Scarlet Fever." It had been described as far back as 1556 by Ingrassias, of Palermo, under the name "Rosalia." The equivalents are:—*Latin*, Febris Rubra (Heberden). *Germ.* Scharlachfieber, or, shortly, Scharlach. *Fr.* Fièvre rouge, Scarlatine. *Ital.* Scarlattina, Febbre rossa. *Span.* Escarlatina. *Danish* and *Norwegian*, Skarlagensfeber. *Swedish*, Skarlakansfeber. *Dutch*, Scharlakenkoorts.

Morton, a contemporary of Sydenham, described the disease as *Morbilli confluentes*, confounding it with measles. Another name was *Morbilli ignei*, that is, "Fiery Measles."

Definition.—An acute specific infectious fever, characterised by a sudden onset with vomiting, rigors, and prostration; early and persistent sore throat, deep injection of the mucous membranes of the throat, which are swollen and inflamed; very rapid pulse-rate and high fever; and especially by the appearance upon the skin after a few hours of a minutely punctiform scarlet rash, which is most intense on the third day, and afterwards fades gradually, to be succeeded by profuse desquamation of the cuticle in both small and large flakes. A specific nephritis is a not uncommon complication or sequela. Three varieties of the disease are recognised—namely, simple, anginose, and malignant scarlatina.

Ætiology.—The origin and native habitat of scarlet fever are

quite unknown. By far its largest area of distribution is on European soil (Hirsch). In Germany, France, the British Islands, Scandinavia, and Russia, it is one of the chief factors in the statistics of sickness and mortality. Up to the present it has a very scanty diffusion in Africa and Asia. It occurs but rarely in India, is said to be unknown in Japan, and assumes generally a mild type in Australia, Tasmania, and New Zealand, in which countries it first broke out in the beginning of 1848. The first appearance of scarlet fever on the soil of North America dates from 1735, when it broke out in Kingston, Massachusetts, thence spreading to Boston and other places near, and finally overrunning the whole of the New England States in the course of the next few years.[a] Nearly a century elapsed before scarlet fever began, about 1830, to be generally diffused over South America.

The **area of diffusion** of scarlet fever is much smaller than that of smallpox or of measles. Its **epidemics arise at long intervals**—ten or twenty years or more often intervening between two successive outbreaks at a given place. Its epidemics are, however, of protracted duration; and sporadic outbreaks arise from time to time in the intervals.

Scarlatina displays extreme variation in its intensity—the **rate of mortality** being almost *nil*, or only from 3 to 5 per cent. of those attacked, in some epidemics; but in others rising to 30 per cent. or upwards. No more striking proof of this can be adduced than the words in which Sydenham described scarlet fever—"*Hoc morbi nomen, vix enim altius assurgit.*" According to Graves[b]—the historian of the disease in Ireland—in the months of September, October, November, and December of the year 1801, scarlet fever "committed great ravages in Dublin, and continued its destructive progress during the spring of 1802. It ceased in summer, but returned at intervals during the years 1803–4, when the disease changed its character, and although scarlatina epidemics recurred

[a] *The Practical History of a New Epidemical Eruptive Miliary Fever.* By Dr. Douglas. Boston: 1736. Reprinted in *The New England Journal of Medicine*, January 1825. P. 1.

[b] *A System of Clinical Medicine.* By R. J. Graves. Dublin: 1843. P. 493. Also *Clinical Lectures on the Practice of Medicine.* By the late R. J. Graves. Dublin: Fannin & Co. 1864. Pages 230, *et seq.*

very frequently during the next twenty-seven years, yet it was always in the simple or mild form." In 1834–35 another destructive epidemic raged in Dublin, and so to the present day outbreaks alternately benign and malignant have been observed, according as the "**Epidemic Constitution**" varied, as Sydenham would say.

Predisposing Causes.—Climatic Influences do not play a prominent part in determining the geographical distribution of the disease, for although the tropical and sub-tropical regions of Asia and Africa have so far almost entirely escaped scarlet fever, yet it has often prevailed epidemically in the tropical countries of South America; and, on the other hand, in certain cold or temperate climates scarlet fever is among the rarest of diseases.

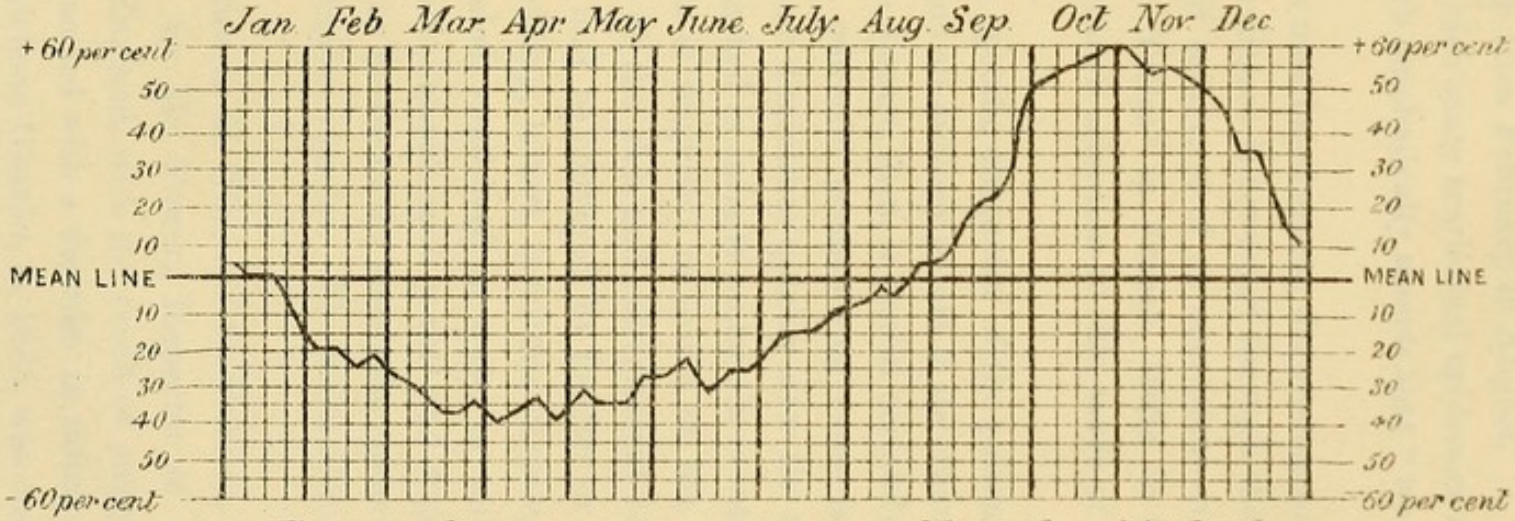
There is, however, evidence that **season** does influence its prevalence. "*Scarlatina*," observes the Registrar-General of England,[a] "discovers a uniform, well-marked tendency to increase in the last six months, and attain its maximum in the December quarter, the earlier half of the following year witnessing a decrease." In Dublin, also, the disease is almost invariably most prevalent and fatal in the fourth quarter of the year.

From an analysis of the weekly death-rate from scarlatina in Dublin it would seem that this fever shows a tendency to increase when the mean temperature rises much above 50°, while a fall of mean temperature below this point in autumn checks the further rise of the mortality.[b] In this city, scarlet fever is most fatal in the forty-sixth week of the year (middle of November) and least fatal in the twenty-fourth week (middle of June). Dr. Edward Ballard draws inferences which confirm these results.[c] The "Annual Summary of Births, Deaths, and Causes of Deaths," of the Registrar-General of England, for 1890, is illustrated by the annexed Diagram (3.), showing the weekly mortality curve for scarlet fever in London on an average of 30 years (1861–1890). The curve consists of a single wave, which rises to its crest (60 per cent. above the mean line, which represents an average weekly number of 44 deaths) in October and November, while the trough

[a] *Twenty-eighth Annual Report of Births, Deaths, and Marriages.* Page 38.
[b] *Manual of Public Health for Ireland.* 1875. Pages 303 and 304.
[c] *Eleventh Report of Med. Officer of Privy Council.* 1868. No. 3. Pp. 54–62.

DIAGRAM 3.—SCARLET FEVER

(30 Years 1861-90)



The mean line represents an average weekly number of 44 deaths.

extends from February to August. It is a suggestive fact that the corresponding curves for diphtheria and enteric (or typhoid) fever are also single wave curves, closely resembling each other and that of scarlatina in rising to a crest in October and November, and showing a trough from February to August.

As to the conditions which cause scarlatinal epidemics to assume a good or bad type, Hirsch emphatically asserts that "we are completely in the dark."

Traumatism should be mentioned among the predisposing causes of scarlatina. When the disease is conveyed through the infection of a **wound**—the result of injury, or of an operation—it is called "**Surgical Scarlatina.**" Trélat[a] in France, Hoffa in Germany, and other authors, have drawn attention to this mode of origin of scarlatina.

Exciting Cause.—The **specific character** of the poison of scarlet fever is unquestionable, and all analogy points to its organic nature. The exciting cause of the disease, then, is almost certainly a micro-organism, while its most powerful predisposing cause is *age*—the **susceptibility** to the disease diminishes quickly after the tenth year of life. It is not well marked in infants under six months old.

The **contagium vivum** is singularly retentive of life and most active. Hence isolation of the sick and convalescent is rendered imperative. The virus may be conveyed by fomites or dress and other articles, such as bedding, letters, furniture, carpets, and such like; it may also be inhaled, swallowed in water or milk, or conveyed by means of domestic animals such as cats and dogs. Scarlet fever may be **inoculated** by means of the blood, the epidermic scales, and the serum from cutaneous vesicles. The urine also is believed to be infective.

An epidemic of scarlatina in St. Giles and St. Pancras, London, in 1882, was investigated by Mr. Power, Inspector of the Local Government Board for England, with the result of suggesting that the disease might be connected with a **disorder in milch cows.**[b] A similar outbreak in Marylebone District, in 1885, was clearly traced to the use of milk obtained from milch cows on a certain farm at

[a] *Le Progrès Médical*, Septembre 14, 1878. [Also see *below under* "Accidental Rashes in Enteric Fever."]

[b] *Fifteenth Annual Report of the Local Government Board*, 1885–86.

Hendon.[a] These cows suffered from an eruptive disease of the udder and teats—a condition first introduced there in November, 1885, by some cows newly arrived from Derbyshire. From the diseased tissues of man and cow alike a **special streptococcus** (*Streptococcus scarlatinæ*) was separated by Professor Klein. This micro-organism grows on gelatin in opaque white colonies, and it does not liquefy the gelatin. It is slower of growth than most micrococci. After several weeks it has a fairly distinct but slightly irregular and crenate outline, here and there beset with dark knobs or linear processes. When grown in milk it coagulates the milk. Some of the elements may be observed as diplococci or as short chains. Cultivated in broth it forms long and exquisite chains. Subcultures of these micrococci have the property, when inoculated into calves, of producing in them every manifestation of the "**Hendon disease**," except sores on the teats and udders. The subcultures produce in rodents a disease pathologically similar to cow-scarlatina and human scarlatina. Calves fed on subcultures obtained from human scarlatina contract the Hendon disease. In the Marylebone epidemic it was proved that children fed on milk from cows with the Hendon disease contracted scarlatina. These facts, in the opinion of Dr. Buchanan, Medical Officer of the English Local Government Board, form a mass of evidence that the Hendon disease in cows is a form of the very disease which we call scarlatina when it occurs in man. This view awaits final confirmation.

Prior to Klein's investigations, micrococci had been described by Coze and Feltz[b] as being present in the blood of patients suffering from scarlatina; and Pohl-Pincus[c] detected them in the scales of the desquamating epidermis and in the discharges and ulcerated tissue of the throat. These micrococci form small colonies, and stain violet with a saturated solution of methyl violet. Their diameter is very small—only about 0·0005 mm.[d]

Clinical History.—Cases of scarlatina are arranged under three headings, namely—

[a] *Seventeenth Annual Report of the Local Government Board*, 1887–88.
[b] *Maladies Infectieuses.* 1872
[c] *Centralblatt für die med. Wissenschaften.* No. 36. 1883.
[d] *Micro-organisms and Disease.* By E. Klein, M.D., F.R.S. 1886. Page 78.

1. **Scarlatina simplex**, or mild scarlet fever, in which the disease runs its course without complications or untoward sequelæ, terminating in an uninterrupted convalescence.

2. **Scarlatina anginosa** (Lat. *ango*, I strangle; Gk. ἄγχω—hence, *angina*, quinsy; Gk. ἀγχόνη, a throttling or strangling), in which the affection of the throat is severe and the cervical glands are sharply engaged.

3. **Scar atina maligna**, in which extreme nervous prostration, with its attendant ataxic or "typhoid" symptoms, is the most striking and ominous phenomenon.

Of simple scarlet fever certain varieties have been described—as *Scarlatine fruste*, *Defaced* or *Latent*, *Rudimentary* or *Abortive Scarlet fever*, *Scarlatina sine exanthemate* or *Scarlatina sine scarlatinis*, and *Scarlatina faucium*.

We will now consider the disease as it passes through its various stages or periods.

I. **Stage of Incubation.**—This period is very short, probably never extending beyond a week and rarely lasting so long. It may be only twenty-four hours, but on the average it varies from **three to five days.** In 1861 Trousseau declared that neither in measles nor in scarlet fever could the duration of the latent period "be rigorously determined in the present state of our knowledge," while still later Obermeier gave it as his opinion that the incubation period of scarlatina was unknown. Dr. Charles Murchison,[a] Consulting Physician to the London Fever Hospital, collected 75 cases of scarlet fever, having any bearing on the question, which had either come under his own observation or been communicated to him in the twenty years ending with 1878. Of the total 75 cases, in not one did the incubation period exceed six days; in 73 cases it could not have exceeded five days; in 54 cases it could not have exceeded four days; in 20 cases it could not have exceeded three days; in 16 cases it could not have exceeded two days; and in 3 cases it could not have exceeded twenty-four hours. It also appears

[a] *Observations on the Period of Incubation of Scarlet Fever, and of some other Diseases.* Clinical Society's Transactions. Vol. XI., page 238. 1878. Cf. *Braithwaite's Retrospect of Medicine.* Vol. LXXIX., pages 1-5. 1879.

that the longest period of incubation made out in any of the cases was four and a half days; and that in only two of the cases was it certainly as long as four days.

Murchison's observations and study of the **incubation period** of scarlet fever led him to the following conclusions:—

1. The duration of the incubation stage may be only a few hours.
2. Probably in a large proportion of cases it does not exceed forty-eight hours.
3. It very rarely exceeds seven days.
4. Consequently, a person who has been exposed to scarlet-fever, and does not sicken after a week's quarantine, may be pronounced safe.

Some of the following cases occurred in the practice of my father, Dr. William Daniel Moore, some in my own practice.

On November 2, 1848, a gentleman, aged twenty, went down to the County Wexford from Dublin three weeks after the disappearance of the rash of scarlet fever. On his arrival the same day he saw two sisters, both of whom took to bed with scarlet fever on November 8—that is, in six days—incubation = five days.

A boy aged eight, sickened with scarlet fever on January 12, 1851. His sister took ill on the 17th, and five days later another sister sickened.

Two girls, cousins, slept in the same bed on April 5 and 6, 1855. One of them complained of sore throat on the night of April 5, and showed the rash of scarlatina on the morning of the 7th. The other sickened on April 10th—that is, on the fourth day.

Of two sisters, one took ill on December 27, 1871, and was at once isolated. The other remained under the same roof, but without seeing her sister until they met by accident on February 4, 1872. She sickened with scarlet fever seven days later, on Feb. 11.

A lad, George G., aged fourteen, complained of pain in his eyes on Saturday, January 5, 1878. Next day he sneezed a little. On Monday and Tuesday he had slight headache. On Wednesday he was admitted to the Epidemic Ward of the Meath Hospital because of a rose-rash on his face. He perspired a good deal and felt thirsty. His temperature was 100·8°, pulse 96, and respirations 24. On

Plate III.

CHARTS OF TEMPERATURE RANGES IN SCARLATINA.

Fig. 1– Scarlatina Simplex.

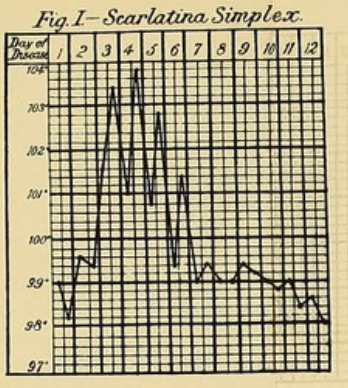


Fig. 2– S. Simplex.

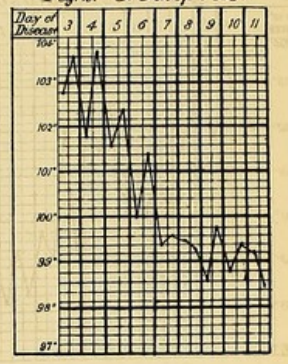


Fig. 3– S. Anginosa.

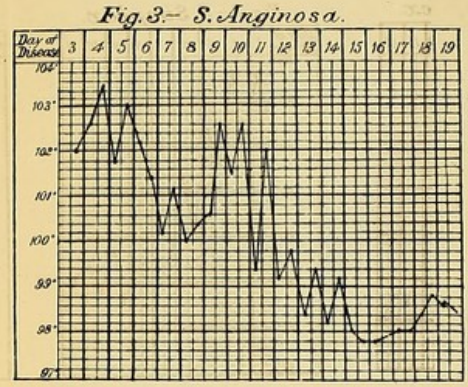


Fig. 4– S. Anginosa.

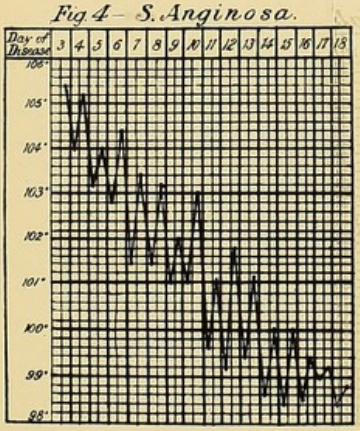


Fig. 5– S. Typhoides.

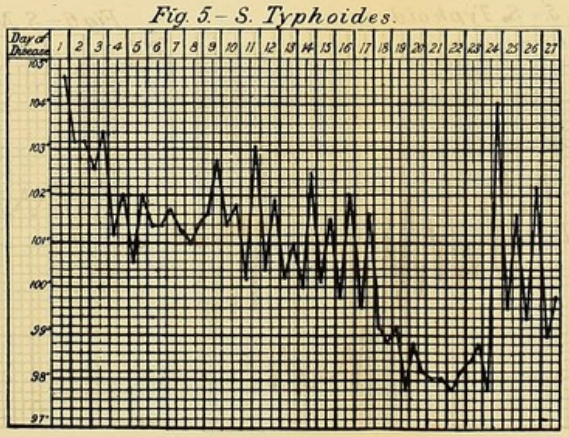
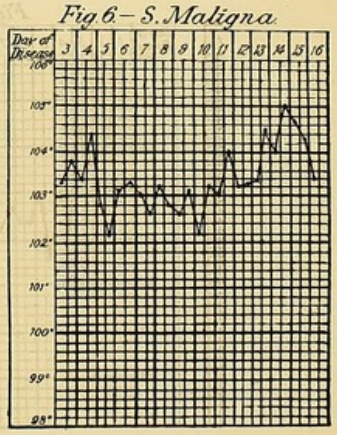


Fig. 6– S. Maligna.



examination he was ascertained to have had measles three or four years previously in the County Carlow. The case, therefore, was regarded as one of Rötheln. On January 14, a bad case of scarlatina was placed in the next bed to that occupied by George G., and between him and the door of the ward. On January 20, he was seized with sore throat and vomiting, and next day the rash of scarlatina came out. The clinical chart of this case will be found in Plate III. (Fig. 1.) The incubation period here could not have exceeded six or seven days.

This stage of latency may be unattended by symptoms, or towards its close there may be slight headache, malaise, lassitude, and loss of appetite.

II. **Stage of Invasion.**—Like the stage of incubation, this period may be of **very short** duration—most commonly only **twenty-four hours.** Sometimes it is still shorter, or very rarely it may extend to several days, as in a case reported by Trousseau, in which rebellious cerebral symptoms persisted for eight days and then suddenly subsided on the appearance of sore throat and the rash of scarlet fever. But such a case is altogether exceptional, and Dr. Hilton Fagge considered that a question might fairly be raised as to its original nature. This pre-eruptive, prodromal, or initial stage is shorter in scarlatina than in any other fever.

The onset of the malady is abrupt. **In children,** the earliest symptoms are usually **vomiting** and diarrhœa, rigors, or a convulsive seizure. According to Trousseau, the occurrence of **convulsions** during the first or second day is always a **sign of danger,** whereas in other exanthemata it is not of evil omen (Hilton Fagge). In **adults, sore throat** is generally the first symptom: it is accompanied by chilliness or rigors, headache, malaise, and prostration. Meanwhile both pulse and temperature rise quickly. Even on the evening of the first day, a child's pulse may range between 140 and 160, his temperature may rise to 103°, 104°, or even 105°. This rapidity of the pulse-beat is almost pathognomonic of scarlatina. The skin is hot and dry. Hilton Fagge says that from the time of Addison the **pungent heat of the skin** in scarlet fever has been spoken of at Guy's Hospital as comparable only with that which is

to be felt in acute pneumonia (**calor mordax**). The tonsils, soft palate, and uvula are deeply injected and often more or less swollen with pappy exudation, resembling ulcers, upon their surface. They are plum-coloured. The neighbouring lymphatic glands in the neck also are frequently swollen.

The Schneiderian mucous membrane and the conjunctiva are seldom engaged--thus affording a diagnostic from measles.

III. **Stage of Eruption.**—The rash generally shows itself within 12 or 24 to 30 hours from the first symptoms. It may be detected very early on the sides of the neck and over the chest, as well as in the neighbourhood of large joints. It afterwards spreads to *parts* of the face, to the abdomen, and over the limbs. Thomas insists that only the forehead and temples are invaded by the rash, the cheeks showing only the ordinary flush of fever. The centre of the chin and a zone round the mouth usually remain free from it.

The rash consists of **minute red dots**, with a general or patchy **suffusion of the skin**, of a bright scarlet colour, which suggested to Sir Thomas Watson, Bart., a comparison to the colour of a boiled lobster. On the forearms and legs and on the backs of the hands and feet, the papules are larger and more prominent than elsewhere. The palms and soles show only a faint diffused blush. The eyelids, cheeks, hands, and feet usually swell slightly. Bouchut drew attention to a sign of some diagnostic value, which is called the **Tache scarlatinale.** This is a white stripe or streak which develops and lasts for a few moments when the finger is drawn across the reddened surface—the contractile power of the cutaneous arterioles being increased. Accidental pressure, by the bedclothes, &c., produces a precisely similar effect, which is well represented in Plate XXII. of Willan and Bateman's "Delineations of Cutaneous Diseases." Probably nowhere is the eruption so marked as on the abdomen and along the inside of the thighs.

Sometimes a "millet-seed" rash of tiny vesicles (*miliaria*) is observed. These become filled with a milky (lactescent) fluid after 36 to 48 hours. In other cases the eruption is blotchy, macular, or papular. According to Trousseau, the amount of the rash is in

general proportionate to the severity of the attack, but in complicated or malignant cases it may be badly developed.

The rash reaches its limits by the second or third day, then fades gradually, leaving persistent blood-coloured (petechial) lines in the folds in front of the elbows, in the axillæ, and popliteal spaces. These streaks may be of use for diagnosis.

The appearances presented by the **tongue** in scarlet fever are characteristic. At first, it is covered with a thick, creamy, white fur, through which the enlarged and hyperæmic papillæ project as little scarlet protuberances. The fur is soon shed (early desquamation), leaving the tongue red and raw, so as to resemble a ripe strawberry—hence the expression "**strawberry-tongue**" and "**cat's tongue.**"

The **fever** runs **very high** in this stage, and the pulse beats very quickly (140–160).

Defervescence is gradual, extending over from 3 to 8 days. The temperature "spikes" slightly in the evenings, but remits in the morning. To such a temperature chart Wunderlich assigns the name "easel-like." This term will be explained further on, under the heading "Temperature."

IV. **Stage of Desquamation.**—The process of "peeling" sets in on the neck and chest between the sixth and ninth days; then it affects the limbs, the hands, and lastly the soles of the feet. Branny scales come off from the face (furfuraceous desquamation), flakes of cuticle are shed from the trunk, and sometimes "gloves" of skin from the hands and feet—even the nails may be shed. Much more frequently, however, interference with the nutrition of the nails is shown by the formation of a transverse groove upon them. This was fully described by A. Vogel in the case of typhus fever, and is illustrated by drawings in Murchison's work on the "Continued Fevers of Great Britain." Dr. Samuel Wilks drew special attention to this atrophic furrow in the nails of scarlet fever patients.

In this stage, **albuminuria** often makes its appearance, one reason being the shedding of the tubal epithelium in the kidneys, in consequence of which albumen escapes into the urine.

Hilton Fagge describes the process of desquamation as taking place in a manner which I have often observed. "The first step," he says, "towards the throwing off of the epidermis at a particular spot is often the formation of a little opaque raised vesicle, very like those which are characteristic of eczema, but dry; this breaks at the summit, leaving a free edge in the shape of a ring, which gradually becomes larger and larger."

The duration of the stage of desquamation is really indefinite—in some cases lasting for a fortnight only, in others for several weeks—perhaps, even months.

Relapse is a very infrequent phenomenon in scarlatina. The most recent contribution to the literature of the subject will be found in the number of the *Edinburgh Medical Journal* for October, 1891, in which Mr. George P. Boddie reports two cases of what he terms "a true relapse or recrudescence in scarlet fever," illustrated by the temperature charts. Of the nature of the second case Mr. Boddie speaks with some reserve, but he has no doubt as to the first case. A boy, aged fourteen, passed through an ordinary attack of scarlatina simplex, followed by slight albuminuria and desquamation, partly scurf-like, partly flaky. He was out and about when, on the 37th day, he was again seized with symptoms of scarlatina. Desquamation began on the 7th day, and was more extensive than in the first attack. At the end of five weeks—72 days from the first illness—the boy was perfectly well.

Mr. Boddie has appended to his clinical record a very full *résumé* of the literature on the subject of "**Relapse in Scarlatina.**" Trojanowsky[a] states that among 300 cases (260 children and 40 adults) he has seen 18 relapses (15 children and 3 adults), that is, 6 per cent. Thomas and Körner, both of Leipzig, distinguished between what they call **a pseudo-relapse** (*ein Pseudo-recidiv*) and **a true relapse** (*ein wahres Recidiv.*) These authors have severally seen cases of the latter. Körner, in particular, supplements his paper by giving notes of 38 cases of true relapse as reported by various writers, whom he names. Henoch[b] gives notes of 8 cases.

[a] *Jahrbuch für Kinderheilkunde.* 1873.

[b] *Lectures on Children's Diseases.* New Syd. Soc. Vol. II., p. 232, *et seq.*

Gumprecht, among 228 cases of scarlatina at the Friedrichshain Hospital, says he met with 13 "pure cases of relapse"—*i.e.*, 5·7 per cent.

French writers, such as Jaccoud,[a] and Rilliet and Barthez[b] express the opinion that scarlatina is capable of recrudescence or relapse like typhoid fever, and that we possess well authenticated cases of this phenomenon.

English writers are more sceptical. Thus Hilton Fagge[c] asserts that "relapses are seldom or never seen." Drs. Ashby and Wright, in their *Handbook on the Diseases of Children* (page 213) say, "Reinfection or relapses *are said* to take place in some instances." In supposed cases of relapse which came under their own notice, "there was considerable doubt as to the correctness of the original diagnosis."

Dr. E. O. Hopwood,[d] of the London Fever Hospital, estimates, however, that "a relapse, or true second attack of scarlet fever, occurs in about one-half per cent. of cases at any time from the tenth day of the first attack, and is followed by a second desquamation. It may be of any grade of severity."

Fürbringer, the writer of the brilliant article on "Scharlach" in the *Real-Encyclopädie der gesammten Heilkunde*, states that it always appeared to him that the relapse was "a fever following the true initial scarlatina, independent of it, caused by no apparent complication, of very variable type, and lasting for days or weeks."

As for my own experience, I cannot recall a single instance in which a true relapse came under my cognisance, although I have often observed accidental febrile movements during convalescence from scarlatina.

[a] *Traité de Pathologie Interne.* 1883. III. 558.
[b] *Traité des Maladies des Infants.* 1891. III. 153.
[c] *Principles and Pract. of Med.* Vol. I., p. 216. 1886.
[d] *Dict. Pract. Med.* Edited by J. K. Fowler. 1890. Art. "Scarlet Fever."

CHAPTER XVII.

Scarlatina (*continued*).

Aberrant Forms—Complications and Sequelæ—Temperature.

Irregular or aberrant forms of scarlatina: (1.) Rudimentary or abortive: (α.) simple scarlatinal angina (*scarlatina faucium*), (β.) latent scarlatina (*scarlatine fruste*); (2.) Scarlatina anginosa—diffuse cellulitis of neck ("*tippet-neck*"), diphtheria; (3.) Scarlatina maligna: (α.) Angina maligna, (β.) ataxic scarlatina, (γ.) hæmorrhagic scarlatina—"Scharlachtyphus."—Complications and Sequelæ: Diphtheria, acute rheumatic arthritis and serous inflammations, acute desquamative nephritis, pleuritis, bubonic swellings, pyæmia, boils and abscesses, otitis, eye affections, eczema, chorea.—Temperature ranges in Scarlatina.

In the last Chapter a word-picture was attempted to be drawn of scarlet fever as the disease presents itself in a typical or normal case. But a striking feature in the malady is its polytypical character, the great variability of its symptoms and of its course. It will be necessary, therefore, shortly to describe some of its leading **irregular** or **aberrant forms**, as Hilton Fagge calls them.

1. Of the **rudimentary** or **abortive**, and therefore mild and favourable forms, the most frequent is (1.) **Simple Scarlatinal Angina.** This is observed usually in adults, whose predisposition to the disease is slight, during the prevalence of an epidemic. Its features are—fever of moderate intensity, slight sore throat with redness and some swelling of the mucous membrane, pain in the neck, and, it may be, enlargement of the cervical glands. This mild scarlatinal angina was described by Dr. Alex. Tweedie, in an excellent monograph on *Scarlatina*,[a] under the name **Scarlatina Faucium.** In Graves's "Clinical Lectures"[b] will be found a series of cases of it detailed by the late Dr. John Ridley, of Tullamore,

[a] *The Cyclopædia of Practical Medicine.* London: 1834. Vol. III. Art. "Scarlatina."

[b] *Loc. cit.* Page 247.

King's County. Thomas[a] says: "Such attacks generally disappear in a few days, but they should receive the same attention which is paid to the unmistakable disease; every throat affection during a scarlet fever epidemic is suspicious!" This is all the more necessary as the most malignant forms of the disease may arise from *scarlatina faucium*. (2.) In other cases, the throat affection is absent or so trifling that it does not account for the existing fever. There may be a slight rash, and the cervical glands may be enlarged. In such attacks, joint affections of a rheumatic nature or kidney trouble may supervene. This is disguised or **Latent Scarlet Fever**—the form to which Trousseau gave the name of "*defaced* scarlatina" (**Scarlatine fruste**). As an inscription is *defaced* by being in part obliterated, so a disease may be defaced by the absence of some of its prominent and distinguishing features.

2. In marked contrast to the foregoing mild forms, we often meet with that severe variety to which the name **Scarlatina anginosa** is given. It is the second of the three classical forms. In this anginose variety the constitutional disturbance and fever are well marked, and, in addition, the patient as early as the second day complains of stiffness and pain about the neck and jaws, and under the ears, the throat feels rough, deglutition is difficult and painful, and there is some hoarseness. The tonsils are swollen, and of a deep purple or plum-like colour. On their surface small whitish specks of pappy or pultaceous exudation appear. These resemble ulcerations, but are really accumulations of secretion from the mucous crypts or follicles—which have taken on increased action—and of epithelium, which is already desquamating freely. The mucous membranes of the pharynx, mouth, and nose, all share in this affection, so that both respiration and deglutition may be seriously interfered with; an offensive sanious discharge takes place from the nostrils, the breath is fœtid, the voice becomes nasal, and drinks return by the nose. The scarlatinal affection of the nasal mucous membrane may also extend to the cavities adjacent to the nose, especially the antrum of Highmore; or not infrequently to the Eustachian tubes, in which latter case deafness of a temporary,

[a] Von Ziemssen's *Cyclopædia of Medicine*. Art. "Scarlatina."

sometimes of a permanent, nature, is added to the patient's troubles.

Nor does the mischief end here. In this form of the disease, the tonsils may suppurate, or, still worse, become gangrenous after an abscess has formed and burst—the destructive process extending to and eating away the arches of the palate, the uvula, and even the whole of the soft palate itself. Ulceration of the tonsils may also lead to erosion of the internal carotid artery, necessitating ligature of the common carotid of the same side.

It is in scarlatina anginosa, also, that after the eighth day the cervical glands swell sometimes enormously, a low erysipelatous or phlegmonoid inflammation engaging the cellular tissue of the neck. The swelling which attends this "**diffuse cellulitis**" gives rise to the expression "**tippet-neck**"—a condition said to be fatal if it involves both sides of the neck. The brawny swelling is often white, smooth, glossy, and hard as a board; it pits on pressure, but conveys no sense of fluctuation. It is situated at the angle of the jaw, or involves the whole lower jaw, stretching upwards to the temples and downwards to the clavicles.[a]

The diseased mucous membranes in scarlatina anginosa lastly afford a fertile soil for the growth of the micro-organisms of **diphtheria**—the *Streptococcus* described by Oertel, and the *Bacillus* of Löffler. As a matter of fact, diphtheria is a not uncommon complication of the disease.

3. Some of the severer forms of scarlatina anginosa just described might justly be included under the heading **Scarlatina maligna.** But apart from the malignant form of scarlatina anginosa, formerly called **Angina Maligna** (Fothergill, 1754), the term Scarlatina maligna is reserved specially for two terrible varieties of the malady—**Ataxic Scarlatina** and **Hæmorrhagic Scarlatina.**

Nervous or **Ataxic Scarlet Fever** is ushered in with rigors, convulsions, and even tonic spasms with trismus, incessant vomiting

[a] A good account of "Diffuse Cellulitis of the Neck," by Mr. Henry Gray Croly, President of the Royal College of Surgeons in Ireland, will be found in the *Dublin Journ. of Med. Science* for May, 1873, Vol. LV., page 407, Nov. 17, Third Series.

and diarrhœa, wakefulness, agitation, and restlessness with delirium, and high fever (**hyperpyrexia**). A temperature of 115° F. was observed by the late Dr. Bathurst Woodman in some patients,[a] but this must be considered as altogether phenomenal. The maximum observed by Wunderlich was 110·3° F. (43·5° C.) The foregoing symptoms are quickly followed by another group indicative of profound nervous prostration (**ataxia**)—namely, muscular tremors (*subsultus tendinum*), plucking at the bed-clothes (*carphology* or *floccitatio*), the *coma vigil* described by Sir Wm. Jenner, Bart., dilated pupils, coma, quick shallow breathing, and extreme rapidity and feebleness of the heart's action, and profuse cold or clammy sweating. Needless to say that such attacks prove rapidly fatal. The rash is badly, if at all, developed, and the diagnosis of scarlet fever depends largely on the occurrence of undoubted cases in the same house or the immediate neighbourhood.

In the other variety of malignant scarlet fever, with early severe constitutional symptoms of a "typhoid," "ataxic," or "septic" character, the rash comes out late and imperfectly. Its colour is dark—"a livid violet," as Hilton Fagge calls it—and reddish brown points of **hæmorrhage**, with petechiæ and vibices, are found scattered over a large part of the surface in children—less extensively in adults, over the neck, chest, back, and skin near the large joints. When the rash presents this hæmorrhagic form the tonsils and gums are generally of an abnormally dark colour (Mayr). Such a rash is figured in Plate XXII. of Willan and Bateman's "Delineations of Cutaneous Diseases." Hæmorrhages take place from the mucous membranes, especially of the nose, colon, and urinary passages.

In women, menorrhagia may occur. The internal organs may be the seat of hæmorrhages in both sexes, and finally pleuritic or pericardial hæmorrhagic extravasations may take place (Thomas).

To these grave forms of the disease Hebra gave the name of **Scharlachtyphus**, or **Scarlatinal Dissolution** or **Decomposition of**

[a] *Medical Mirror*, February, 1865. See note 2, page 204 of Dr. Woodman's Translation of Wunderlich's *Medical Thermometry* for the New Sydenham Society. London. 1871.

the Blood, regarding them as instances of a scarlatina without localisation and affecting the blood alone (*eine Scarlatina ohne Localisation, ein im Blute verlaufender Scharlach*).[a]

Complications and Sequelæ.—Among the complications of the acute stages of scarlet fever we may include the **ataxic symptoms** above described, the **acute hæmorrhagic diathesis** (hæmophilia) of the malignant variety, and the **sore-throat** of the anginose form, with its consequences. But, in addition to these, mention must be made of the following complications :—

1. **Diphtheria**, in which the mucous membranes of the mouth, pharynx, and nostrils become covered to a greater or less extent with a false membrane of an ashy gray colour and leathery consistence, closely adherent, and, if removed, quickly replaced on the ulcerated and bleeding, or simply ulcerated surface. This complication is almost invariably associated with albuminuria and is generally followed by various paralytic symptoms and dangerous heart failure, or attacks of *angina pectoris vaso-motoria* of Nothnagel.

2. **Acute rheumatic arthritis** and **inflammations of the serous membranes** occur tolerably often in the course of scarlet fever—all the phenomena of an attack of acute rheumatism may be present, and permanent endocardial mischief may result.

3. **Acute desquamative nephritis** generally sets in towards the close of the first, or in the second week, or at any time during convalescence. It is a parenchymatous and interstitial (E. Klein) inflammation, and is ushered in with suppression of urine (anuria) or hæmaturia, œdema of the eyelids, pallor, thirst, and high temperature. When the urine is passed it is found to be scanty, highly albuminous, as the result—(1) of hæmorrhage from intense hyperæmia, (2) of an abnormal perviousness of the walls of the glomeruli in consequence of which transudation of albumen takes place into the urine (Strümpell). The urine also is of high specific density (1020–1030), smoky appearance, and acid reaction. Some of the densest urines are met with in acute scarlatinal nephritis, and Dr. Walter G. Smith[b]

[a] Hebra. *Diseases of the Skin.* New Syd. Soc. 1866. Vol. I., page 201.
[b] Saundby. *Lectures on Bright's Disease.* Bristol: John Wright & Co. 1889. Page 177.

has recorded a sp. grav. of 1065 in one instance. Under the microscope altered red blood corpuscles and granular, bloody, and epithelial tube-casts are visible. Barthez and Rilliet have observed anasarca in one-fifth of all their cases of scarlet fever. The affection may terminate—(1) in recovery; (2) in death, with convulsions; or (3) very rarely in chronic general nephritis.

4. **Pleuritis, with purulent effusion** as in any septic fever.

5. **Bubonic swellings**, which occur chiefly in the neck towards the decline of the rash.

During convalescence, the **sequelæ** most likely to occur are—

1. **Pyæmia.**

2. **Boils** and **abscesses.**

3. **Otitis**, with perforation of the membrana tympani from pressure by pus pent up in the middle ear. Wendt contrasts the catarrhal inflammation of the Eustachian tube or of the middle ear which accompanies measles, with the suppurative *otitis media* of scarlatina. A still more serious condition is when mastoid disease occurs, with its consequences—inflammation of the dura mater, thrombosis of the cerebral sinuses, pulmonary embolism and purulent infiltration, cerebral abscess, panophthalmitis from embolism.

5. **Eye affections**, namely, **diphtheritic conjunctivitis, atrophic keratitis, panophthalmitis, retinitis albuminurica.**

6. **Eczema.**

7. **Chorea**, or St. Vitus's Dance (*Dance de St. Guy*), in two, three, or even six months after the attack.

Temperature.—The behaviour of the temperature in scarlet fever is less typical than it is in smallpox or measles. Usually, however, one of the first symptoms is a **rapid and continuous rise of temperature** in a few hours to 103° or 104° F. With the coming out of the rash the thermometer may be seen to rise slowly, with only slight morning remissions, the height finally reached being almost always above 104°, very commonly over 104·9°, but seldom in cases which terminate favourably over 105·8° (Wunderlich).

As a rule, the **intensity of the fever** bears a tolerably close **relation to that of the exanthem.**

When the rash begins to fade, *defervescence* commences. It may occur quickly and uninterruptedly by crisis; but in an overwhelming majority of cases it is protracted, requiring from three to eight days for its completion. "As a rule," says Wunderlich, "it occurs in this fashion, that from day to day the temperature gets gradually lower and lower, and slopes like an **easel**, or almost **easel-wise** (*Staffel-weise*), or goes down with trifling remissions, falling especially at night, remaining about the same from morning to evening, or perhaps sinking a little till it reaches the normal." It is only very seldom that a remitting defervescence produces a remote resemblance to that which is peculiar to typhoid or enteric fever.

Complications, of course, interfere with the usual temperature ranges.

A **subnormal** temperature—seldom, however, below 96·8°—accompanied by other symptoms of collapse, is not of infrequent occurrence towards the close of the stage of defervescence.

An **anomalous** course of temperature is not uncommon—thus, it may be persistently rather low—a condition which does not exclude danger, and by no means guarantees a favourable termination.

Again, in **Scharlachtyphus** (the so-called "*typhoid-scarlatina*"), the temperature may remain more or less high for a fortnight or longer after the rash has faded. It is sub-continuous or remittent in form, yet in general it takes a descending course.

In *fatal* cases, the temperature is very erratic, but sometimes it reaches very high degrees indeed. Wunderlich met with a pre-agonistic reading of 43·5° C. (110·3° F.), and Woodman's still more extraordinary observations have been already noted.

Plate III. contains a series of charts culled from my own Notes, which will fairly illustrate the behaviour of the temperature in scarlatina.

CHAPTER XVIII.

Scarlatina (*continued*).

Pathology, Diagnosis, and Prognosis.

Pathology of Scarlatina: The blood—the cutaneous affection—the throat and sub-maxillary glands.—Cerebro-spinal system.—The abdomen—small intestine—*psorenterie*—kidneys.—Glomerulo-tubal Nephritis (Klebs).—E. Klein's views.—Diphtheritic Pyelitis.—Changes in the urine.—Dropsy.—Inflammations of (α.) serous membranes, (β.) of synovial membranes of the joints.—The heart—acute parenchymatous myocarditis—cardiac failure.—Diagnosis: Erythema, Smallpox, Measles, Rötheln, Erysipelas, Diphtheria, Acute Rheumatism.—Prognosis and Mortality.—Causes of Death.

Pathology.—The **blood** is darkened in colour, thin, and generally contains an excess of white blood corpuscles. The walls of the blood-vessels often imbibe the colouring matter of the blood, and thus appear inflamed (Thomas). The **cutaneous affection** of scarlatina is not merely a hyperæmia, but is also characterised by an exudation into the rete Malpighii. Histologically, Löschner, of Vienna, found exudation cells in this structure, and Dr. Hilton Fagge thinks they were probably seen by Dr. Fenwick also, who further observed that the basement membrane of the sweat-glands was thickened, and their channels were obstructed by an overgrowth of epithelium or by extravasated blood. That the eruption bears no definite relation to these glands or to the hair follicles, seems to follow from a case reported by Landenberger and quoted by Thomas,[a] in which it did not fail to develop itself over an immense cicatrix from a burn which extended over the thigh, abdomen, and back, "the skin having been destroyed in its whole thickness." Slight desquamation followed, and the scar underwent diphtheritic necrosis throughout a portion as large as the hand.

The process of desquamation may involve the **nails** of the fingers and toes, and the **hair** may fall out. Lentin and Bicker have seen **warts** drop off after scarlet fever.

[a] Art. "Scarlatina," in von Ziemssen's *Cyclopædia of Practical Medicine*. Page 212.

The changes in the **organs of the throat** are as essential features in the pathology of this disease as the exanthem itself. They have been sufficiently described under the heading *Scarlatina anginosa.*

Barthez and Rilliet found the **submaxillary glands** enlarged, hyperæmic and softened, and in a later stage, grayish, soft, and the seat of purulent infiltration. The affection of the cellular tissue is undoubtedly inflammatory, and probably results from septic poisoning, or from hæmorrhage—it is a **diffuse cellulitis.**

Notwithstanding the occurrence of severe cerebral and spinal symptoms in bad cases, the **cerebro-spinal** pathology of scarlet fever may be regarded as a negative quantity.

As regards the **abdomen** and its contents, the only specific changes are those met with in the **small intestine** and in the **kidneys.** From the duodenum to the cæcum and colon the mucous membrane is more or less injected, and Brunner's and Lieberkühn's glands, as well as the solitary follicles and Peyer's patches, are swelled and prominent, giving rise to the appearance known as **psorenterie.**[a] Deiters describes the patches as being sometimes ulcerated, when a most striking resemblance to the changes found in typhoid or enteric fever is presented.

Next to the skin and throat, the **kidneys** are the organs most frequently affected by scarlatina. Early in the disease a **catarrhal condition** is found, the epithelium in the medullary substance being cloudy, desquamating in large masses, and being rapidly washed away. Later, a **true parenchymatous nephritis** sets in, when the cortical substance is particularly affected. In the severer cases, the capsules of the kidneys are easily detached and the organs are enormously enlarged (Biermer).

Klebs has occasionally found the kidneys firm and hyperæmic but not enlarged, with nucleolar growths in the connective tissue between the capillary loops of the Malpighian corpuscles, completely compressing the calibre of the vessels and so causing **anuria.** The morbid process begins in the Malpighian corpuscles, and then

[a] Gk. ψώρα, *scabies* or *itch, mange ;* ἔντερον, *the intestine.* The French use the word *Psore* as a generic title for vesicular and pustular maladies of the skin, and the idea of *roughness* is also connected with the Greek ψώρα.

follows the course of the convoluted tubes (**Glomerulo-tubal Nephritis**).

Even when no renal symptoms are present, E. Wagner says that the kidneys are in a condition of **congestive hyperæmia** either with or without a more or less marked degree of albuminous infiltration. Very rarely the severer forms of parenchymatous nephritis are met with even in the beginning of scarlet fever; as a rule, however, not before the end of the second, or in the third week.

So far as I know, the fullest account of the pathological changes in the kidney induced by scarlatina is contained in a communication made to the Pathological Society of London by Dr. E. Klein, on April 17, 1877.[a]

The early changes are met with in the vascular apparatus and certain of the glandular parts of the kidney. The vascular changes are limited, for the most part, to the cortical portion of the organ. They are—(1.) Increase of nuclei (probably, epithelial nuclei), covering the glomeruli of the Malpighian corpuscles. (2.) Hyaline degeneration of the afferent arterioles of the Malpighian corpuscles. (3.) Multiplication or germination of the nuclei of the muscular coat of the minute arteries, and a corresponding increase in thickness of the wall of these vessels.

The changes referring to the glandular part of the kidney are indications of **parenchymatous nephritis** consisting in swelling up of the epithelial lining of some of the convoluted tubes and germination of the nuclei of epithelial cells, especially in portions of the ascending tubules lying close to an afferent arteriole of a Malpighian corpuscle. Granular matter, and even blood, may be found in the cavity of Bowman's capsules and in the convoluted tubes, and also cloudy swelling and granular disintegration of the epithelium in some parts of the convoluted tubes. In some cases there appears to be detachment of epithelium from the membrane of the larger ducts of the pyramids.

Klein considers that the multiplication of the muscle-nuclei and

[a] *The Anatomical Changes of the Kidney, Liver, Spleen, and Lymphatic Glands in Scarlatina of Man.* Trans. of the Path. Soc. of London. 1877. Vol. XXVIII., p. 430.

the corresponding increase in thickness of the coat, accompanied by an increase of thickness and number of the muscle-fibres, does not mean a real hypertrophy. He suggests, also, that the **anuria** and **uræmia** of scarlatina are brought about, not, as is supposed by Klebs, by a *compression* of the vessels of the glomeruli by the nuclear germination, but by the glomeruli being *shut out of the circulation* owing to the changed state of the arterioles, which are abnormally contracted, probably under the influence of some stimulus (perhaps, some blood irritant).

In early cases the parenchymatous changes found are slight.

The second set of changes refer to cases which died later than the first week, beginning with about the ninth or tenth day. Here we find changes due to **interstitial as well as parenchymatous nephritis**—they are (1.) The appearance of round cells, lymphoid cells, or whatever they may be called, in the connective tissue of the kidney; (2.) Certain concomitant alterations of the urinary tubes. The infiltration with round cells is observable after the end of the first week in the connective tissue around the large vascular trunks, whence it spreads into the basis of the pyramids, and especially into the cortex. Portions of the latter, and—very seldom—portions of the basis of the pyramids also, are converted into a pale, firm, round-cell tissue, in which the original urinary tubes of the cortex become gradually quashed and lost.

This **interstitial nephritis** begins about the **end of the first week**, and is followed by a marked increase in the parenchymatous nephritis. This latter consists in crowding of the urinary tubes with lymphoid cells, granular and fatty degeneration of the epithelium of the uriniferous tubes, and the formation of cylinders of different kinds in the tubes. The intensity of the parenchymatous change is, in fact, dependent upon the degree of the interstitial nephritis.

A very curious fact, pointed out by Klein, is the **deposit of lime matter** in the epithelium and lumen of the urinary tubes, first of the cortex, then also of the pyramids at an early stage of scarlatina, when the kidney otherwise shows only very slight change.

Biermer, Coats, and Wagner regard the occurrence of interstitial nephritis in scarlatina as unusual. Each of these observers describes

one case as unique. According to Klein, on the contrary, the general rule is that cases of scarlatina which die after about nine or ten days show more or less well-marked interstitial nephritis.

In scarlatina hæmorrhagica, Huguenin demonstrated a diphtheria of the mucous membrane of the pelvis of the ureter (**diphtheritic pyelitis**) which had given rise to copious hæmaturia.

The **changes in the urine** which mark the rise, progress, and subsidence of acute parenchymatous nephritis are the following:—

1. An **increasing turbidity**, with grayish-white or dark-coloured sediments, consisting of an excess of epithelium—cloudy and degenerated or swollen—granular detritus, and red and white blood corpuscles.

2. When hæmorrhage from the renal parenchyma supervenes, the urine becomes reddish-brown, or "**smoky**," and **very turbid** from urates, depositing them and an abundance of epithelium and casts—bloody, epithelial, and granular. The secretion is very concentrated—shown by a high specific gravity (1025–1030), much diminished in quantity, sometimes almost suppressed. **Albumen** is present in daily increasing quantities. The albuminuria has been already explained above (see page 170).

3. **Improvement** is indicated by an **increase in the amount** of urine secreted. It becomes **clearer** and of **lower specific gravity**, loses its dark colour, no longer contains blood or throws down sediments, and contains albumen in daily diminishing quantity. The tube casts are now less numerous, and are partly epithelial or granular, and partly hyaline or waxy. The usual duration of the albuminuria is from two to three weeks, and casts are discharged for about a month.

Dropsy is a common result of scarlatinal nephritis. It usually shows itself as anasarca, when desquamation apparently ceases; less frequently as an effusion into the serous sacs (ascites, hydrothorax, hydro-pericardium, hydrocephalus), or as œdema of the lungs and glottis, or as a general dropsy. It is right to mention that dropsy may also be present without albuminuria, and not due to any obvious cause, so far as the kidneys are concerned. It sometimes even occurs without marked anæmia.

Inflammations of the serous membranes, with sero-plastic or purulent exudation, occur now and then. They are independent of the kidney affection, although often observed during its course. Pleuritis is the most common of these inflammations; meningitis, peritonitis, and pericarditis, occur less frequently.

Inflammations of the synovial membranes of the joints are usually met with just when desquamation is beginning, but may occur at any other period. They constitute what is known as scarlatinal rheumatism. The condition presents itself either as a more or less intense synovitis acuta with serous effusion, or as a suppurative arthritis, terminating in ostitis and periostitis, caries and necrosis, or pyæmia and death. Scarlatinal rheumatism may also be accompanied by peri- or endocarditis.

The **heart muscle** suffers severely, especially from the hyperpyrexia which accompanies the severer forms of scarlet fever. Its fibres are the seat of an acute molecular disintegration or of a more chronic fatty degeneration, the result of an **acute parenchymatous myocarditis.** At the close of the first volume of his "Lectures on Children's Diseases," translated for the New Sydenham Society in 1889, by Dr. John Thomson, Professor E. Henoch, of the University of Berlin, speaks of "the fatty albuminous degeneration of the heart muscle which occurs pretty often after acute infectious disease, especially **scarlet fever,** diphtheria, and typhoid, and clinically gives rise to no symptoms, except, perhaps, those of cardiac debility." (Page 491.)

In the second volume of the "Cyclopædia of the Diseases of Children"[a] there is a short, but interesting, article on "Acute Parenchymatous Myocarditis," from the pen of Dr. J. Mitchell Bruce, Physician and Lecturer on Therapeutics at the Charing Cross Hospital, London. This article seems to me to throw much light on the ætiology of the anginal attacks of acute febrile disorders. Under the names of "**acute parenchymatous degeneration,**" "**albuminous degeneration,**" "**febrile softening of the heart,**" "**infectious myocarditis,**" Dr. Bruce says that from time to

[a] Edited by Dr. John M. Keating, of Philadelphia, and published by Messrs. J. B. Lippincott and Company, of the same city.

time has been described a kind of acute change in the muscular tissue of the heart, which occurs in acute febrile and infective diseases. The opinions of pathologists as to the nature of this disease have long been, and are still, conflicting, some maintaining that it is truly inflammatory, others that it is degenerative only.

"Parenchymatous myocarditis" is the result of acute febrile and infective processes, such as scarlatina, diphtheria, variola, typhus, typhoid, and relapsing fevers, septicæmia and pyæmia, more rarely measles. The condition may be set up during the later, as well as in the earlier, stages of these diseases, or even during convalescence. In it the heart is sometimes distinctly dilated; the myocardium is of a dirty grayish red or grayish yellow colour, with occasional extravasations; its consistence is soft; its substance is lax, flabby, and friable. Thrombi may be found in the ventricles. Microscopically, the muscular fibres are swollen, their striation is more or less lost and replaced by granular (albuminous) and fatty molecules; occasionally they undergo waxy degeneration (Zenker). Along with these evidences of degeneration, there are found certain appearances which suggest *regeneration*. Lastly, the blood-vessels are dilated and the seat of thrombosis, with obliterative endarteriitis of the arterioles.

Dr. Bruce points out that the pathological connection between this acute parenchymatous change and its cause is still unsettled. It may be the result of the specific action of the several poisons, or of the pyrexia, or of both, on the protoplasm. It is closely related to fatty degeneration of the heart—indeed, if the destructive part of the process be in excess, it rapidly proceeds to fatty degeneration, which then covers, or takes the place of, the other changes.

As regards the symptoms, **cardiac failure** is the chief evidence of this condition of the myocardium.

Diagnosis.—We have to distinguish Scarlatina from Erythema, Smallpox, Measles, Rötheln, Erysipelas, Tonsillitis, Diphtheria, and Acute Rheumatism.

1. **Erythema** is distinguished by the fact of its limited distribution—being absent from the neck and extremities, and by its irregular mode of spreading. The fever is slight. There is no sore throat, or swelling of the cervical glands, or kidney affection, or desquamation.

2. It is only at the beginning of an attack of **Smallpox** that an error of diagnosis could be committed. The adventitious prodromal rash of smallpox sometimes closely resembles scarlatina, and the throat also may be sore. In arriving at a diagnosis, bear in mind the prevailing epidemic, note the pulse-rate and temperature, examine the throat and cervical glands, and watch the case closely.

3. Scarlatina is distinguished from **Measles** by the early appearance of the rash, the absence of coughing and sneezing, and the character of the fever. In measles—the early angina, the strawberry tongue, and the glandular swellings of scarlet fever are wanting.

4. The diagnosis between Scarlatina and **Rötheln** is often most difficult. We must be guided by the prevailing epidemic, the short duration of the rash, which shows on the face also, and of the fever in Rötheln, the trifling angina, and the comparatively slow pulse of that disease, in which also the kidney affection and other complications of scarlatina are wanting.

5. In **Erysipelas**, which, by the way, is often accompanied by sore throat, the rash is localised and not punctate, the surface is smooth, there is marked œdema of the connective tissue, and vesicles or bullæ may form. In scarlatina, desquamation may occur in places where there has been no antecedent eruption; in erysipelas this is never the case.

6. **Acute Tonsillitis, Cynanche tonsillaris**, or **Quinsy**, with its high temperature, swollen tonsils, œdematous uvula, and plum-coloured fauces is not infrequently confounded with scarlatina. The diagnosis depends on the history of exposure to infection or otherwise, and the absence of the vomiting, the rash, and the albuminuria of scarlatina. Generally also one tonsil is more engaged than the other in tonsillitis, whereas the scarlatinal affection is bilateral.

7. In a case, which seems to be one of primary **Diphtheria**, a careful search should be made for the ill-developed rash of scarlet fever, and regard should be had to the occurrence of other cases of the latter disease in the immediate neighbourhood of the patient.

8. **Acute Rheumatism**, although often accompanied by sore throat and by accidental papular or miliary eruptions, is distinguished by usually attacking adolescents and adults rather than

children, by its profuse acid and sour-smelling perspirations, by the absence of the strawberry tongue, glandular swellings, and renal complications of scarlatina, and by its whole course.

Prognosis.—This is **uncertain under all circumstances**, for scarlet fever is one of the most treacherous of maladies, and therefore the opinion should always be guarded no matter how mild the attack may seem to be. Mayr wrote: "*That scarlatina, even in the mildest form, is never a trifling complaint*, is a maxim which has been only too fully verified by many sad cases."[a] Sydenham's opinion as to the benign character of the disease has already been quoted—"*hoc morbi nomen*;" but Löschner some forty years ago used an equally epigrammatic and a truer phrase, when he said, "I have never seen a benign epidemic." The **mortality** very frequently reaches between 13 and 18 per cent.; but in not a few outbreaks it is as high as 25 per cent., or may reach even to 30 and 40 per cent. **Age** influences the death-rate to a great degree. It is, on the average, 20 per cent. among children under 5 years. So also does **social status**, but to a far less extent. **Family idiosyncrasy** plays an important part in determining the mortality from scarlet fever. Even in a mild epidemic, a family may here and there be decimated, the disease seeming to act as a deadly poison in certain households. As to adult cases, the mortality is highest among **pregnant** and **puerperal women** and **invalids.**

Unfavourable Signs in a case are: Hyperpyrexia, dyspnœa, extreme rapidity and feebleness of the pulse, early collapse, badly developed and dark-coloured rashes, persistent vomiting and diarrhœa, delirium or coma, sloughing of the fauces (angina gangrænosa), diphtheria, purulent arthritis, and other severe complications—especially nephritis with anuria, and diffuse cellulitis of the neck.

Death occurs, according to Hebra, from—

(1.) **Dissolution of the Blood.**

(2.) **Paralysis of the Nervous Centres** (Ataxia).

(3.) **Suffocation, from Œdema of the Glottis.**

(4.) **Pyæmia.**

(5.) **Renal Disease.**

[a] Hebra. *Diseases of the Skin.* New Syd. Soc. 1866. Vol. I., page 213.

CHAPTER XIX.

The Treatment of Scarlatina.

Prophylaxis not so difficult of attainment as in the case of Measles.—Suggested prophylaxis by drugs not reliable.—Effectual prophylaxis consists in Isolation.—Curative Treatment: no antidote yet discovered.—Biniodide of Mercury (Illingworth).—Treatment must be largely *symptomatic*.—Treatment of Scarlatina simplex: expectant.—Hebra's recommendations.—Guaiacum and Ozonic Ether Test for Blood-pigment in Urine.—Treatment of Scarlatina anginosa: cold water treatment, quinine, ice, cold compresses to neck, drugs.—Treatment of Scarlatina maligna: combat ataxic symptoms; in hæmorrhage, use local and general astringents.—Treatment of Complications and Sequelæ: Diphtheria, rheumatism, acute desquamative nephritis, uræmic convulsions, pleuritis, endocarditis, bubonic swellings, diffuse cellulitis, pyæmia, acute furuncular diathesis, diseases of the ear, conjunctivitis, keratitis, acute eczema, chorea.

I. The **Prophylaxis** of Scarlet Fever is not attended with the vast difficulties which beset the attempt to control the spread of Measles. Although it would be going too far to deny that scarlatina is infectious in the stages of invasion and of early eruption, yet there can be little doubt that the poisonous virus is shed in greater quantity during the later stages of the disease, in the discharges from the nose and throat, in the motions from the bowels, most likely in the urine, but above all, in the desquamated cuticle. Hence, breathing time is allowed the physician in which to plan and give effect to precautionary measures.

Again, the early appearance of the rash and the sore throat establish the diagnosis long before the period when measles declares itself—that is, on the fourth or fifth day. Time, therefore, is not permitted to the malady in which to run riot through a family or household.

In speaking of the Preventive Treatment of Scarlatina, I may state at the outset that **all known drugs are without avail.** Godelle vaunted hydrochloric acid as a prophylactic; Giersing suggested the internal administration of carbolic acid; Hufeland

and Hahnemann ascribed to belladonna a protective influence against scarlatina, and I have often known this drug to be given for such a purpose by orthodox physicians. But all in vain. Dr. W. G. Walford, of London, states that out of nearly 100 children exposed to infection and to whom full doses of liquor arsenicalis were given, in only two did the disease develop. Children bear arsenic well. Walford gives from 1 to 3 or 4 minims of Fowler's solution, according to the child's age, thrice daily. He combines with the dose 15 to 30 minims of sulphurous acid and a little syrup of red poppy (*syrupus rhœados*, B. P.)[a]

Notwithstanding these statements I am of opinion, with Mayr and Hebra, that we must conclude that "**the only effectual prophylaxis of scarlatina consists in isolating the patients** from those who are unaffected as early and as completely as possible." This isolation should be kept up until desquamation has finally ceased. Reference may be made to Chapter IV. for the details which should be followed in carrying isolation, disinfection, and other preventive measures into effect.

A scarlatina patient may go home or rejoin school, provided he and his clothes have been thoroughly disinfected, *in not less than six weeks from the appearance of the rash*, if desquamation has completely ceased, and there is no complaint or sign of sore throat or of discharge from the nose or ears.

II. So far, we have not succeeded in discovering an antidote for the virus of scarlatina—we have **no specific remedy** for it. The biniodide of mercury, if given early in frequent doses, is stated by Dr. C. R. Illingworth,[b] of Accrington, to shorten the disease and relieve the throat affection. He advises that a child aged between 2 and 6 years should be given every second hour 10 minims of solution of the perchloride of mercury with half a grain of iodide of potassium in a drachm of water. Dr. Eustace Smith recommends the addition to this mixture of small doses—say, half a grain—of chlorate of potassium.

The term "**Curative Treatment**" must, however, be taken in a

[a] *Lancet*, 1882. And *Brit. Med. Journ.*, 1884.
[b] *Brit. Med. Journ.*, 1886.

qualified and restricted sense to express the measures we adopt to help the patient safely through his illness. The treatment must, in a word, be **largely symptomatic**, and directed mainly against those complications and sequelæ which disturb the regular progress of the disease.

In mild **scarlatina simplex** we may adopt an expectant treatment more or less like that recommended by Hebra.

1. The patient should keep his bed throughout his illness in a fresh, airy room, with just as much covering over him as will prevent him feeling cold. Feather bed, coverlets, and movable screens, should all be avoided.

2. The patient should not be allowed to leave his bed until, for two or three days there has been complete absence of fever, thirst has disappeared, the skin has been soft and perspiring, and the pulse has been quiet. On getting up, a flannel or Jaeger suit should be worn.

3. About the end of the third week the patient may be ordered to take a tepid bath daily, or every second or third day, according to circumstances.

4. As soon as desquamation has ceased on the hands and feet, as well as on the face and body—that is, in the fourth week, the patient may be allowed to go out into the open air—weather permitting, and unless any fresh symptoms should arise to prevent this.

5. Cool, refreshing drinks, such as cold spring water, lemonade, acidulated water, are to be given freely, and at short intervals.

6. The diet should consist of weak meat-broth, chicken-broth, gruel, milk (peptonised, or mixed with aerated water or lime water in varying quantity), oranges, and cooked fruits.

7. The patient's hair may be combed every day; his face and hands should be washed with soap and water. The bed and body linen may be changed as often as required. Tepid sponging of the whole body is useful and most refreshing. It may be practised once or twice a day.

8. The urine should be examined daily. With a view of anticipating the danger of acute scarlatinal nephritis, it will be well daily to use the test for hæmoglobin suggested by the Collective

Investigation Committee of the British Medical Association in their Report on Paroxysmal Hæmoglobinuria[a]—that is, the guaiacum and ozonic ether test. With this the urine gives the blue tint characteristic of blood-colouring matter, when hæmoglobin is present even without blood corpuscles.

This test is best performed by adding to about a drachm of urine in a test-tube a few drops of fresh tincture of guaiacum and twenty or thirty drops of ozonic ether. The whole is to be well shaken and then allowed to settle, when the ozonic ether rises to the top, holding in solution the red colouring matter of the guaiacum tincture, of—if blood or its colouring matter be present—the blue pigment to which this has been transformed. In applying the test, instead of using ozonic ether, the same quantity of old turpentine will answer equally well. The best plan also is to make a fresh solution of a few grains of guaiacum resin in a little methylated spirit before each experiment. This test suffices, when a spectroscopic examination cannot conveniently be made, to establish the diagnosis of hæmoglobinuria, even before renal hæmorrhage (hæmaturia) has taken place.

The hot air or vapour bath may be used if albuminuria is present, and during desquamation the tepid bath will be found both grateful and beneficial. It may be given daily and followed by dry rubbing and **inunction** with oil (Dähne, 1810), fat bacon, or suet (Schneemann, of Hanover, and Charles West, of London). In my own practice, I employ a weak carbolised oil (1 to 2 per cent.), or soft paraffin (vaseline), or camphorated oil (linimentum camphoræ, B.P.). Louis Starr[b] recommends that the whole surface, including the scalp, should be anointed daily during desquamation with an ointment consisting of carbolic acid, 20 grains, thymol 10 grains, vaseline, or simple ointment, 1 ounce. The patient, after the anointing, should be put into a warm bath for five minutes, protected from cold, and then removed to bed, the body being wiped dry beneath the bedclothes.

I am aware that the practice of inunction has been objected to

[a] See *Brit. Med Journal*, Jan. 26, 1884. P. 189.

[b] *Archives of Pediatrics.* July, 1890. Philadelphia.

on the ground that it interferes with the free action of the skin, but this objection is merely theoretical. I am satisfied that to keep the skin smooth, moist, and pliable in scarlatina is essential, and this indication is met by inunction or anointing. The other advantages of the practice are—as regards the patient, relief from itching and general discomfort, reduction of surface temperature, protection from cold, lessened risk of kidney congestion owing to the free action of the skin. From a prophylactic point of view, the *fixing* of the infectious scales of the epidermis by inunction is a consideration of the first importance.

9. Scarlatina is a great **blood-destroyer**, and in convalescence iron will be required, as well as change of air to the mountain slope or to the seaside.

In **Scarlatina anginosa** additional treatment to the foregoing will be required. With the object of **reducing the excessive fever** (hyperpyrexia) which so frequently accompanies this variety of scarlet fever, the **cold-water treatment** described in Chapter V. (see pages 53, *et seq.*) should be carried out. **Quinine** also may be given freely with a twofold object—first, as an efficient antipyretic, secondly as an equally useful antiseptic. There is scarcely any form of "sore-throat" over which quinine does not exercise a more or less specific action. Again, the constant **swallowing** of fragments of **ice** is both grateful to the patient and effectual. **Cold compresses** of lint moistened with water and glycerine should be wrapped round the throat. The nostrils, mouth, and pharynx should be frequently washed or sprayed with chlorine water or warm water containing some common salt, or chlorate of potassium, or permanganate of potassium (15 grains to the ounce), or sulphurous acid (1 in 8) or carbolic acid and glycerine. In the case of the mouth and pharynx, these remedies may also be used in the form of a disinfecting, antiseptic, and deodorising gargle. The following formula for a guaiacum gargle is copied from the *Pharmaceutical Record*, January 5, 1891:—

℞. Tinct. Guaiaci Ammoniati;
Tinct. Cinchonæ Compositæ, āā ℥ss;
Potassii Chloratis, gr. 60;

Mellis Purissimi, ℥ss ;
Pulv. Gummi Acaciæ, gr. 60 ;
Aquæ, ℥iiss.

M. ft. gargarisma. *Signa :* To be used as a gargle, and a teaspoonful may be swallowed every second hour.

Caustics of all kinds, in my opinion, had best be avoided in scarlatinal sore-throat. It is, however, right to mention that Trousseau recommends cauterisation with equal parts of strong hydrochloric acid and honey. He, no doubt, advises caution in the use of the remedy, which—if awkwardly applied—might cause spasm of the glottis.

Among drugs other than quinine which are likely to be of use in this serious affection, the ferric chloride, sulpho-carbolates, hypo-sulphite of sodium, guaiacum, and salicylate of sodium may particularly be noticed. The perchloride of iron may be given with advantage in combination with chlorate of potassium and quinine. In America, a combination of boric acid and iron is, according to Fenwick, a favourite remedy. He gives this formula :—Boric acid, 30 grains; chlorate of potassium, 120 grains; tincture of ferric chloride, 2 drachms ; glycerine, one ounce ; syrup, one ounce ; water, 2 ounces. One teaspoonful every second hour for a child of five years. Lastly, a moderate—sometimes, a free—allowance of **stimulants** is necessary.

The treatment of **scarlatina maligna** is too often of no avail—the patient dying poisoned in a few hours or days. It is our duty, however, **to combat ataxic symptoms** by the free administration of **food and stimulants** and by such remedies as carbonate of ammonia, quinine, bark, iron, camphor, and musk. "**Derivation to the surface**"—more particularly if the rash fails to appear or is badly developed—may prove of use. Wrapping the lower limbs and body in flannels or cloths wrung out of mustard and hot water is often effectual, and I have myself practised the method with success. Wunderlich recommends the warm, or hot, bath. Thomas speaks highly of the cold pack and cold affusion followed by warm wrappings. Max. Langenbeck has adopted with good results the use of the hot flat-iron, combined with a mustard bath and subse-

quent warm wrappings. Blisters have been applied, but to my mind this is a doubtful procedure, regard being had to the usual tender age of the patients and to the kidney delicacy which so often accompanies the disease. Sloughing too would very likely follow the application of a blister.

In the **hæmorrhagic variety** of scarlatina maligna we must resort to **general and local astringents**, such as ferric chloride in full doses, gallic or tannic acid, ergot, hamamelis (or hazeline), assisted by a nutritious diet and port wine. Owing to kidney delicacy, turpentine is usually contraindicated in scarlet fever. **Ice**, in every form of application or use, is an invaluable remedy.

We have now to consider the **treatment of the complications** and **sequelæ** mentioned in Chapter XVII.

1. **Diphtheria.**—The awful prostration which usually accompanies this affection must be combated by food, wine, diffusible stimulants, bark, and quinine or iron. Young children, as a rule, take quinine very well if it is mixed with milk, and not dissolved in an acid medium, as it is too much the fashion to prescribe it. When there is a diphtheritic exudation, a spray should be used containing glycerine of carbolic acid (1 in 8), or lime water—the solvent power of which is very marked, or solution of mercuric chloride, or lactic acid (the strong acid, 1 drachm; glycerine, 1 drachm; water, 14 drachms), or papain (1 to 2 parts in 10 each of glycerine and water). Fenwick gives this formula: Carbolic acid, 1 drachm, or boric acid, 3 drachms; "liquor potassæ," 1 drachm; chlorate of potassium, 2 drachms; glycerine, 2 ounces; lime water, 8 ounces—to be used as a spray, "when the exudation is foul, jagged, and of a dirty brown appearance." At the London Hospital, a solution of sulphurous acid (one part to two or three of water) is frequently used as a spray. Powders, too, may be employed with an insufflator, such as salicylic acid, 2 drachms; subnitrate of bismuth, 2 ounces (Fenwick). Oertel, the author of the monograph on "Diphtheria" in von Ziemssen's "Cyclopædia of Practical Medicine," highly recommends the constant inhalation of steam at 113° to 122° F., passed into the mouth through a funnel.

2. The **rheumatic affections** of scarlatina are best treated like ordinary acute rheumatism ("rheumatic fever"). The affected joints should be packed in wadding, or cotton wool, and bandaged; while salicylic acid, salicin, salicylate of sodium, or salol should be given internally in large and frequently repeated doses. Should peri- or endo-carditis occur, much relief will be obtained by the application of a small blister over the epigastrium or præcordial region, as recommended by Dr. Alex. Harkin, of Belfast.

3. **Acute Desquamative Nephritis.**—Dr. Austin Flint lays down the following indications for treatment in acute nephritis, including, of course, the condition met with in scarlet fever—(1.) Diminution of the intensity of the renal inflammation, promotion of resolution, and restoration of the excretory function of the kidneys; (2.) Diminution or removal of dropsical effusion; (3.) Elimination of urea through the skin and gastro-intestinal mucous membrane, if uræmia exists or is threatened.

These indications will be met by adopting the line of treatment here sketched out.

The patient should remain in bed, wrapped in a blanket, warm but not overwhelmed with bed-clothes; clad in a long flannel night-dress from head to foot. He should be placed on a mild unstimulating diet of milk, skimmed or in the form of buttermilk, farinaceous food, and light broths (veal, mutton, chicken, in moderation), all highly nitrogenous foods, such as eggs, butcher's meat, and strong beef tea being avoided. His thirst should be relieved by copious draughts of cold water or of one of the mineral effervescing waters which will safely increase pressure in the glomeruli, so that an augmented transudation may wash away coagula from the tubules of the kidney. Dry cupping, followed by poulticing over the kidneys, will almost certainly do good, and if the patient is robust and at the same time in much pain, local depletion by leeching or wet cupping over the loins may be practised with benefit. Even venesection may be practised in a fairly strong subject (Thomas and Romberg).

The bowels should be kept open by hydragogue cathartics, like jalap, bitartrate of potassium, 30 grains, with a drachm of honey

or treacle, scammony, elaterin (?), senna, and colocynth—more particularly if dropsical symptoms have supervened.

All stimulating diuretics must, however, be shunned, for they only increase renal hyperæmia. In this stage, much benefit will be derived from hot air, vapour, or even warm water baths. The hot air bath should not be prolonged beyond twenty minutes. It may easily be improvised, as suggested by Dr. Saundby, by setting a spirit lamp carefully under a stool or cradle in the bed, if one of the cheap tin lamps sold for the purpose is not available.

The "wet-pack" already described may also be used with advantage to promote elimination through the skin. If **uræmic convulsions** supervene, a full dose of sulphate of magnesium (Epsom salts) well diluted, or a bolus of calomel and jalap, may be administered, although mercurials should, as a rule, be avoided in this affection—certain it is, that a patient in nephritis is very susceptible to the influence of mercury, and even one dose of calomel may cause salivation. For the relief of **convulsions**, also, the temples may be dry-cupped or leeched, and Trousseau's plan of compressing the common carotid artery in the neck on the side opposite to that affected by the convulsions may be tried. Compression may, indeed, be practised during fifteen minutes on each side of the neck alternately. Other means of relief are: a combination of chloral hydrate with one or other of the bromides, or inhalations of chloroform; or the nitrites—trinitrin (*nitro-glycerine*), nitrite of sodium, nitrite of amyl; or citrate of caffein or theobromo-sodio-salicylate (Diuretin); or an enema of black coffee (*café noir*). To **prevent uræmia** Frerichs recommends benzoic acid in doses of from three to fifteen grains, or more, according to age, either in pill or as a soluble benzoate. Four grains may be made up into a pill with a minim of glycerine or a grain of Canada balsam; or ten grains of the benzoate of sodium, with ten minims of tincture of digitalis may be given in an ounce of infusion of gentian to an adult thrice a day (Saundby).

Pilocarpin ($\frac{1}{40}$ gr. to $\frac{1}{20}$ gr. every six hours, or $\frac{1}{20}$ gr. subcutaneously, for a child five years old) is mentioned by Dr.

Fenwick, as recommended in American practice. It has been used in this country also for many years, but requires great caution in its administration.

In convalescence the patient should wear woollen clothing day and night, and in all seasons. The "Jaeger system" is both popular and efficient. In this stage of the complaint iron and quinine are the most useful drugs, and albuminous food should be given once all evidence of kidney inflammation has passed away.

4. For the relief of **pleuritis** and other **serous inflammations**, the thorough application of cold is recommended by Thomas, to prevent excessive exudation. Should effusion proceed rapidly in pleuritis, paracentesis thoracis (thoracentesis) must be practised. **Endocarditis** is best controlled by absolute rest, the continuous application of cold to the præcordial region, and digitalis, convallaria, or strophanthus with nux vomica internally.

5. **Bubonic swellings** may be treated by diligent poulticing and painting with iodised glycerine. In the later stages, chloride of calcium, cod liver oil, iodide of iron, and saccharated solution of lime, are all well worth a trial. In the **diffuse cellulitis of the neck**, Mr. Croly, in the paper referred to already, strongly advocates early, deep, and free incisions, as recommended by the late Mr. W. H. Porter, Surgeon to the Meath Hospital. Only in young children is this practice open to question.

6. The treatment of **pyæmia**, and of the "**acute furuncular diathesis**," of which Trousseau speaks in the case of smallpox, has already been discussed when speaking of that disease.

7. The **diseases of the ear** in scarlatina require early and skilled attention, if their dangerous results are to be avoided. Wendt advises that both as a preventive and as a curative measure the secretions should be removed from the nostrils, posterior nares, pharynx, throat, and even from the proximal portion of the Eustachian tubes by douches and antiseptic or detergent gargles. The external auditory meatus should be kept clean and free by gentle syringing. Air should be forced into the middle ear from time to time, of course *not* by means of the catheter. If intense pain in the ear is complained of, leeching—even the application of

a single leech sometimes—gives great relief, followed by poulticing. The bathing treatment of severe scarlatina need not be interrupted, provided the auditory meatus is plugged with oiled cotton during the bath.

8. **Conjunctivitis** is relieved easily and effectually by water-dressings; **keratitis**, by the application of cold and dilatationof the pupil by atropin. If the cornea threatens to slough, it should be punctured by a skilled hand.

9. An ointment of salicylic acid (10 grains to the ounce) often acts almost specifically in the **acute eczema** of children, such as follows scarlet fever. Or the paste recommended by Dr. Charles Szadek may be used, namely, salicylic acid, 30 grains; vaseline, half an ounce; oxide of zinc and pure starch, of each 360 grains.

10. In **chorea**, tonics such as the scale preparations of iron, quinine, valerianate of zinc, and so on, together with change of air and scene, will prove beneficial.

Note.—The most recent contribution to the literature of the Bacteriology of Scarlatina is a communication by Dr. H. Kurth, Königl. preuss. Stabsarzt, entitled:—"Ueber die Unterscheidung der Streptokokken und über das Vorkommen derselben, insbesondere des Streptococcus conglomeratus, bei Scharlach." ("On the differential diagnosis of the Streptococci, and on their occurrence, especially that of the *Streptococcus conglomeratus*, in Scarlatina"). Dr. Kurth's monograph was published in the seventh volume, second part, of the "Arbeiten aus dem Kaiserlichen Gesundheitsamte," Berlin, 1891.

CHAPTER XX.

Rötheln, or Epidemic Rose Rash.

Nomenclature.—Definition.—Ætiology (historical sketch).—Clinical History: Incubation, invasion, eruption, desquamation.—Temperature.—Complications and Sequelæ.—Pathology.—Diagnosis.—Claims of Rötheln to be considered a distinct disease.—Prognosis: entirely favourable.—Treatment.

Nomenclature.—Rötheln. *Synon.*—Rubeola (Orlow, 1758;[a] Hildenbrand, and even Thomas of Leipzig[b]); Rubeola notha (other German writers); Rubeola sine catarrho; Rubella (a diminutive of "Rubeola"); Roseola Epidemica. *English*, German Measles, Epidemic Rose Rash, Epidemic Roseola, Bastard Measles. *Fr.* Roséole (Trousseau); Exanthème fugace (synonym mentioned also by Trousseau). *Germ.* Rötheln (from *roth*, red). Borsieri described the disease under the name *Essera Vogelii* (Trousseau).

Definition.—A specific and infectious eruptive fever, **distinct and separate**, of its own kind (*sui generis*); **neither a hybrid** of scarlet fever and measles, **nor a modified form** of one or other of those diseases—**Rötheln breeds true.** The disease begins suddenly after an incubation of about twelve days, with ordinary febrile symptoms of moderate intensity. The rash appears on the first or second day. There are slight catarrhal and anginal symptoms. Enlargement and induration of the lymphatic glands in the occipital and cervical regions is a constant epiphenomenon. The febrile movement is brief, and recovery is generally uninterrupted and complete.

Ætiology.—The assumption of a specific roseola is based chiefly on the fact that at certain times epidemics appear in which individual cases bear a causal relation to one another. Further,

[a] *De Rubeolæ et Morbillorum Discrim. Progr.* Königsb. 1758.

[b] Von Ziemssen's *Cyclopædia of the Practice of Medicine.* Art. Rubeola. Vol. I., p. 199. 1875.

this disease affords no protection against either measles or scarlatina, whereas it does protect an individual from a second attack of itself. Lastly, it may attack those who have lately or previously passed through either measles or scarlatina.

It is especially a disease of childhood, but may occur in adults up to the age of 40. Seitz reports a case in a woman aged 73.

There is reason to believe that epidemics of Rötheln occur periodically like epidemics of measles. In his account of the disease, Thomas mentions two such epidemics as having prevailed at Leipzig in 1868 and 1874. In 1874, also, an epidemic was observed in New York by Dr. J. Lewis Smith. The first British writer on the disease was Dr. Robert Paterson, of Leith, and he adopted the German term Rötheln, which seems to be the most satisfactory name, for it precludes any possibility of confusion in nomenclature. Sir William Aitken draws a distinction between *Roseola* and *Rubeola* or *Rötheln*, for his description of the latter impresses the reader with its severity compared with the trivial nature of the former. Trousseau,[a] however, makes no such distinction between *Roseola* and *Rubeola*, nor, indeed, does Wunderlich, who, in common with German writers generally, employs *Rubeola*, *Rubeola notha*, and Rötheln, as synonymous terms.

It is curious to compare Sir William Aitkens' description of *Rubeola* with those given by the two authors named. He says:[b] "The prognosis requires to be as guarded as in scarlatina; for, like scarlatina, *rubeola is often an extremely and rapidly fatal disorder.*" Trousseau writes: "De toutes les fièvres éruptives la roséole est la plus bénigne: *jamais elle ne présente de gravité*, et toujours elle se termina spontanément sans que le médecin ait en aucune façon besoin d'intervenir." Wunderlich's translator, Dr. Bathurst Woodman, renders that author's opinion as follows: "Rubeola (Rubeola notha, Rötheln, or the so-called hybrid between measles and scarlatina, sometimes called Roseola also) does not necessarily entail any fever at all, and only a slight transient attack before and during the eruption.

[a] *Clinique Médicale de l'Hôtel Dieu de Paris.* Deuxième Edition. 1865. Tome I., page 151.

[b] *The Science and Practice of Medicine.* Third Edition. Vol. I., page 351.

The elevations of temperature are generally subfebrile, or *at the worst moderately febrile.*"

Dr. Hilton Fagge points out, in his "Principles and Practice of Medicine," that a precisely similar difference of opinion as to the severity of Rötheln is shown to exist when one compares the portrait of the disease drawn by Paterson, Copland, and other writers of 40 or 50 years ago, and the account given of it by Thomas, of Leipzig, in von Ziemssen's "Cyclopædia of the Practice of Medicine." The result of this difference of opinion was to shake Hilton Fagge's belief in the separate identity of Rötheln. Trousseau, however, puts the matter fairly and well when he asserts that **Rötheln bears the same relation to measles and scarlatina that varicella, or chickenpox, does to smallpox.** This is equivalent to saying that it is **a totally distinct disease.** In a Clinical Lecture on Rötheln, delivered at the Middlesex Hospital in 1874, Dr. Robert Liveing, no mean authority on the subject, condemns the synonyms "hybrid measles," or "hybrid scarlatina" assigned to this exanthematic fever as "most objectionable, inasmuch as they give colour to the erroneous notion that the disease is a combination of measles with scarlatina."[a]

Clinical History:—

I. Stage of Incubation—To the *Irish Hospital Gazette* for June 15, 1874, I communicated a series of three cases of Rötheln which had come under my notice within the previous year or so. Of these the first two occurred in a brother and sister, the latter of whom sickened 12 days after her brother. Dr. Murchison, speaking of a case, says:[b] "About ten or fourteen days subsequently, a second child in the same family had a similar attack." In the New York outbreak of 1874, reported by Dr. J. Lewis Smith, the incubative period did not seem to be uniform. In some instances it appeared to be from seven to ten days, and in others from eighteen to twenty-two days. Thomas assigns to this stage a duration of from $2\frac{1}{2}$ to 3 weeks.

[a] *The Lancet.* March 14, 1874. Page 360.
[b] *The Lancet.* October 29, 1870. Page 595.

II. **Stage of Invasion.**—The premonitory symptoms are sometimes absent—or very badly marked. In a considerable number of the cases in New York in 1874, the patients were not known to be sick until the rash was observed covering the surface. Usually, however, the initial phenomena of a feverish attack are fairly pronounced—the patients are dull and languid, and complain of headache, chilliness, general discomfort, loss of appetite, thirst, pains in the limbs, vomiting and diarrhœa. The throat is injected, but the coryzal symptoms of measles are absent or are only slightly developed. In exceptional cases convulsions may occur. Dr. Lewis Smith met with clonic convulsions in a boy of 8 years. Nausea is a common symptom.

III. **Stage of Eruption.**—In a few hours or on the second day the rash appears in different parts of the body, thence extending to the legs the next day. It may show itself first upon the face and neck, or down the back, or over the chest. Most frequently it resembles the rash of measles; sometimes it is scarlatiniform in appearance, although Thomas denies this. The compound or hybrid eruption has been observed by Dr. Küttner, of Dresden.[a] Lewis Smith says that the rash in the New York epidemic resembled more that of measles than of any other eruptive fever, but in one case, a boy of three and a half years, it presented over the trunk very much the scarlatinous appearance. Dr. Liveing also describes the rash as consisting "of small rounded collections of minute red papules, which after a time coalesce and form larger irregular patches, just as in measles, but with apparently less tendency to become of a horse-shoe or crescentic shape. After a time the patches may all unite, and then the skin becomes to the naked eye of a uniform red colour, closely resembling that in scarlet fever." It will be observed that Liveing speaks of "**papules**," and the appearance in one of my cases quite bears out this description and Sir William Aitken's remark, that "the eruptive patches are felt to be distinctly elevated above the skin, some more than others, and always greatest in the centre of the patch." L. Smith says the rash "disappeared on pressure, caused a *little roughness*, as ascertained by carrying the

[a] Cf. *Dublin Hospital Gazette.* Dec. 15th, 1858.

fingers over the surface, and faded without desquamation." The slightly raised rose-coloured spots or maculæ of Rötheln vary in size from a mere point to one-sixth of an inch in diameter.

On the other hand Trousseau states that the patches of roseola do not project above the surface of the skin like those of measles—"Les taches rubéoliques ne sont plus en effet saillantes comme le sont les taches morbilleuses."[a] All writers are agreed that troublesome **itching** accompanies the rash, which fades gradually, sometimes (as in one of my cases) leaving dark and dirty, or yellowish stains like those in measles, which are visible for five, six, or more days.

Simultaneously with the dermatitis, there is a **mild inflammation of the mucous membranes** covering the buccal, pharyngeal and nasal surfaces, and of the conjunctivæ. This gives rise to a certain degree of sore-throat, sneezing, running from the nose, suffused, watery, or reddish eyes, with slight œdema of the lids, and often a muco-purulent secretion which, drying, glues them together, especially in the mornings.

Lastly, allusion must be made to an almost pathognomonic sign of Rötheln, and this is **enlargement and induration of the lymph-glands**, particularly those on the mastoid processes and behind the sterno-mastoid muscles, and those of the posterior chain below the occipital protuberance. Analogy would lead us to infer that the bronchial glands share in this pathological change—a point which has a material bearing, as I hope to show, on the prognosis.

IV. **Stage of Desquamation.**—There is but little "**peeling**" in Rötheln. Lewis Smith says the rash "faded without desquamation." In one of my cases there was "slight desquamation on the bridge and at the sides of the nose;" in another case, the skin was shed in large flakes as in scarlatina, and even the nails came off.

Temperature.—The **febrile movement** is usually **slight.** Wunderlich's opinion on this point has been quoted above.[b] Dr. Edward Ellis says: "The temperature in my own experience, has been remarkably low, rarely reaching and never exceeding 100° F.;

[a] *Clinique Médicale.* Tome I. 1865.

[b] *A Practical Manual of the Diseases of Children.* Second Edition. Page 73.

sometimes ranging from 97° F. to 99° F." To the same effect, Dr. Lewis Smith observes: "The febrile movement was ordinarily mild, the pulse in ten uncomplicated cases ranging from 80 to 100, and the temperature from 98½° to 100°."

In my own three cases the maximal temperatures noted were 102·4°, 98·2°, and 99 8° respectively.

Complications and Sequelæ may almost be said not to exist in Rötheln. In one instance, notwithstanding, I could not help regarding an attack of this disease as the starting point in a young barrister of chest delicacy, which nearly cost him his life, through profuse and repeated hæmoptyses. Happily, after some years of invalid life he completely recovered. The occipital glands were much enlarged during this gentleman's attack of Rötheln, and in the light of his subsequent history the conviction was forced upon me that a similar enlargement of the bronchial glands had been followed by caseation and even tubercular disease.

At all events, *the possibility* that Rötheln may be the starting point of phthisis pulmonalis can never in future be absent from my mind when called upon to attend a case of this disease.

Pathology.—This topic has been sufficiently discussed in the preceding remarks on the ætiology and clinical history of the affection. There is, in fact, little that is specific in the Morbid Anatomy of Rötheln.

Diagnosis.—Writing in 1874, Dr. Liveing[a] observes:—"German measles is not yet fully recognised by the profession in this country; little or no account is given of it in our ordinary text-books on medicine, and its name does not find a place in the 'Nomenclature of Diseases,' drawn up by a Committee of the Royal College of Physicians. Under these circumstances, it is not surprising that errors of diagnosis should sometimes bring discredit on our profession." In a Clinical Lecture on Rötheln, also delivered at the Middlesex Hospital, in April, 1870, the late Dr. Charles Murchison spoke in the same strain.[b] After describing two cases, he remarked: "The ailment from which these patients suffered is not generally

[a] *The Lancet*, March 14, 1874. Page 360.
[b] *The Lancet*, October 29, 1870. Page 595.

recognised as a distinct disease; and cases of it, when they occur, are apt to be puzzling, and sometimes to get the medical attendant into trouble from his inability to determine their real nature. Yet, on the whole, they are not very rare."

The following cases will exemplify the difficulties which arise in connection with the diagnosis of Rötheln. They are culled from the note-books of a physician of much skill and experience:—

A lad, aged seven, showed the rash of measles on June 18, 1852. A second child, a girl, aged five and a-half, had the rash on June 29; a third, a girl, aged two, on June 30. The mother of these three children told the physician that the boy had a year before an attack of what was thought to be "spurious measles," the rash more vivid than on the present occasion, with sore throat and followed by desquamation of cuticle. He did not appear sick, was not confined to bed, and did not communicate the disease to any of the other children.

On November 18, 1851, the same physician vaccinated a baby girl, aged three months. On the fifth day, the eyes watering, an eruption of "roseola" came out and spread over the whole body. It was very evident on the scalp and trunk, was attended with a good deal of restlessness, and a disinclination to take the breast. It was at its height on the seventh day, and nearly gone on the eighth day after vaccination, in no way retarding the progress of the vesicle.

On the 13th of the following month an eruption like measles came out on the baby's sister, aged nearly five years. She passed through her attack with very little constitutional disturbance and without much cough or catarrh.

On July 20, 1854, the rash of genuine measles came out on the aforesaid baby, then aged four years. She had taken the disease from her elder brother, aged eight years and nine months, who, after three or four days of "off and on" feverishness, showed well-marked catarrhal symptoms and the rash of measles on July 8, 1854. On the 28th the measles eruption came out on the sister, who had been infected with Rötheln in 1851, and who was now seven and a half years of age.

In the three cases I detailed in the *Irish Hospital Gazette*, the chief points of diagnostic interest were—

(1.) The trifling degree of pyrexia observed.

(2.) The early appearance of the eruption—always within 48 hours.

(3.) The presence of only slight hyperæmia of the fauces without acute inflammation or ulceration.

(4.) The evidence afforded by the second case of the series as to the infectious nature of this exanthem and as to the length of its period of incubation.

(5.) The changeable character of the eruption.

Notwithstanding the formidable account of this disease presented to us by Sir William Aitken, the balance of opinion seems to be in favour of its comparatively trivial nature. Rötheln, indeed, may be said to derive its chief importance from so often and sometimes so closely resembling measles or scarlatina.

The **arguments** which may be advanced in support of **the claim of Rötheln to be considered a distinct disease** are these:—

1. It occurs in persons who may already have had either measles or scarlatina, or both these fevers.

2. It does not protect from an attack of either of these diseases.

3. It does protect from itself, just as measles protects from measles, smallpox from smallpox, scarlatina from scarlatina. Trousseau, indeed, does not hold this view, for he says: "Une roséole antécédente ne préserve pas de nouvelles attaques." Borsieri went still further when he wrote: "*Qui semel iis laboravit facile iterum pluriesque prebenditur.*" "He who has once suffered from it, is easily attacked by it again and again."

4 It can propagate itself—in the words of Dr. R. Liveing—"the seed, as a gardener would say, comes up true."

5. The subfebrile temperature of the early stage at once tends to preclude the possibility of scarlatina, and the probability of measles.

6. The early appearance of the rash distinguishes it from that of measles, while its usually "measly" character aids the diagnosis from scarlatina.

7. The anomalous combination of the coryzal symptoms of measles with the sore-throat of mild scarlatina, should excite suspicion as to the probable presence of Rötheln.

8. The whole subsequent course of the disease and of its eruption affords grounds for a differential diagnosis.

From ordinary **Rose-rash** or **Roseola**, and indeed from **Erythema** also, Rötheln is distinguished by its pyrexia, enlarged glands, and throat symptoms.

Prognosis.—From the close resemblance of the initial symptoms and eruptions, sometimes to those of measles, sometimes to those of scarlatina, the diagnosis of Rötheln is often, as we have seen, a matter of considerable difficulty. Under such circumstances, the early prognosis should always be guarded. It is far better to have been mistaken in our diagnosis in assuming, for the time being, that we had to deal with either measles or scarlatina, than to have imperilled the lives of many children by neglecting to isolate the sick from the healthy in due time.

Once the diagnosis is made, we may, **in most cases**, pronounce **an entirely favourable opinion.** Let us bear in mind at the same time that even this "lightest of the acute exanthems"—as Thomas calls it—may occasion grave disturbances or even death in a delicate subject, and that the enlargement of the lymph glands which so constantly accompanies it may have far-reaching and untoward effects upon the patient's health and constitution.

Treatment.—A case of Rötheln should be treated exactly as if it was one of simple measles. The child should be kept in bed while fever lasts, protected against cold, and suitably fed. Tepid or cool sponging will relieve itching. Watch should be kept upon the catarrh of the pharynx and of the air-passages. Warm baths are most useful in convalescence. In a word, good nursing and sound common sense alone are wanting to tide a patient over an attack.

CHAPTER XXI.

Erysipelas.

Nomenclature.—Definition.—Ætiology.—Erysipelas both a local and a specific disease.—Medical or Idiopathic and Surgical or Traumatic Erysipelas.—Predisposing Causes: traumatism, a previous attack, sex, age, climate, season.—Exciting Causes: contagion, inoculation. Doctrine of the Contagiousness of Erysipelas not proved.—Bacteriology: *Streptococcus Erysipelatis* (Fehleisen).

Nomenclature.—Erysipelas (*Greek*—ἐρυσίπελας, Hippocrates, B.C. 430). *Synon.*—St. Anthony's Fire (*Lat.* Ignis Sacer). In Scotland it is called the "Rose." *Germ.* Rothlauf, Rose, Hautrose, Erysipelas. *Fr.* Erysipèle, Feu Sacre, Feu de Saint Antoine, Mal de Saint Antoine. *Ital.* Risipola, Erisipela. *Span.* Erisipela. *Dutch*, Roos, St. Antonie's Vuur. *Swedish* and *Norwegian* or *Danish*, Rosen.

The **Etymology** of the word "Erysipelas" is not settled. In the the *Vet. Med.* and *Aphorisms* of Hippocrates the word occurs in its Greek dress ἐρυσίπελας. Liddell and Scott derive it from ἐρυθρός, red, and πέλλα, skin. Billroth substitutes πέλας, "a tumour" or "swelling," for πέλλα; and πελός (*pallidus*) is also erroneously suggested as the root of the second portion of the word. Another fanciful derivation is from ἐρύω, *I draw*, πέλας, *near*.

Definition.—An acute, specific febrile disease, of varying course and uncertain duration, undoubtedly contagious or infectious, characterised by a polytypical range of temperature; but, above all, by a special form of dermatitis, with sharpely-defined redness and swelling of the skin. This spreads indefinitely by the lymphatics from its starting point in a breach of surface, however slight, in the skin or mucous membrane, through which the specific poison, virus, or contagium of the disease, gains an entrance into the system from without (**Inoculation**).

Ætiology.—Erysipelas is certainly to be regarded as **a specific disease,** because it depends upon the entrance into the system of a specific micro-organism—the *Streptococcus erysipelatis* of Fehleisen. In one sense, it is a **local disease,** inasmuch as the virus settles upon the spot which is afterwards to become the focus of the erysipelatous inflammation—the infection of the blood, and therefore of the whole system, being secondary. In this respect erysipelas is precisely analogous to diphtheria. A strong argument in favour of this view is afforded by the complete want of symmetry in the distribution of the skin affection which is so constantly observed. With equal propriety, however, erysipelas is classed with **general diseases of specific origin,** for like them it requires a certain incubative period for the development of its constitutional symptoms.

Dr. J. J. Pringle[a] well observes that the term erysipelas "is, unfortunately, applied loosely to many other diseases accompanied by an erythematous blush (simple lymphangeiitis, various erythemata), and also to certain conditions of phlegmonous and diffuse cellular inflammation, which may complicate erysipelas but are due to a different virus—viz., the microbe of septicæmia."

The old and classical division of erysipelas into Medical and Surgical, proposed by Borsieri and sanctioned and adopted by Sir Thomas Watson, Bart., is purely artificial, and being useless as well as unfounded in fact, should be abandoned. When the disease affected the head and face, apart from surgical injury, it was described as "**Idiopathic**" or "**Medical Erysipelas.**" When it attacked the limbs, the body, or even the head after injuries, it was dubbed "**Traumatic**" or "**Surgical Erysipelas.**"

To Trousseau belongs the merit of pointing out the unreality of any such distinction. So long ago as 1856 Gubler[b] showed that facial, or the so-called "Medical" erysipelas, was only a **propagation of the disease from the pharynx,** and **not a metastasis,** a theory which before that had been often advanced. Trousseau

[a] *A Dictionary of Practical Medicine.* Edited by James K. Fowler, M.A., M.D. Art. "Erysipelas." London: J. & A. Churchill. 1891.

[b] *Mémoires de la Société de Biologie.* 1856. Page 40.

adopted Gubler's view, and as a result of most careful clinical observations maintained that the so-called idiopathic or medical erysipelas had almost always a starting point, which though it could not, strictly speaking, be called a wound, was at least a lesion—a very slight lesion it might be in some cases, such as a scratch, an eczema, an herpetic ulceration, an inflammation of the gum from a carious tooth, or a slight abrasion of the integuments at some point on the face, such as the corner of the eye, the nose, the lips, behind the ear, or on the hairy scalp.[a]

In these views Volkmann,[b] B. König,[c] Hirsch,[d] and Zuelzer[e] all concur, and the question may be regarded as settled.

Predisposing Causes.—(1.) It follows from the foregoing observations that we are to regard **Traumatism** as the most powerful predisposing cause of erysipelas in the abstract. So frequently has the disease arisen and spread in the wards of surgical hospitals, that the name "**Erysipelas nosocomiale**" has been applied to it. In a hospital the poison of erysipelas often clings to particular wards, and even to particular beds, with extreme obstinacy (Hilton Fagge.)

(2.) Although classed with the eruptive fevers, erysipelas presents some remarkable **points of contrast** to the other diseases included in this category. The brief period of invasion, the frequency of relapse, the peculiarity of its constantly starting from some definite point, and finally the atypical course of the disease, protracted sometimes in wandering erysipelas for weeks and even months, are points of difference enumerated by Zuelzer, of Berlin, the author of the monograph on Erysipelas in von Ziemssen's "Cyclopædia of the Practice of Medicine." To these should be added the striking fact that **one attack** of erysipelas **so far from protecting** an individual **from**, actually **predisposes him to, another attack.**

3. **Sex** does not seem to exercise any marked influence in pre-

[a] *Clinique Médicale de l'Hôtel-Dieu de Paris.* 1865. Tome I., page 165.

[b] Art. "Erysipelas," in von Pitha and Billroth's *Handbuch der allgemeine und specielle Chirurgie.*

[c] *Archiv. der Heilkunde.* XI., 23.

[d] *Handbuch d. histor. und geograph. Pathol.* I., page 243.

[e] Von Ziemssen's *Cyclopædia of the Practice of Medicine.* Art. "Erysipelas."

disposing to this disease. Of the fatal cases in England between 186: and 1868, 56 per cent. occurred in males.

4. Erysipelas is a disease of the **prime of life**, the period from the 20th to the 45th year being specially concerned (Volkmann).

5. The influence of **climate** is not well marked, although erysipelas is mostly a disease of **the temperate zone** of both hemispheres.

6. As regards **season**, Hirsch believes that erysipelas is particularly prevalent in damp, changeable weather, with unstable temperature. Most writers are of opinion that the disease occurs more frequently in the colder than in the warmer months of the year.

Exciting Causes.—These are briefly two—(1.) **Contagion**; (2.) **Inoculation.**

The doctrine of the **contagiousness of Erysipelas**, first promulgated by Wells[a] at the close of the last century, has been adopted in the United Kingdom since the time of Graves. In France it has received the influential support of Trousseau and other physicians of the foremost rank. In Germany Zuelzer and Hirsch accept it.

Mr. C. de Morgan, in Holmes' "System of Surgery," cites, on the authority of Dr. Goodfellow, a most extraordinary instance in which erysipelas spread in regular order through a ward of thirteen beds to almost every patient in turn, going down one side of the ward and then up the other side. In the "Archiv. für Heilkunde" for 1870 (Vol. XI., 23) Dr. B. König, of Rostock, records the facts relating to a small epidemic in the hospital of that town. The outbreak was clearly traced to infection from the pillows on the operating table, which had become deeply discoloured by dried blood from other patients. From the day when these pillows were removed no fresh cases of erysipelas occurred. The pillows were now soaked in water, and a brownish solution was obtained which was inoculated into two rabbits, with the result that one of them had an affection closely resembling erysipelas. From the point of

[a] *Transactions of the Society for the Improvement of Medical and Chirurgical Knowledge.* 1800. Vol. II., page 213.

inoculation, a diffuse dermatitis spread over the belly, produced œdema of the prepuce, bullæ and crusts in places, and disappeared after twelve days.

Not to quote authors at unnecessary length on a point which may be regarded as settled, I will merely add my own experience on this subject.

In November, 1874, I was placed by Dr. Stokes and Dr. Alfred Hudson in charge of a gentleman, aged thirty-seven, who was the subject of progressive locomotor ataxy, and who two days after exposure to cold on Friday, the 20th, displayed constitutional symptoms, which proved to be connected with an erysipelas of the scrotum. This first showed itself on Wednesday, the 25th, and by the 28th it had become gangrenous and involved the penis. On the 30th the right groin was implicated, and next day an erysipelatous patch appeared on the back of the right hand. Towards midnight of this day, December 1, a band of erysipelas spread across the nose and downwards into the month, finally invading the tongue, which became hugely œdematous. The urine was now considerably albuminous, spec. grav. 1018. It deposited amorphous urates and a few fragments of granular tube-casts. The patient, after violent delirium, began to sink rapidly, and expired at 4 a.m. of Thursday, December 3.

A married sister, aged thirty-six, attended the patient with singular devotion for several hours before his death. On Tuesday, December 8, she complained of sore throat and weakness. Her pulse was extremely rapid, and she looked seriously ill. Next day the throat felt and appeared to be better; but the mucous membrane of the nose looked unhealthy, and the orifice of the nostrils was swollen, puffy, and sore. On the following day the nasal mischief was more pronounced, but the throat continued to improve. On Friday, the upper lip became œdematous, and an attack of facial erysipelas was clearly in progress. On Saturday, the swelling spread upwards, and engaged the eyelids and forehead; across the latter a well-marked line of demarcation ran. Temp. in axilla was now 102·3°; pulse, 128. By 10 p.m. the inflammatory œdema had engaged the left ear, and reached the hairy scalp.

T. 103·5°. On Thursday, the 13th (6th day) the case was a typical one of facial erysipelas—P. 128; T. 103·8°.

This lady's illness proved exceptionally severe, and for many days her life hung in the balance. The temperature rose to 105·4° on the evening of the tenth day, and violent delirium was followed by symptoms of profound nervous prostration and by paralysis of the bladder. Ultimately she recovered perfectly. On January 11, 1875, I took this note: "She is desquamating everywhere, as if after an attack of scarlatina."

It should be mentioned that the valet of the unfortunate gentleman whose illness cost him his own life and placed his sister's in such jeopardy, was admitted to the City of Dublin Hospital on December 11, 1874, a few days after his master's death, suffering from abscess in the right tonsil and an unhealthy-looking herpetic eruption, involving the right side of the neck and the adjoining ear.

On February 21, 1884, Jane M. was admitted into Cork-street Fever Hospital, Dublin, on the fourteenth day of a severe attack of facial erysipelas. She died next day. On February 25th, Ward-maid Mary Lennon, who attended this patient, complained of not being well. On the 28th she was unable to leave her bed, complaining of severe shivering, headache, and pain in the back. It was ascertained she was suffering from facial erysipelas. Incubation in this case was apparently four or five days.

The most remarkable series of cases due to infection which have ever come under my notice, occurred in the year 1882. They illustrate especially the intimate relation which exists between erysipelas of the throat and ordinary cutaneous erysipelas. In the month of February in the year named, the Secretary of the Meath Hospital occupied as his sleeping apartment one of the Collis Wards; in the other, separated by a central corridor, lay a surgical patient suffering from traumatic erysipelas. In a few days the Secretary complained of painful sore throat, which was accompanied by severe constitutional disturbance, high fever, and extreme prostration. After a short time the larynx became engaged, and symptoms of œdema of the glottis supervened. The treatment

adopted happily proved successful, and the patient gradually recovered. He was devotedly attended and nursed by a sister who, in a few days, fell ill of a sharp attack of *facial* erysipelas, which ran a normal course. And now comes the interesting part of the story. During his sister's illness the Secretary sent bulletins as to her state to a married sister living in the County Sligo, at a distance of 130 miles. Letters written by himself were despatched on the 10th and 13th of March, and a post card followed on the 17th. The lady incautiously placed these letters and post card under her pillow at night, with the result that on the 21st of March she sickened with severe sore-throat, accompanied by intense pain and much swelling. She was attended by a domestic servant, who in a few days developed an attack of facial erysipelas.

Hirsch points out that **epidemics complicated with throat-affection** have been repeatedly described. Of one such outbreak at Montrose in 1822, Gibson[a] says: "The disease was not so much confined to the head or face as common erysipelas, but it frequently attacked other parts of the surface of the body. *Sometimes the internal fauces were attacked*, and if it spread to the trachea it generally proved fatal."

Trousseau[b] narrates a strikingly similar instance to those I have just detailed of the spread of erysipelas and of its alternating phases in different individuals. I give his very words:—"J'étais appelé en consultation par mon honorable ami M. le docteur Paris, auprès d'un M. E. . . . chez lequel un de nos chirurgiens les plus habiles, M. le Professor Nélaton, avait été obligé de pratiquer le débridement du méat urinaire afin de faciliter l'introduction d'instruments lithotripteurs. M. E. . . . succombait à un érysipèle gangréneux du prépuce, qui avait eu pour point de départ cette petite incision. La veille de sa mort, sa femme, qui l'avait soigné avec une grande sollicitude, fut prise de frissons; le lendemain elle avait une angine violente, et vingt-quatre heures après un érysipèle de la face d'une extrême gravité, qui l'emporta alors qu' elle semblait entrer en convalescence. La femme de chambre tomba malade en

[a] *Trans. of the Edinburgh Med. Chir. Soc.*, 1828. Vol. III., page 94.
[b] *Clinique Médicale de l'Hôtel Dieu de Paris.* 1865. Vol. I., page 171.

même temps que sa maîtresse, elle n'avait cessé de donner des soins à M. E. . . . La maladie chez elle fut caractérisée surtout par une violente angine, et par un érysipèle qui se limita aux paupières."

The question of the **inoculability** of erysipelas has been definitely settled by experiments on animals conducted by Orth,[a] Koch,[b] Tillmanns,[c] and others, and most decisively of all by Fehleisen's inoculation experiments on man with micrococci of pure cultivation.[d]

Bacteriology.—According to Lukomsky,[e] minute cocci occur in chains (*Streptococci*) in human erysipelatous skin, and in the fluid of erysipelatous bullæ. They occupy the lymphatic channels of the skin, and spread along them as the disease progresses. These **Streptococci erysipelatis**, or **Micrococci erysipelatosi**, as Fehleisen called them, can be cultivated artificially in nutrient gelatine, or agar-agar, and according to Orth produce typical erysipelas when re-inoculated in man or animals. The characteristic erysipelatous blush is produced by inoculating these micro-organisms in the ear of a rabbit. In the human subject the disease was produced in fifteen to sixty hours after inoculation.[f]

Fehleisen[g] made a number of **inoculation experiments** on human subjects with **the therapeutical intention of dispelling tumours.** All the experiments succeeded, and the erysipelas always ran a perfectly normal course. A beneficial result was obtained in cases of lupus, cancer, and sarcoma. Hence this induced disease has received the name "**Erysipèle salutaire.**"

The pathogenic micro-organism of erysipelas spreads, as has been stated, by the lymphatics, which may be seen infiltrated with aggregated masses of spherules; thence it penetrates into the tissues and forms chains or swarms of spherical cocci, ·3μ in diameter. It excites a specific inflammation and leads to tissue necrosis. Cultivated on gelatine, it forms whitish colours but does not liquefy it.

[a] *Arch. für experiment. Pathologie.* 1873. I., 81.
[b] *Untersuch. über die Aetiologie der Wundinfections-Krankheiten.* 1878.
[c] Billroth and Lücke's *Deutsche Chirurgie.* V., 75.
[d] *Deutsche Zeitschrift für Chirurgie.* 1882. XVI., 391.
[e] *Virchow's Archiv.* 1874. LX., 418.
[f] E. M. Crookshank. *Manual of Bacteriology.* Second Edition. 1889. P. 200.
[g] *Deutsche Zeitschrift für Chirurgie,* 1882, XVI., 391; and *Sitzungsber. der Würzb. phys.-med. Gesellschaft,* 1882. No. 8.

CHAPTER XXII.

Erysipelas (*continued*).

Clinical History—Temperature—Diagnosis—Prognosis and Mortality.

Clinical History.—Varieties of Erysipelas according to depth of surface affected: (1.) Simple or cutaneous; (2.) Phlegmonous, or cellulo-cutaneous; (3.) Diffuse cellulitis.—Stages of incubation, invasion, eruption, defervescence. Erysipelas faucium, pulmonum.—Temperature.—Diagnosis.—Prognosis and Mortality.

Clinical History.—Bearing in mind the polytypical nature of erysipelas, we are not surprised to find that its course is less cyclical than that of the other eruptive fevers. In fact no two cases correspond exactly in mode of origin, symptoms, and duration. All stages of the exanthem may be observed simultaneously in the same individual—its beginning, acme, decline, and resulting desquamation. Notwithstanding this, it will be convenient to consider the disease in the same systematic way as the others of its class, premising that it is classified as regards the degree or depth of surface to which the inflammation penetrates as—

1. Simple, or cutaneous, Erysipelas.
2. Phlegmonous, or cellulo-cutaneous, Erysipelas.
3. Diffuse inflammation of the subcutaneous areolar or cellular tissue, or Diffuse Cellulitis.

This is a commonly adopted but quite artificial classification.

1. **Stage of Incubation.**—This period is of rather indefinite duration. On an average it lasts about seven days, being sometimes shorter, sometimes longer—for example, Dr. Roberts assigns to it a duration of from 10 to 14 days. In the cases from my own practice, which I have detailed, the incubative period varied in length from 4 or 5 to 8 or perhaps 10 days, and these limits are probably the most common.

II. **Stage of Invasion.**—Taking its origin in a herpetic ulceration, or other more or less trivial lesion of the pharynx, mouth, lips, nose, ears, or eyes, facial erysipelas causes *general discomfort*, *loss of appetite* (*anorexia*), *vomiting*, and *diarrhœa*. It is usually ushered in by *chills*, *rigors*, or even *epileptoid convulsions*, as pointed out by Dr. Bathurst Woodman. The *temperature rises rapidly* in a few hours, often to 104° F. (40° C.), or even higher. *Severe pain in the throat* is commonly felt, especially in cases due to contagion. There is *a vivid redness* over the *uvula*, *velum palati*, and *tonsils*, and the *glands* in the neck, and under the lower jaw, are nearly always *swelled* and *tender on pressure*.

In an account of an outbreak observed in the Paris hospitals, Cornil describes a purple-red, shining, œdematous swelling of the whole pharyngeal mucous membrane, which appears as if varnished. The tonsils may remain free from these changes. Great pain is felt in swallowing (*dysphagia*), and there may be *salivation*. I would like to lay special stress on the painful nature of erysipelatous angina. It is a symptom the presence of which I have repeatedly verified by personal observation. To this variety of erysipelas the name **Erysipelas Faucium** is applied.

III. **Stage of Eruption.**—After a few hours, or one, two, or three days, the erysipelatous inflammation spreads from the pharynx or its other point of origin on to the face, every part of which may be invaded *except the chin*, for an anatomical—or rather histological—reason, which will be explained under the heading "Pathology." The dermatitis tends to advance in an ever-enlarging circle from the original focus, while a well-defined and raised margin marks the approach to a given spot of the accompanying swelling and redness. The natural lines of cleavage of the skin modify the rate and manner of extension of the inflammation. Where the skin is tightly stretched (as on the scalp) or firmly bound down (as on the chin), it progresses slowly and irregularly, or in a zigzag fashion, along the lines of least resistance. On the other hand, regions where the subcutaneous areolar tissue is lax (such as the eyelids and scrotum) become rapidly and enormously swollen (J. J. Pringle). The patient now complains of *stiffness* and a *burning*,

tingling sensation in the affected area of the skin, the surface of which is swollen, red, hard, and tense from effusion of a serous fluid, rich in young cells, into the subcutaneous areolar tissue.

The most striking word-picture of erysipelas with which I have ever met is Sir Thomas Watson's description. He says:—"The lips swell enormously, the cheeks enlarge, the eyes are sealed up by their œdematous and prominent lids, and all traces of the natural countenance are effaced. I know of no disease except, perhaps, the confluent smallpox, which so completely and speedily deforms and disguises the visage of the patient. A stranger seeing a young female in the height of the disorder, and revisiting her after her recovery, is astonished at the change. It seems as if, by some magic process, such as we read of in our nursery tales, a hideous monster has been metamorphosed into a comely damsel."[a]

It has been already stated that the swelling and redness generally stop at the edge of the hairy scalp. This was stated by Watson to be particularly the case if the dermatitis spread symmetrically across the face from the nose; but the accuracy of the observation is with reason called in question by Trousseau.[b] Sometimes—and in this respect it exactly resembles a scald—the inflamed surface becomes covered with irregular *blebs* or *blisters* (*bullæ*), particularly on very vascular surfaces like the cheeks. Vesication, however, is often wanting, so there is nothing diagnostic in this phenomenon. The vesicles, or blebs, dry up into crusts, as a rule; but where the dermatitis dips deeply, or the tension from œdema is extreme, as in the eyelids, suppuration is apt to take place, and the tissues may even slough.

As regards a given portion of surface, when the redness has lasted three or four days, it fades, swelling quickly subsides, and desquamation sets in. Nothing is more curious than to see these changes taking place while the advancing disease is attacking other parts for the first time. During all this time the fever ebbs and flows according to the progress of the malady.

[a] *Lectures on the Principles and Practice of Physic.* Third Edition. London: John W. Parker. 1848. Vol. II., page 827.

[b] *Clinique Médicale de l'Hôtel Dieu de Paris.* 1865. Tome I., page 171.

IV. Stage of Defervescence.—When the swelling and redness cease to spread, the temperature falls rapidly in uncomplicated cases. In other instances defervescence is a very tedious process indeed, occurring by lysis rather than by crisis. Or the temperature range may closely simulate that of pyæmia. This happens when the dermatitis *wanders* all over the body, or *jumps* (as it did in one of the cases detailed by me above) from one part of the body to another. This form of the disease receives the name of **erratic or vagrant erysipelas (erysipelas migrans)**—the "**erysipèle ambulant**" of French writers. In such cases the illness may be protracted for one or two months, and not only every part of the surface of the body, but the whole tract of mucous membranes, and even the lungs, and, I believe, the pleuræ, may in turn become affected. Dr. Peter,[a] of Paris, has drawn attention to the spread of the inflammation from the pharynx to the respiratory passages, causing bronchitis, bronchiolitis (capillary bronchitis), and pneumonia.

In a case observed by me at Cork-street Fever Hospital many years ago, the reverse of this happened. A man was admitted suffering from *Pneumonia migrans*. After some days, a blush of erysipelas showed over one shoulder, and spread thence down the back, and simultaneously the pneumonic symptoms subsided. So great was the impression made upon me by this case that ever since I have recognised the propriety of considering **erysipelas pulmonum** as a distinct species of the great genus **Pneumonia.**

Temperature.—But little remains to be added to the incidental remarks on the behaviour of the temperature which have been made in the foregoing account. "Facial Erysipelas," writes Wunderlich,[b] "is pre-eminently a polytypical disease, and in many cases it is quite atypical." He adds: "At present it is not possible to associate special forms of erysipelas, or special causes of it, with particular forms of fever curves."

The temperature commonly rises in a few hours to nearly 40° C. (104° F.) or even higher. So long as the cutaneous or anginal

[a] *Dictionnaire Encyclopédique des Sciences Médicales.* Tome IV, page 720. Art. "Angines."

[b] *Medical Thermometry.* New Syd. Soc. 1871. Page 351.

inflammation develops and extends, so long does the temperature tend upwards to 104°, 105·8°, or even 106·7° in the evenings, falling to 102°, 103°, or only 104° in the mornings. Sometimes there are intermissions of pyrexia at times, with great exacerbations at other times. When defervescence does set in, it generally goes on with such speed that in the course of a single night, the thermometer falls to normal or nearly so. In some instances, after several days a fresh and striking rise of temperature occurs, which either accompanies or heralds a new extension of inflammation.

In fatal cases **death** usually occurs with **very high temperatures.**[a]

Complications and Sequelæ.—For the most part, the complications of erysipelas are one or other of the ordinary symptoms presented in an aggravated form. Thus diarrhœa, nausea and vomiting, œdema of the fauces, glossitis, sloughing and abscess of the eyelids, may one and all interfere with the normal course of the malady and endanger the patient's life by prolonging his illness and shattering his strength.

Apart from the foregoing, the complications are:—

1. **Cerebral symptoms,** namely, headache, delirium, insomnia, tremors, mania, and coma, ending in death. This formidable group is usually dependent on **hyperpyrexia** and **not on meningitis,** which is extremely rare in erysipelas.

2. **Bronchitis** and **Pneumonia**—the latter either of the nature of a lobular pneumonia in bronchiolitis, or of severe croupous pneumonia (Trousseau, Ritzmann.)

3. **Hypostatic Congestion of the Lungs,** especially in protracted cases (Volkmann.)

4. **Pleuritis,** considered by Lawrence, Wutzer, and Volkmann, to be a direct extension of erysipelas to the serous membrane—by contiguity, not continuity.

5. The "**Puerperal State**" either in lying-in women or in newly born infants. In the latter case the disease is called in France "**Erysipèle des Nouveau-nés.**" It usually begins in infants a few

[a] Wunderlich, *loc. cit.*, page 354; Blass: *Beobachtungen der Erysipelas*, Leipzig, 1863; Eulenburg, *Centralblatt*, 1866, page 65; Ponfick, *Deutsche Klinik*, 1867, 20–26.

SIPELAS.

Fig. 3.—Facial Erysipelas.

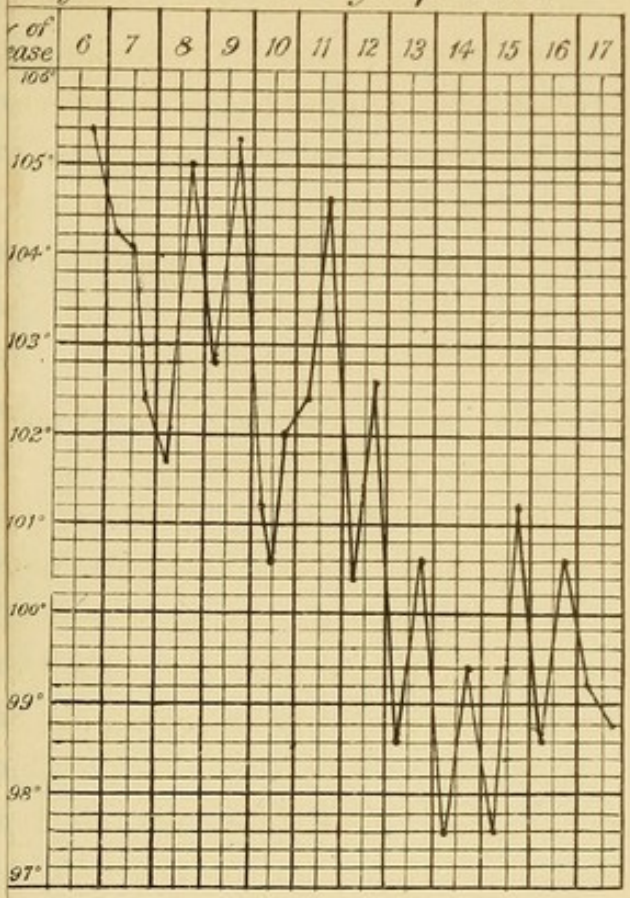




Plate IV.

CHARTS OF TEMPERATURE RANGES IN ERYSIPELAS.

Fig I—Facial Erysipelas.

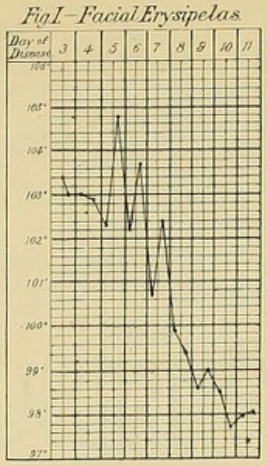


Fig 2—Facial Erysipelas.

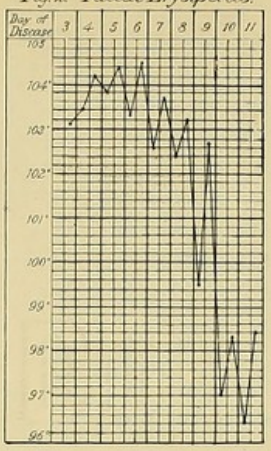


Fig 3—Facial Erysipelas.

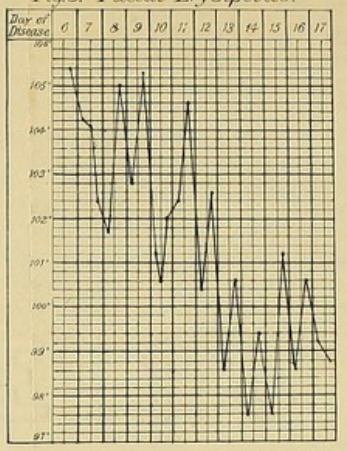
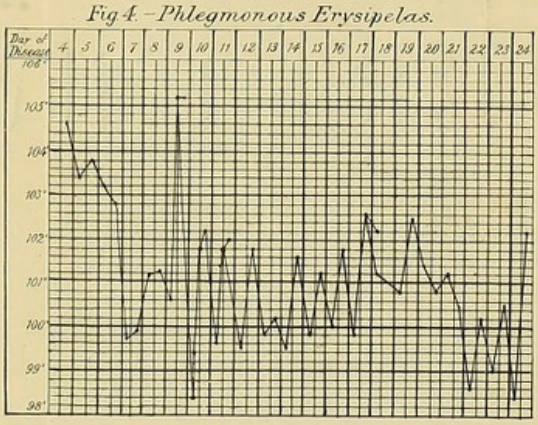


Fig 4—Phlegmonous Erysipelas.



hours or days after birth as a red blush over the hypogastrium rather than about the umbilicus. It commonly ends in peritonitis and is invariably fatal in such young children, the explanation according to Trousseau and Paul Lorain[a] being that the same "epidemic puerperal influence" affects both recently delivered women and new-born infants.

6. **Peritonitis**, regarded by Dr. Samuel Wilks[b] as, in some cases at all events, the result of a direct extension of erysipelas from the abdominal walls.

7. **Leucocytosis.**—In his "Cellular Pathology," Virchow[c] pointed out that the acute irritation of the glandular apparatus led to this condition, which becomes serious in "wandering erysipelas" when this form of the disease attacks debilitated persons.

8. **Pyæmia** is a rare sequela. In erysipelas associated with severe or extensive injuries, Zuelzer and Volkmann both consider that pyæmia may actually be of use, by favouring the destruction and consequent dispersion of any embolic infarctions and thrombi which may have formed.

9. Extensive **gangrene** of the skin often points to severe pyæmic and septicæmic phenomena generally of evil omen, as in one of the cases detailed by me in the foregoing pages. A marked bloody hue of the contents of the blebs is often the first sign of commencing gangrene, which may at times be checked by early puncturing (Zuelzer).

Pathology.—There is **little that is specific** in the morbid anatomy of erysipelas. According to Ponfick, heart, spleen, and kidneys are the seat of a more or less parenchymatous degeneration; but this change was shown by Liebermeister to be common to all severe febrile affections. The **morbid histology** of erysipelas has been studied with care by Biesiadecki,[d] Volkmann, and Steudener. The first-named observer showed that, in the facial form of the disease, simple erysipelas, like phlegmonous inflammation, affects the skin in its whole thickness and the subcutaneous

[a] *La Fièvre puerperale.* Paris. 1855.

[b] *Guy's Hospital Reports*, 1861.

[c] *Cellular Pathologie.* 1871. Page 230.

[d] *Sitzungsbericht d. k. Acad. der Wissenschaft zu Wien.* 1867. Vol. II., p. 231.

areolar tissue. In the same way, Volkmann found the deeper strata of the cutis, as well as the subcutaneous tissue infiltrated with enormous numbers of granular leucocytes. In 1869, Dr. Charlton Bastian,[a] finding the small arteries and capillaries of the brain plugged with embolic masses of white blood-corpuscles in a man who died from erysipelas in a state of delirium and stupor, suggested that this pathological condition was the cause of the cerebral symptoms named.

The globular bacteria of erysipelas (*Punctiform Bacteria* of Ehrenberg, *Microspheres* of Cohn), were first discovered by Hüter. They were everywhere forthcoming in the inflamed tissues, being especially abundant in the œdematous parts, much less so in the blood. In 1883, Fehleisen[b] described the micro-organism of the disease more particularly as a coccus arranged in chains; to it he, therefore, gave the name of **Streptococcus erysipelatis.** More recently, V. Noorden[c] found *streptococci* in blood taken from the body of a woman who had died of erysipelas.

Richter, of Göttingen,[d] in 1744, first put forward the view that erysipelas was **an inflammation of the lymph vessels.** According to Trousseau, erysipelas is essentially a **capillary lymphangeiitis,** or inflammation of the minute lymphatic vessels of the skin. All this may be true, but it does not shake our faith in the infective, specific, and febrile nature of the disease, and therefore, with all its differences from the other eruptive fevers, we still include erysipelas with them as a true exanthem.

In a very suggestive paper, entitled "Recent Advances in the Ætiology of Diseases of the Skin and their bearing upon Treatment," read on November 20, 1891, before the Royal Academy of Medicine in Ireland, Dr. Walter G. Smith observed that "erysipelas and lymphangitis are not convertible terms." He goes on to say: "It is generally held as settled that *acute* suppuration is—as Ogston first showed—due to the action of micro-organisms; but it is a

[a] *Trans. Path. Soc. of London.* 1869. Page 8.
[b] *Die Aetiologie des Erysipels.* Berlin. 1883.
[c] *Münch. med. Wochenschrift.* Vol. XXXIV. No. 3. 1887.
[d] *De Erysipelate Dissertatio. Opuscula Medica.* Göttingen. 1744. I.

difficult matter to determine whether erysipelas is an entity, a truly specific disease, or whether it does not represent a phase or mode of action of the pus-producing organisms. Watson Cheyne is inclined to uphold the specific character of the erysipelas germ, while Levy in a valuable paper (Archiv. für experiment. Path. und Pharm., xxix., 'Ueber die Mikro-organismen der Eiterung,' page 135) teaches that *Streptococcus pyogenes* is at once an exciter of suppurative processes and of erysipelas.

"It is more than probable that there are several species of erysipelas due to different bacteria."

Pfleger considers that the mode of advance of the dermatitis depends not so much on the course of the lymph channels, as originally held by Billroth, but mainly upon the arrangement of the subcutaneous connective tissue bundles. They everywhere interlace so as to form rhomboidal meshes; but these are usually horizontal or oblique, except upon the chin, where their direction is vertical. Hence, the chin always remains untouched by erysipelas, as pointed out by Volkmann. Pfleger also maintains that wherever the skin is tied down to the deeper parts the spread of the disease is retarded or arrested, as, for example, along the crest of the ilium, and Poupart's ligament. Billroth[a] uses a striking illustration when he says that the redness advances through the skin in many cases, just as fluids spread in blotting-paper, rounded tongue-like subcutaneous projections shooting out, which are followed by a broader advance.

The formation of vesicles or blebs has no specific significance.

Metschnikoff has described cells which he calls "**microphages**"[b] and "**macrophages**"[c] in connection with the erysipelatous process. The microphages are small free leucocytes which he discovered in the margin of an erysipelatous patch of skin, and which he believed possess the power of destroying the infecting micrococci of the disease. The macrophages are large leucocytes also found in

[a] *Lectures on Surgical Path. and Therapeutics.* New Syd. Soc. London. 1888. Vol. II., page 10.

[b] Gk. *μικρός, small, φαγέω, I devour.*

[c] Gk. *μακρός, great, φαγέω, I devour.*

structures affected with erysipelas. Metschnikoff says that they consume and destroy the *débris* of the dead and dying microphages.[a]

According to Volkmann and Steudener, as the inflammation wanes, the leucocytes disappear in the subcutaneous tissue with extraordinary rapidity, breaking down in a few hours into a granular *débris*. In the superficial layers of the cutis they remain visible a little longer; but within a day or two all signs of tissue changes may have vanished. Meanwhile, the vesicles or bullæ have dried up into yellowish crusts. The cuticle is subsequently shed either in flakes or as a branny powder (Hilton Fagge),

When the scalp is attacked, the hair usually falls off during convalescence. Haight says that this happens because the external root-sheath is separated from the vitreous layer of the follicle as far as its junction with the papilla by the serous infiltration. The hairs quickly form again.

Diagnosis.—The recognition of erysipelas is generally easy once the eruption has shown itself upon the skin. Frank has pointed out that when a patient has had febrile symptoms for some hours accompanied with pain, tenderness, and swelling of the lymphatic glands, erysipelas is doubtless coming on. Chomel held the same view, and Campbell de Morgan relates that "Busk is so convinced of the invariable occurrence of affection of the glands before erysipelas appears, as to consider it a pathognomonic symptom."[b]

In this view Trousseau[c] also concurs, and he quotes the following passage from Borsieri, who noted swelling of the lymphatic glands as marking the onset of the malady:—"Illud etiam memoriâ probe tenendum est, quod crebris ex observationibus constitit, si erysipelas artubus inferioribus incubiturum sit, inguinis et femoris glandulas conglobatas, vasis cruralibus additis, antequam se exserat, leviter dolere atque intumescere consuevisse, axillares vero ac cervicales, si brachiis *aut superioribus locis* immineat."

The **anginal form** is often not easily recognised. The pain-

[a] See the New Syd. Society's *Lexicon of Medicine, sub vocibus* "Microphage" and "Macrophage."

[b] Thomas Bryant. *Manual for the Practice of Surgery.* London: 1884. Fourth Edition. Vol. I., page 100.

[c] *Clinique Médicale de l'Hôtel Dieu.* 1865. Tome I., page 169.

fulness, œdema, and implication of the cervical glands are important elements of diagnosis.

Erysipelas has to be distinguished from—

1. **Simple erythema** (E. simplex), which is a transitory hyperæmia of the skin, running its course with little, if any, pyrexia, without pain, swelling, or desquamation. In it the glands also are not involved.

2. **Urticaria**, or **nettle-rash**, which is intensely itchy and consists of wheals distributed here and there over the body.

3. **Simple lymphangeiitis**, which shows a streaked or spotted, seldom a confluent, redness, extending centripetally, the inflamed lymphatics appearing like firm cords. It often terminates in suppuration.

4. **Diffuse phlegmonous inflammation**, which shows a darker redness, nowhere sharply defined, and a board-like hardness. It generally ends in suppuration.

5. **Pemphigus.**—This is distinguished by its relative chronicity, its localisation, the absence of œdema, and the inconstant implication of the lymphatic vessels and glands.

6. **Eczema rubrum**, in which the dermatitis is severe, painful, and œdematous. The pyrexia, however, and constitutional disturbances are ill-defined, and the disease is comparatively limited. A coloured drawing of acute eczema will be found in the New Sydenham Society's "Atlas of Skin Diseases" (Plate XVI.).

7. Hilton Fagge has known blunders repeatedly committed in the differential diagnosis of **herpes zoster of the forehead and face**, and erysipelas.

Herpes does not cross the middle line; there is no desquamation, and the vesicles in time dry up into characteristic dark brown eschars, embedded in the skin. I would add that the febrile movement in herpes zoster is not acute as a rule.

Prognosis.—This, so far as facial erysipelas is concerned, is **generally favourable.** One of the Aphorisms of Hippocrates on Erysipelas is well worth quoting. It runs thus: "*Ἐρυσίπελας ἔξωθεν καταχεόμενον εἴσω τρέπεσθαι οὐκ ἀγαθόν, ἔσωθεν*

δὲ ἔξω ἀγαθόν.[a]—"For superficial erysipelas to turn inwards is not a good sign; for internal erysipelas to become superficial is a good sign."

Billroth says the disease commonly lasts from 2 to 10 days: 14 days is an unusual duration. The longest case that he ever saw lasted 32 days and terminated fatally. Zuelzer assigns a much longer duration than this, and one of my own cases ran to 66 days. The average duration is—12 days (Velpeau); 13½ days (Heyfelder); 10, 12, or 14 days (Zuelzer). Relapses and recrudescences are common.

The **mortality** varies much according to circumstances—Wunderlich had only 3 per cent. of deaths, Volkmann 5 per cent.; in the American War (1862) the mortality among surgical cases was 11 per cent. Of 137 cases without complications which Billroth had under his care at Zurich, 10 died.

Puerperal women, new-born children, old people, the subjects of debilitating chronic diseases, sufferers from any of the other eruptive or continued fevers, the victims of intemperance, all run a bad chance if attacked by erysipelas.

Erysipelas migrans is particularly dangerous, but ordinary erysipelas may kill, according to Trousseau, by **coma**, consequent on effusion within the head; by **asphyxia**, owing to œdema of the glottis; or by **asthenia**. This last cause of death operates especially in the wandering or erratic variety.

Although erysipelas most frequently ends in recovery, the prognosis should be guarded in all cases. Bad signs are: evidences of blood-poisoning, severe head-symptoms, typhoid or ataxic symptoms, extension of the inflammation to the larynx or bronchial tubes, a dark coloured rash with livid vesicles or bullæ, and—as pointed out by Dr. F. Roberts—a sudden disappearance of the external inflammation with a coincident development of internal symptoms.

[a] *Hippocratis Coi Aphorism.* Lib. vi. Sec. 7. Aphor 25. Editio Foesii. Francofurti. 1624.

CHAPTER XXIII.

Treatment of Erysipelas.

Expectant Treatment.—Constitutional (or general) and Topical Treatment.—Constitutional Treatment: tincture of the perchloride of iron, quinine, ammonia and bark in effervescence, salicylate of sodium (Hallopeau), cold baths, alcoholic stimulants, effervescing draughts in gastro-intestinal disturbance, opium or morphin in threatening delirium.—Topical, or Local Treatment: indications—(1.) to relieve pain and tension; (2.) to check the spread of inflammation; (3.) to destroy the infectious matter *in situ*.—Leeches are contra-indicated.—"Ectrotic" method.—Topical use of nitrate of silver.—Rectified oil of turpentine.—Sulpho-carbolate of sodium.—Sprays.—Special treatment of œdema of the eyelids, sore throat, laryngitis, and œdema of the glottis, tension of the skin, gangrene.

Many patients suffering from uncomplicated facial erysipelas quickly recover, with very simple management indeed. The **expectant treatment** is the **rational treatment** in such cases. Trousseau having alluded to the active measures adopted in some hospitals against the disease, somewhat satirically says: "Vous avez vu, *malgré cela*, la maladie guerir," and adds: "l'érysipèle est donc une de ces affections qui guérissent d'elles-mêmes, je parle de l'érysipèle qui surprend l'individu en bonne santé, et non plus de celui qui survient dans le cours d'autres maladies."[a]

This is all quite true, but we are often called upon and bound to adopt other means than those which the great French physician embraces under the term "expectant treatment," namely, to keep the patient in bed protected from draughts, to prescribe slightly acidulated diet drinks, to give laxatives as required, to relieve vomiting by purgatives, and to give nourishment freely—his very words are dramatic: "J'alimente, j'alimente alors même qu'il y a de la fièvre, alors même qu'il y a du délire . . . au lieu de les tenir à une diète rigoureuse, je reste spectateur de la lutte de laquelle, je le sais, la nature sortira victorieuse, si je ne la trouble pas dans ses opérations; je me tiens les bras croisés: et, je le

[a] *Clinique Médicale de l'Hôtel Dieu de Paris.* Tome I., page 174. Paris: 1865.

répète, parmi le grand nombre d'érysipèles que j'ai vus, trois tout au plus ont eu une terminaison fatale; dans tous les autres cas la maladie s'est éteinte d'elle-même."

The treatment of erysipelas naturally falls under two headings—**constitutional**—or **general**—and **topical**, and yet it is hard to dissever one from the other, for to destroy the infectious matter is one of the objects alike of constitutional and of topical treatment.

Constitutional Treatment.—Apart from the management of the surroundings, dieting, and nursing of an erysipelatous patient, the following remedies have enjoyed a well-merited reputation while not attaining to the rank of a specific against the disease:—

1. **Tincture of the perchloride of iron**, recommended by Hamilton Bell in 1851, has since then been given, especially in facial erysipelas, in full doses (20 to 30 minims) often repeated—that is, every second, third, or fourth hour. It may with advantage be prescribed with equal quantities of glycerine, and in peppermint or chloroform water. Mr. de Morgan, of the Middlesex Hospital, says that this treatment is most efficacious in shortening the duration of the attack and securing a rapid and satisfactory convalescence. He has given as much as an ounce to an ounce and a half of the official tincture in twenty-four hours, in the more severe forms of the disease. The ethereal tincture of the German Pharmacopœia, which contains one per cent. of ferric chloride in a mixture of one part of ether and three parts of alcohol, is a favourite preparation in Germany under the name of "Bestuscheff's tincture."

In that country, however, **quinine** now enjoys a higher reputation, in consequence of the researches of Binz[a] and the recommendation of Liebermeister. When given in doses up to four and a half grains every two hours, quinine reduces the fever and shortens the attack. A long experience leads me to recommend that quinine in such doses should be administered, not in an acid solution, but mixed with milk or plain water. There is really nothing new in the quinine treatment of erysipelas, for long ago a mixture of **ammonia** and **bark in effervescence** was regarded as a sovereign remedy.

[a] *The Elements of Therapeutics.* Translated by Edward I. Sparks, M.A., M.B, Oxon. London: J. & A. Churchill. 1877. Pages 206, *et seq.*

Ninety grains of carbonate of ammonium in six ounces of decoction of bark, to be taken in ounce doses, effervescing, with half an ounce of fresh lemon juice every four or six hours, was a standard prescription.

M. Hallopeau[a] has employed and recommends the following method in the treatment of erysipelas with **salicylate of sodium**:— 1. Application to the part of compresses wet with a solution of salicylate of sodium (1 in 20) covered with oiled silk and frequently renewed. 2. Internal administration daily of four grammes (3j) of the salicylate, in three doses, in weak grog. M. Bochefontaine's experiments have shown that, if to a joint compresses wet in a solution of salicylate of sodium (1 in 20) be applied and covered with oiled silk, the drug will soon appear in the urine. Hallopeau begins the internal treatment with a calomel purge, and then gives sulphate of quinine and salicylate of sodium alternately, at a day's interval the one from the other.

The results of this treatment, observed in twelve cases, were as follows:—1. The temperature was undoubtedly lowered in those cases which presented marked febrile disturbance. 2. In most cases the duration of the disease was much abridged, judging from the statement of Velpeau that twelve days is the usual course. In several cases the disease seemed quite promptly controlled. 3. Up to the time at which he wrote the author had not observed the accidents which have occurred within his knowledge in typhoid-fever patients taking the same doses of the remedy. In one case there was a slight temporary delirium, which may not, however, have been due to the medicine. The author recommends suspension of salicylic acid treatment in this and other diseases as soon as cerebral disturbance or dyspnœa makes its appearance.

I have myself followed this line of treatment in cases of erysipelas and with the happiest results, nor have I ever seen any untoward effects from the use of this almost specific remedy.

To **reduce the temperature** Volkmann recommends **cold baths**— repeated, if necessary, three or four times a day. They are grateful

[a] *L'Union Médical*, May 1, 1881, and *New York Medical Journal and Obstetrical Review*, September, 1881.

to the patient and relieve head symptoms, but do not control the erysipelatous inflammation. The bath should be given at 80° F. and for 15 or 20 minutes.

In ordinary erysipelas, **alcoholic stimulants** are contra-indicated, but in the severer forms of the disease their free administration is called for, subject to the conditions laid down in Chapter V., page 58.

In **gastro-intestinal disturbance,** effervescing draughts are useful, or small doses of bicarbonate of sodium with rhubarb and calumba. Schacht's "Liquor Bismuthi" also is an excellent adjuvant. A single dose of calomel (5 grains) is often advantageous in this condition.

In **threatening delirium,** opium in full, but guarded doses, or hypodermic injections of morphin are most valuable remedies.

Topical or **Local Treatment** has three ends in view—First, **to relieve pain and tension;** secondly, **to check the spread of the inflammation;** thirdly, **to destroy the infectious matter** *in situ.*

The first indication is met by covering the affected part with cotton wool so as to exclude the air; by dusting it over with flour, or powdered starch, or a mixture of oxide of zinc and salicylated starch, or—best of all (for when wet, it does not cake on drying)—with a mixture of oxide of zinc and lycopodium powder, of each half an ounce, intimately shaken up with 15 to 30 minims of liquefied pure carbolic acid. If these means fail, the part should be fomented with flannels wrung out of a hot decoction of poppies (Sir Thomas Watson), or covered with spongiopiline, soaked in hot water and sprinkled with laudanum. The old-fashioned prejudice against "wetting the rose" has long since been given up, in medical circles at all events.

Marc Sée, of Paris,[a] employs subnitrate of bismuth as a dressing. It is a preventive as well as a curative agent. It should be dusted as a powder topically over the solution of continuity, which is the point of departure of the malady.

Leeches are contra-indicated in erysipelas, because each leech-

[a] Paul Lefort: "*La Pratique Journalière des Hôpitaux de Paris.*" 1891. Page 153.

bite would be a *trauma* or wound from which the specific dermatitis would take a new departure. Cold evaporating lotions, lead lotion, and poultices are supposed to predispose to gangrene in erysipelas.

With the view of checking the spread of the dermatitis, various ointments for smearing over the erysipelatous surface have been proposed. Such are mercurial ointment (Ricord), and an unguent consisting of dried sulphate of iron, 60 grains; glycerine, 20 minims; lard, an ounce and a half (Alfred Hudson).

The erysipelatous patch may be painted over with collodion, mixed with glycerine (1 part to 15 parts) or cod liver oil (Zuelzer and Fenwick.) Griscom employed glycerine, because it dehydrates the tissues and so exerts an antiphlogistic effects upon the inflamed parts.

Under the name of the "**ectrotic**[a] **method,**" Higginbottom introduced the **topical use of nitrate of silver.** A broad band of skin thoroughly washed and freed from fat, outside the advancing line of inflammation, was cauterised with nitrate of silver in substance or in strong solution (one in eight parts). Nunneley and Hasse recommended painting a *cordon* half an inch wide in the same way with tincture of iodine.

To destroy the virus *in situ*, Copland applied **rectified oil of turpentine**—a practice which has gained the approbation of Lücke, Borgien, Bonfigli, and Zuelzer. With the same object in view, Nyström and Westerland, two Swedish physicians, introduced the use of "aseptin," a powder, and "aseptin-amykos," a liquid, composed of boric acid and oil of cloves. Still more recently **carbolic acid** (phenol) has been used, and especially by Hüter in 1874 in the form of **hypodermic injections** of the strength of 2 per cent., which besides checking the spread of the erysipelas exert a local anæsthetic action, which is very grateful and beneficial.

Wilde got good results from injecting hypodermically into the inflamed skin from 15 to 30 minims of a ten per cent. solution of **sulpho-carbolate of sodium.** The local application of compresses

[a] "Ectrotic" (from the Greek ἐκτρωτικός, *belonging to abortion*) is applied to medicines or modes of treatment which tend to produce the abortion or sudden cutting short of a disease.

wet with a 5 per cent. solution of salicylate of sodium, recommended by Hallopeau, has already been mentioned.

Talamon, of Paris, with Richardson's spray-producer sprays for a minute over the external erysipelatous zone, both within and on the outside of the swelling, with the following solution :—

℞. Hydrargyr. perchloridi, gr. 15½;
Acid. citrici, vel tartarici, gr. 15½;
Spt. rectificati, min. lxxx.;
Ætheris sulphurici, ad ℥iv.

As this solution is caustic, the spray should not be directed on the eyes or the neighbourhood of the nostrils. The spray should be repeated twice or thrice daily. This is the best treatment for erysipelas in Talamon's opinion. If it is employed from the outset, the cutaneous inflammation yields from the first day and the attack ceases on the fourth day.

In **œdema of the eyelids**, bathing with warm water coloured with Condy's permanganate of potassium fluid, collyria of sulphate of zinc (½-1 grain to an ounce of rose water and a few drops of glycerine), and a blister to the nape of the neck as a derivative are measures, all of which have been recommended by high authorities. Trousseau objected to blistering as establishing a trauma or wound, but Graves and Hudson practised it with good results.

In most cases, the hair should be cut close; in some, the head should be shaved. In the latter event, great care should be taken to avoid hurting the skin, because of the influence of traumatism. For this same reason the vesicles or blebs of erysipelas should not be punctured, unless gangrene threatens.

Sore-throat, laryngitis, and **œdema of the glottis**, are to be prevented or combated as suggested under the heading of scarlatina.

Great tension of the skin is relieved by superficial punctures or incisions and by warm poulticing. In this way, also, the occurrence of **gangrene** may be avoided. Should this serious accident happen, or abscesses form, free incisions should be made, and the parts should be antiseptically dressed and poulticed.

Prof. A. Wölfler, of Vienna, recommends the **application of strips of adhesive plaster** as a mechanical means of checking the spread of erysipelas.

PART III.

THE CONTINUED FEVERS.

PART III.—THE CONTINUED FEVERS.

CHAPTER XXIV.

General Considerations.

Classification into Exanthemata, Continued Fevers, and Intermittent Fevers is non-essential but convenient.—Claims of Typhus and Typhoid or Enteric Fevers to be classed as Exanthemata. Three reasons why they are not so-classed.—Cullen's definition of the Continued Fevers.—Murchison's classification of these Fevers. Objections advanced to certain of his statements.

By no hard and fast lines are the fevers we are about to study separated from the group which has up to this engaged our attention. The **accepted classification** into **Eruptive, Continued,** and **Intermittent Fevers** is one rather of convenience than of absolute necessity. It is, in fact, to a large extent artificial and arbitrary. I have already shown that some authors have, without hesitation, included typhus among the exanthemata—indeed, "Typhus exanthematicus" is one of the names by which this fever is known in Germany[a] as well as in England[b]—it is "Das exanthematische Nervenfieber" of German writers. In 1831, too, Dr. Roupell, of London, wrote of this fever as "Typho-rubeoloid," in allusion, no doubt, to the resemblance of the rash to that of measles. He and Peebles[c] both maintained the right of typhus to rank with the exanthemata.

[a] For example, Hildenbrand, *Ueber den ansteckenden Typhus, Wien,* 1810; and Schulz, *Typhus exanthematicus beobachtet in den Wintermonaten des Jahres,* 1847–48. *Prag. Vierteljahrsschr.* 1849.

[b] For example, C. West: Account of Typhus exanthematicus in St. Bartholomew's Hospital in 1837-38. *Edinb. Med. and Surg. Journ.*, Vol. L., 1838. But see also the *Edinb. Med. and Surg. Journ.*, April, 1840, where Dr. West discusses the very question whether typhus should be classed among the exanthematous Fevers, and decides it in the negative, while employing the term "Exanthematic Typhus."

[c] *Edinb. Med. and Surg. Journ.* Vol. XLIV., 1835.

Even typhoid or enteric fever has been considered an exanthematic fever. In 1699 F. Hoffmann, of Halle, described a fever under the name of *Febris petechizans* vel *spuria*, in contradistinction to *Febris petechialis vera* (or true typhus.) This fever was characterised by an insidious commencement, vomiting and purging, and by the appearance about the seventh day of an eruption on the trunk, consisting of elevated papules, which disappeared completely upon pressure. Is not this an accurate picture of enteric fever?

Why, then, do we **separate** some fevers from the exanthemata and group them together as "Continued Fevers?" Briefly, **for three reasons.**

First, because two of these fevers—simple continued and relapsing fever—throw out no rash at all. Therefore, in no way could they be classed with the exanthemata.

Secondly, because the occurrence of a rash, or eruption, is much less constant in both typhus and typhoid than it is in the case of eruptive fevers, properly so-called. It is true that Murchison says the eruption of typhus is very rarely absent. During twenty-three years it was noted in 93·2 per cent. of the 18,268 cases admitted into the London Fever Hospital. Murchison thinks this estimate too low, and says that in 1864 it was noted in all but 55 out of 2,493 cases, or in 97·77 per cent. But we should not lose sight of an important point bearing on this matter, which is that the cases admitted to hospital are, generally speaking, those in which a positive diagnosis of typhus had been made *before* admission, and most probably because of the presence of the characteristic rash, which Murchison himself says is pathognomonic. The slight and non-maculated cases are left behind in their homes, and so do not share in the elaboration of statistical results. Many years experience at Cork-street Fever Hospital has convinced me that the typhus rash is often absent, at all events in children. Murchison himself admits that, "in children it is oftener absent than in adults."

As for enteric fever, the eruption is by no means invariably present. In the London Fever Hospital, in twenty-three years, it was detected in only 76·92 per cent. of the cases, Murchison remarking as to this, that "the fact of the spots not being observed was,

perhaps, due to their not having been looked for with sufficient care." According to Edmund Parkes,[a] spots are absent in 20 per cent. of the cases of enteric fever—a result which very fairly agrees with the statistics at the London Fever Hospital.

Thirdly—Whereas the rashes of smallpox, measles, scarlatina, and erysipelas are shown, both clinically and histologically, to be true inflammations of the skin (*dermatitides*), the same cannot be alleged of either the rose-spots of typhoid or the maculæ and petechiæ of typhus. Both rose-spots and maculæ do not as a rule pass the stage of hyperæmia, and the petechiæ are, in Murchison's own words, "due to an infiltration of dissolved hæmatin into the tissue of the cutis."

Cullen's definition of the continued fevers runs as follows:—"**Febres, sine intermissione, nec miasmate paludum ortæ, sed cum remissionibus et exacerbationibus, parum licet notabilibus, perstantes: paroxysmis quovis die binis**" ("Fevers which run their course without intermission, and do not arise from a paludal miasm, but continue with remissions and exacerbations, which are not, however, very marked: two paroxysms being observed in the course of any given day of twenty-four hours.")

Murchison's **Classification of the Continued Fevers** of Great Britain and Ireland is the following:—

A. **Non-Specific.** I. *Simple Fever*, caused by exposure to the sun, fatigue, surfeit, &c.

B. **Specific**
- II. **Endemic** (*Enteric*, *Typhoid*, or *Pythogenic*), the poison being contained in drinking water, emanations from sewers, &c.
- III. & IV. **Epidemic**
  - *Typhus*, caused by contagion or by the concentrated exhalations from squalid human beings.
  - *Relapsing Fever*, arising from contagion or famine.

In this Table, two points may fairly be criticised as being too dogmatic and certainly open to question. These are—the unqua-

[a] *Association Medical Journal*, 1856, p. 993.

lified use of the term "Non-specific" in connection with the intimate nature of Simple Fever, and the doctrine that typhus may arise *de novo*, that is, may be spontaneously generated by the concentrated exhalations from squalid human beings.

If simple fever is absolutely non-specific in its origin, it should find no place among a group of acute infective and essential fevers, which from the very nature of the case are to be considered as specific.

Again, the doctrine of the spontaneous origin of a febrile disease like typhus lands us in the midst of greater difficulties than those which such a doctrine is intended to solve. It is opposed to all analogy and, in fact, it is quite unnecessary if we bear in mind the marvellous vitality of "resting spores" and the influence which a powerful combination of predisposing causes may have in calling into action the proper exciting cause of a catching or infectious (contagious) disease like typhus.

These questions will come up for further consideration in our study of each of the Continued Fevers in the succeeding chapters. The order in which I propose to describe the different fevers is—(1) **Simple Fever**, (2) **Typhus**, (3) **Relapsing Fever**, (4) **Typhoid** or **Enteric Fever**.

CHAPTER XXV.

Febricula, or Simple Fever.

Nomenclature.—Definition.—Ætiology and History.—Probably of specific origin, like the other fevers—probably auto-infective—Clinical History: Four Forms—1. Ephemera; 2. Synocha, or Acute Inflammatory Fever; 3. Ardent Continued Fever of the Tropics; 4. Asthenic Simple Fever.—Diagnosis.—Prognosis.—Pathology.—Treatment.

Nomenclature.—Simple Continued Fever (*Lat.* Febris Continua Simplex, Lieutaud, 1776). *Syn.*—Acute Continual Fever (Langrish, 1735); Simple Inflammatory Fever (Huxham, 1739; Fordyce, 1791). *Germ.* Entzündungsfieber. *Fr.* La Fièvre Inflammatoire. *Ital.* Febbre Inflammatoria. *Gk.* Καῦσος (Hippocrates). *Spanish*, La Calentura (Piquer, 1751). Ardent Fever [of the Tropics] (Burnett, 1812; Sir Ranald Martin, 1841; Copland, 1844); Synocha (Cullen); Fièvre Synoque (Davasse, 1847).

From its **duration**, this fever received many names, of which the chief are:—Synocha septimo die soluta (Hoffmann, 1700); Febris ephemera (Riverius, 1623; Sennertus, 1641; de Sauvages, 1760); Ephemera simplex (Boerhaave, 1738); Diary Fever (Strother, 1728). *Germ.* Das eintägige Fieber. *Fr.* Fièvre Éphémère. *Ital.* Effimero. *Span.* Efemera.

From its **causes**, it was described by Boissier de Sauvages (1760) as "Ephemera à Frigore" and "Ephemera à Calore;" and by Scriven, in 1857, as "Sun Fever."

The term "**Febricula**" is, of course, the diminutive of "Febris," and has reference to its comparatively trivial nature and short duration.

It is also sometimes called "**Herpetic Fever**," from the frequency with which crops of herpes break out during its course, especially about the nostrils and mouth, and on the lips.

Definition.—An acute Fever, said to be non-contagious, occurring sporadically in general, but occasionally assuming an epidemic

form, arising from exposure to the sun, over-fatigue, a surfeit, inebriety, running a course of from one to seven, or rarely ten days, without eruption, but with marked febrile symptoms, tongue furred but moist; terminating critically with profuse sweating; often accompanied by herpetic eruptions; rarely fatal unless in the Tropics; no specific lesion in fatal cases.

Ætiology and History.—This form of fever has been identified since the time of Hippocrates. In the paroxysm of any fever there are three stages—(1.) The *cold stage*, ushered in by rigors (also called the pyrogenetic stage); (2.) the *hot stage*, or stage of reaction (the fastigium, or acme); and (3.) the *sweating stage*, or crisis (defervescence). In simple fever these may all occur and be completed within twenty-four hours. Hence such names for the disease as "ephemeral[a] fever," "diary fever," "das eintägige Fieber," and so on.

In 1728, Strother described a "diary fever," distinct on the one hand from "spotted fever" (typhus), and on the other from "slow fever" (typhoid). The diary fever, he said, resulted "from hard drinking, or too great heat of the sun, or from a little cold;" it needed "little help from physic," and it did "not last above three or four days."

There can be no doubt that the term "Simple Fever" has become a refuge of many cases of mild typhus (*T. levissimus*, of Hildenbrand) and ill-defined typhoid fever or relapsing fever without the relapse, and so the separate identity of febricula has been called in question. But, as we have seen, from the earliest times the existence of this disease has been recognised. Its ordinary causes are exposure to great heat or cold, surfeit or inebriety, gastric derangement, imperfect excretion, and mental or bodily fatigue.

It is probable, but not yet proved, that simple continued fever may result from **some specific contagion** as yet not isolated. Analogy, in the first place, points in this direction. Next, although this fever is a sporadic disease, as it is commonly observed in temperate climates, yet epidemics of it have been described from time to time—for example, by Ingrassias, of Palermo, as occurring

[a] Gk. ἐπι, *upon*; ἡμερα, *a day*.

in Sicily in 1557; by Hoyer at Mulhausen towards the end of summer in 1700. In India, the "Ardent Fever," which according to Murchison is only the tropical variety of simple fever, often assumes an epidemic form during the hot dry season. Again, the whole cyclical course of the malady almost proves its specific origin, and lastly, one variety of it—the "feverish cold" or "acute catarrh"—is certainly infectious, or "taking," in a high degree.

It may be that in most cases the virus of this form of fever is inbred in the body, rather than introduced into the system from without—that it is an **auto-infective**, or **autochthonous**[a] disease.

Children are most liable to suffer from febricula, adolescents are less so, and the infection becomes more and more infrequent as middle life is reached, and as old age approaches.

Clinical History.—Apart from the **Ardent Continued Fever of the Tropics**, and another variety to which Murchison gives the name of **Adynamic Simple Fever**, simple continued fever shows itself under two principal forms—true **Ephemera**, and **Synocha** or **Inflammatory Fever.**

1. In the first or ephemeral form, the patient is suddenly seized with chills or rigors, followed by quick full pulse, flushed face, dry hot skin, loss of appetite, thirst, and headache. The limbs feel sore, as if bruised, the bowels are confined, and the tongue is coated but moist. The urine is scanty, high-coloured, dense (Spec. Grav. 1025–35), and towards the close of the fever deposits urates (lithates) in abundance. The symptoms pass off in twelve, twenty-four, or thirty-six hours, with profuse sweating, and the patient who has been restless, or, if a child, perhaps delirious, falls into a tranquil sleep.

2. In the synochal[b] form—so-called from its continuance—the febrile movement is more prolonged and the symptoms are more acute and severe, while they belong to the same category as before. From time immemorial a tendency of this fever to terminate on odd

[a] *Autochthonous*, indigenous. From Gk. *αὐτόχθων, sprung from the land itself, of native stock.*

[b] *I.e.*, continued; from Gk. *συνοχή, a holding together.*

days—the 3rd, 5th, 7th, or 9th—has been observed, so that these are sometimes called **critical days.**

Modes of Crisis.—The crisis in ephemera and synocha may take place in one or more of many ways—namely, sweating, copious epistaxis or nose-bleed, hæmorrhage from the womb or rectum, an attack of vomiting, a sudden diarrhœa, diuresis with a dense deposit of lithates in the urine, or the development of a crop of herpetic vesicles on the lips (herpes labialis) or about the nostrils, hence the term "Herpetic Fever."

Simple Fever is not attended by any skin-eruption. In a few cases Davasse[a] observed pale bluish spots—the *taches bleuâtres* of French writers—which are also occasionally met with in typhoid fever, acute pneumonia, and other diseases. There is nothing characteristic about these markings, which are supposed by Strümpell[b] and German writers generally to be connected with the irritation of lice. This view is gaining ground in British Medical literature, for Jamieson, writing in the *British Journal of Dermatology* (Volume I., No. 10), includes among the rarer effects of the presence of pediculi the development of *maculæ cæruleæ* (taches bleuâtres) upon the skin. Facing page 516 of the third edition of Murchison's Treatise on the Continued Fevers there is a beautiful tinted lithograph, representing these taches bleuâtres in enteric fever. In several instances he saw them distributed along the course of the small subcutaneous veins.

The **Ardent Continued Fever** of the tropics is merely an exaggerated form of the synocha of Great Britain and Ireland. Murchison gives a graphic account of it as he observed it among the European troops at Calcutta in 1853 and in Burmah in 1854. In many cases the symptoms commenced immediately after incautious exposure to the direct rays of the sun. Hence Scriven's name for it: "**Sun Fever.**"[c] It appears to be a very formidable fever indeed. About the fourth or fifth day there was often acute

[a] *Des Fièvres Éphemère et Synoque.* Paris, 1847. P. 23.

[b] *Text-book of Medicine.* By Adolf Strümpell. English Translation. London: H. K. Lewis. 1887. Page 16.

[c] J. B. Scriven: *On Indian Fevers.* Indian Annals of Medical Science, No. 8, 1857.

delirium, followed by more or less unconsciousness, contracted pupils, and sometimes complete coma. Between the sixth and the ninth day, death took place by coma, or there was a copious perspiration, followed by a rapid fall of the pulse, an increased flow of urine, an abundant deposit of urates, and convalescence. The subsidence of the fever was, however, occasionally followed by sudden, or even fatal collapse.[a]

The **Adynamic** or **Asthenic Simple Fever** may run on for two or three weeks, with increasing weakness. Murchison frequently observed attacks of this kind after great mental or bodily fatigue, and Alfred Hudson[b] had a similar experience. "At the same time," says Murchison, "it must be remembered that cases of this sort are never fatal, and that enteric fever often assumes characters very like those now described."

Diagnosis.—This is often a matter of difficulty, or even impossible. From **typhus** and **typhoid fevers** it is ultimately distinguished by its short duration and the absence of an eruption. An outbreak of herpes on the face about the fifth day of a feverish attack would suggest simple fever in the absence of **pneumonia** or of typhus. Resembling the first paroxysm of **relapsing fever** by its intensity, simple fever is distinguished from that malady by the comparative insignificance of the muscular and arthritic pains which accompany it, and by the absence of enlargement of the spleen and liver, and of jaundice. Besides, it occurs at times and in places other than those selected by relapsing fever.

Prognosis is almost invariably **favourable** in this country, unless some complication should arise. The ardent fever of the tropics is stated to be a serious and often fatal disease.

Pathology.—There is **no special lesion.** Murchison observed **great congestion** of all the internal organs in the fatal cases of ardent fever examined in India.

Treatment.—The simple continued fever of this country requires **no special treatment** beyond rest in bed, a suitable diet,

[a] Murchison: *On the Climate and Diseases of Burmah.* Edinb. Med. and Surg. Journ., Jan. and April, 1855.

[b] *Lectures on the Study of Fever.* Dublin: 1867. P. 262.

and attention to the bowels, kidneys, and skin. As to the ardent fever of the tropics, all writers on Indian diseases advise venesection or leeches to the head, followed by cold effusion, the continued application of cold to the shaven scalp, purgatives, and diaphoretics.[a] For the asthenic form Murchison recommends quinine and the mineral acids, with a nutritious diet and wine.

[a] Cf. Morehead: *Clinical Researches on Diseases in India*, 1860. Second Edition. Page 166. Also, Sir Ranald Martin: *Influence of Tropical Climates*. 1856. Page 208.

CHAPTER XXVI.

Typhus Fever.

Nomenclature — Literature — Definition — Geographical Distribution—Ætiology—Exciting and Predisposing Causes—Facts known relative to the Specific Poison of Typhus: 1. Modes of infection; 2. Its striking distance not great; 3. Poison readily absorbed by "fomites;" 4. Period of infectiousness: Convalescence; 5. Non-inoculable; 6. One attack confers immunity; 7. Of light specific gravity; 8. Destroyed by dry heat; 9. Typhus not an epizoötic—Murchison's doctrine of the spontaneous generation of typhus—Proofs of its infectiousness—Arguments for and against its spontaneous origin—Predisposing Causes: Sex, Age, Season, Temperature and Moisture in the Atmosphere, Occupation, Idiosyncracy, Intemperance, Bodily Fatigue, Mental Fatigue and Depressing Emotions, Previous Illness, Recent Residence in an infected district, Overcrowding and defective Ventilation, Destitution and deficient Alimentation—Conclusions.

Nomenclature.—No fewer than 98 names for this form of Continued Fever are specified by Murchison in his elaborate account of the disease. Of these I can here give only a selection:—

Typhus.—(Boissier de Sauvages, 1760; Cullen, 1769; and all modern British writers) Gk. τῦφος, *smoke*, *mist*, *fog*. The term was employed by Hippocrates to define a confused state of the intellect, with a tendency to stupor ("*stupor attonitus*"), and in this sense it is aptly applied to typhus with its slow cerebration and drowsy stupor.

Previous to the time of Boissier de Sauvages (1760) typhus was known as "Pestilential" or "Putrid Fever," or by some name suggested by the eruption, or expressive of the locality in which it appeared, as "Camp," "Jail," "Hospital," or "Ship Fever" (Murchison).

From its **contagious nature** it was called "Parish Infection" (English Bills of Mortality, 1600–1700), "Infectious Fever" (Lind, 1763), "Contagious Fever" (Bateman, 1818), "Tifo contagioso" (Rossi, 1819), "Der ansteckende Typhus" (Hildenbrand, 1810).

From its **prevalence in epidemics**, it received the name of "Febris epidemica" (J. Burserius, 1625), "Febbre epidemica" (Rasori, 1813).

From the **characteristic rash**, it was described as "Morbus pulicaris"[a] (Cardanus, 1545); "Febris petechialis"[b] (N. Massa 1556; Sennertus, 1641, and others), "Febbre petecchiale" (Rasori, 1809); "Spotted Fever" (Strother, 1729; Short, 1749); "Das Fleckenfieber" (Reuss, 1814); "Petechial Fever" (Peebles, 1835); "Exanthematic Typhus," "Petechial Typhus," "Typho-Rubeoloid" (Roupell, 1831.) Hirsch[c] informs us that Fracastori, who flourished at Verona from 1483 to 1553, and whose most valuable work, "De Morbis Contagiosis," contains the first description of typhus, based on his Veronese experiences, mentions the epidemic under the name of "Lenticulæ" (freckles), "Puncticula," or "Peticulæ," as a contagious disease indigenous in Cyprus and adjoining islands, well known to the older physicians, but now observed for the first time in Italy. Fracastori's account, "*De curatione febrium quæ Lenticulæ vocantur*" begins as follows:—"Nunc de particularibus magis agamus, ac primum de curatione earum febrium pertractemus, quæ, quòd non vere pestilentes sunt, malignæ tantum dicuntur, quales illæ fuere quæ annis 1505 et 1528 tam large apparuere, quas alii Lenticulas, alii puncticula, vel peticulas appellavere: de quibus diligentissime agendum videtur, quoniam sæpe per epidemias revertuntur, sæpe sine iis enascuntur, ac nunc etiam per Italiam visuntur, certis autem regionibus maxime familiares."[d]

From the **presence of cerebral symptoms** it was called "Typhus Comatosus" by de Sauvages in 1760, and "Brain Fever" by various authors.

From the **tendency to prostration** which accompanies typhus, Pinel, in 1798, described it under the names "Fièvre Ataxique" and "Fièvre Adynamique."

[a] "*Of*," or "*belonging to, fleas*," from Lat. *pulex, a flea*.

[b] From Italian *petecchio, a flea-bite*.

[c] *Handbook of Geographical and Historical Pathology*. New Syd. Soc., 1883. Vol. I., page 548.

[d] *Hieronymi Fracastorii Veronensis Operum Pars Prior*. Genevæ: Typis Jacobi Stoer. MDCXXXVII., page 228.

From its **prevalence in time of war**, it was called "Pestis Bellica," "Morbus Castrensis" (Sennertus), "Typhus Castrensis" (de Sauvages, 1760), "Camp Fever" (Grant, 1775), "Die Kriegspest" (Hufeland and Reuss, 1814). It was the first and chief of the "*Leaguer Sicknesses*" of the old Irish chroniclers.

From its **prevalence in prisons, hospitals, and ships**, it received among other titles those of "Jayl Fever" (Pringle, 1750; John Howard, 1784), "Typhus Carcerum" (de Sauvages, 1760), "Maladie des Prisons," "Febris Nosocomialis" (Burserius, 1785), "Fièvre des Hôpitaux," "Ship Fever" (Lind, 1763), "Infectious Ship Fever" Blane, 1789).

From its **supposed mode of origin from overcrowding**, Laycock, in 1861, invented for it the name "Ochlotic Fever" (*Gk.* ὄχλος, *a crowd*).

An old Irish designation for it was "Irish Ague,"[a] and in Ireland also it was often called "Catarrhal Typhus," from the common presence of bronchial catarrh as a complication. In Germany it is sometimes called "Der irische Typhus," because it used to be so prevalent in Ireland.

The synonyms for typhus most commonly used in the principal modern European languages are as follows:—*Germ.* Fleckfieber, Flecktyphus; *French*, Typhus Exanthématique; *Ital.* Dermo-tifo, Typho Esantematico; *Spanish*, Tifus, "El Tabardillo" or "Tabardiglio" (from *tabardo*, a cloak of dark cloth worn by the peasantry in Spain); *Dutch*, Vlekkoorts, Kwaadardigekoorts (*i.e.*, malignant fever); *Swedish*, Fläcktyfus; *Norwegian* or *Danish*, Nervefeber, Exantematisk Tyfus.

Definition.—An acute, specific, highly infectious fever, which prevails in epidemics, particularly in times of destitution and in the presence of overcrowding with deficient ventilation. It is characterised by a sudden onset with marked nervous symptoms, rheumatoid pains, rigors, and headache. A peculiar rubeoloid rash appears most commonly on the fifth day of the disease. This consists of slightly elevated spots, at first deleble on pressure (*maculæ*), after-

[a] *Cf.* Dr. Gerard Boate, Physician to Cromwell's Army. *Natural History of Ireland.* Dublin. 1642.

wards persistent and darker (*petechiæ*). There is early prostration of both nervous system (*ataxia*) and muscular system (*adynamia*). In the second week delirium is commonly present, sometimes of an acute and noisy type, but oftener of a low wandering kind, and there is a tendency to stupor or coma. The fever terminates by a crisis which generally happens on or about the fourteenth day. In fatal cases no specific lesion is found beyond a widespread congestion and "dissolution of the blood."

Literature.—Among authors who have described this fever, Sir John Pringle[a] occupies a foremost place. He gave an excellent description of the epidemics observed in the British Army in 1742 and 1745. Von Hildenbrand's classical work on Contagious Typhus[b] gives a complete account of the epidemic during time of war. The work of Rasori[c] is interesting from a historical point of view. An excellent account by Virchow himself of the typhus of Upper Silesia is to be found in the second volume of Virchow's *Archiv* for 1849, and equally valuable is Lindwurm's description of typhus in Ireland.[d] The best general descriptions are those of Murchison,[e] Griesinger,[f] and Lebert.[g] The relations and conditions of the spread of the disease are ably discussed by A. Hirsch in his "Handbook of Historical and Geographical Pathology."[h]

Geographical Distribution.—"The history of typhus," writes Hirsch,[i] "is written in those dark pages of the world's story which tell of the grievous visitations of mankind by war, famine, and

[a] *Observations on Diseases of the Army.* London, 1752. Also *Observations on the Nature and Cure of Hospital and Jail Fevers.* London, 1750.

[b] *Ueber den ansteckenden Typhus.* Wien. 1815.

[c] *Storia della Febr. petech. di Genova.* Milano, 1813.

[d] *Der Typhus in Irland.* Erlangen, 1853.

[e] *Text-Book of the Continued Fevers of Great Britain.* London: Longmans, Green & Co. Third Edition, 1884.

[f] *Archiv. der Heilkunde*, 1861. Vol. II., p. 557, and *Virchow's Handb. der speciellen Pathologie und Therapie.* II. 2 Abth. Art., "Infections Krankheiten."

[g] Von Ziemssen's *Cyclopædia of the Practice of Medicine.* Art.. "Typhus Fever."

[h] New Syd. Society. London : H. K. Lewis.

[i] *Handbook of Geograph. and Hist. Pathology.* New Syd. Soc. 1883. Vol. I., page 545.

misery of every kind." Typhus from time to time has prevailed in all parts of Europe, but it has its peculiar habitat in Great Britain and Ireland, and in Russia. It has been very infrequent in France of late years. The Irish race seems to be especially prone to the disease, most probably from the habits and comparative poverty of this people. "It is a fact," says Graves,[a] "that typhus is more prevalent in this country (Ireland) than in any other European nation." Popham[b] says it "smoulders on until some spark kindles it into a flame."

In the United States and British North America, typhus has prevailed epidemically at various times. It is hardly known in Australia, New Zealand, India, Africa, or the tropical parts of America. Murchison is of opinion that the so-called "Oriental Plague" ("Bubonic Fever") is perhaps the typhus of warm climates. Typhus is mostly a disease of the Temperate and Cold Zones—a fact which is in some measure, at all events, brought about by the mode of living adopted by people living in cold climates. They congregate together in badly ventilated houses rather than live an open-air life. "The idea that *overcrowding in filthy and unventilated rooms affords the essential condition for the development of typhus-foci and for the spread of the disease*, has been completely borne out by the experience of all times"—these are Hirsch's own words.

Epidemics of typhus are yearly becoming more and more rare. The most recent recorded outbreak is one which occurred in Sheffield in 1890, and was described by Dr. Thomson in *Public Health* (Vol. III., p. 17.)

Ætiology.—This subject will be most conveniently considered under the usual headings—the **exciting** and **predisposing causes** of the fever.

I. In all cases, without exception, typhus arises from the reception into the system of a **specific poison**—the virus, or contagium of the disease. Of the intimate nature of this poison we are still profoundly ignorant.

[a] *Lon. Med. Gazette.* January 19, 1837. Page 571.
[b] *Dubl. Quarterly Journal of Med. Science.* May, 1853. Vol. XV., page 290.

Dr. Cayley, the Editor of the third edition of Murchison's "Treatise on the Continued Fevers of Great Britian," says, that "judging from analogy with relapsing fever and other contagious diseases, it seems probable that typhus is due to a specific microbe which requires conditions of overcrowding and imperfect ventilation to develop its virulent and contagious properties."

In 1868, Hallier[a] announced his discovery of a typhus fungus (*Rhizoporus*), but it has not been confirmed. In 1873, Zuelzer[b] claimed to have obtained "positive results" from infection-experiments in animals, but there is no evidence that the disease produced in the animals experimented on was typhus. In 1883, Mott[c] described actively motile dumb-bell cocci in the blood, and plugs of cocci in the lymphatics of the heart, in cases of typhus fever. Lastly, Hlava,[d] in 20 out of 33 cases of typhus examined after death, and in 2 out of 10 cases examined during life, found a peculiar, well-defined bacterium, to which he gives the name of *Streptobacillus*. It was present only in the blood. Injections into mice, rabbits, cats, and pigeons were followed by no result. Young pigs responded with acute febrile manifestations and a red exanthem. Hlava thinks it not improbable that the streptobacillus in question may be the cause of typhus fever, although absolute proof is wanting. Nothing definite, however, has as yet resulted from these and similar researches.

Nevertheless, there are certain interesting facts which we do know respecting the typhus poison.

1. **Actual contact** with the sick is **not essential** for the transmission of this fever. The *materies morbi* is cast off by the breath, from the skin, possibly in the evacuations from the bowels. It is then conveyed through the air or by fomites, and inhaled or perhaps swallowed, so finding its way into the blood of fresh victims.

[a] *Virchow's Archiv.* Vol. XLIII., page 268.

[b] *Vierteljahrsschr. für die med. Wissensch.* No. 36.

[c] *Brit. Med. Journal.* Cf. *Manual of Bacteriology.* By E. M. Crookshank. Second Edition, 1887. Page 214.

[d] *Sborník lékárski.* Praze. *Archives bohémes de Médecine.* Vol. III., No. 1. Sajous' *Annual of the Universal Medical Sciences.* 1891. Vol. I., H.–56.

2. The **striking distance** of typhus is **not great**, provided only a fresh air space separates the sick from the healthy. In other words, the poison of typhus is most readily neutralised on coming into contact with fresh air—probably by oxidation. Unlike the contagia of smallpox and of scarlet fever, the poison of typhus does not cross open spaces.

3. Typhus poison is readily **absorbed** by articles of clothing, bedding, furniture, and beams of wood. It may even adhere to the walls of badly-aired buildings. It may, therefore, be carried by **fomites**, such as garments impregnated with it—especially woollen fabrics, and—as pointed out by Haller, of Vienna—*dark*-coloured rather than *light*-coloured clothing. It may be mentioned that Stork found that in dissecting rooms dark clothes acquired the cadaveric odour sooner than light ones, and ascertained by experiments that the absorption of odours is regulated by the laws which govern the absorption of light.

4. The consensus of opinion is that the poison of typhus is not thrown off by the patient to any great extent in the earlier stages of the fever—probably not until after the ninth day. The late Dr. R. Perry,[a] of Glasgow, was the first to advance the opinion that the **period of convalescence** is the **most infectious** in typhus, and Murchison's observations at the London Fever Hospital confirm Dr. Perry's at the Glasgow Royal Infirmary.

5. The experiments of Dr. O. Motschutkovsky[b] show that neither typhus nor enteric fever can be communicated to man by inoculating the blood—this fever is, therefore, **not inoculable.**

6. One attack of typhus **generally confers immunity** from a second, but there are rare yet notable exceptions—for example, Murchison himself suffered from two severe attacks, each attended by the characteristic eruption. An abortive attack ("*typhisation à petite dose*," Jacquot) probably protects the system no more than an abortive attack of scarlatina.

7. According to Haller, the contagium of typhus is **lighter than**

[a] *Letter on Typhus Fever. Dubl. Quart. Journ. of Med. Science.* Vol. X. 1836.

[b] *Experiment. Stud. über die Impfbarkeit des Rückfallstyphus. Centralblatt für die med. Wissenschaft.* 1876. No. 11.

atmospherical air. His experiment was to admit ozone into the atmosphere of a typhus ward, when it was found to become lost first in the air near the ceiling. He found that typhus spread to the upper stories of a hospital when it was treated on the ground floor, whereas the patients in the lower stories enjoyed perfect immunity when the typhus patients were treated on the top landing. This was so well known at the Meath Hospital that, when I was a student there a quarter of a century ago, the typhus cases were treated in wards at the top of the General Hospital. This plan, of course, has been long since abandoned, as the construction of isolated Epidemic Wards in the grounds of the hospital made it unnecessary to treat infectious fevers in the main building.

The **specific gravity**, then, of typhus contagium is **light.** It is a **volatile poison.**

8. There is strong presumptive evidence that **dry heat** is a **powerful disinfectant** agent in typhus. In 1831, W. Henry[a] made a series of experiments of "doubtful propriety" (Murchison) which went to show that articles of clothing impregnated with typhus poison are rendered innocuous by exposure for some hours to a temperature of 204° Fahr.

9. There is as yet **no evidence** that **typhus is communicable to the lower animals**, or that a disease identical with human typhus prevails amongst them.

While admitting that the primary exciting cause of typhus is a specific poison, Murchison was of opinion that this poison is not only derived from persons previously infected (contagion), but also may be **generated independently.** The conditions under which he supposed that the poison is developed *de novo* are: overcrowding of squalid human beings and deficient ventilation; in other words—the poison is generated by the concentration of the exhalations from living beings, whose bodies and clothing are in a state of great filth.

That typhus is **eminently infectious**, or "taking," few if any will be found at the present day to deny. Probably it is at once the most infectious and the most preventable of the fevers. Murchison

[a] *Philosophical Magazine.* Vols. X. & XI. 1831.

sums up the evidence of its contagiousness by stating five facts in relation to this point. These are the following:—

1. When typhus commences in a house or district, it often spreads with great rapidity.

2. The prevalence of typhus in single houses or in circumscribed districts, is in direct proportion to the degree of intercourse between the healthy and the sick.

3. Persons in comfortable circumstances, and living in localities where the disease is unknown, are attacked on visiting infected persons at a distance.

4. Typhus is often imported by infected persons into localities previously free from it.

5. The contagious nature of typhus is indicated by the success attending the measures taken to prevent its propagation, more especially the early removal of the sick.

So far there is no room for controversy, but it is otherwise when Murchison advocates the doctrine of the spontaneous or *de novo* origin of typhus. In support of this doctrine he appeals to the arising of sporadic cases in the absence of any great epidemic, and of outbreaks in public institutions and among isolated bodies of men. As examples of the alleged spontaneous origin of typhus he instances six historical "black assizes," in which a highly pestilential "jail-fever" broke out; various isolated outbreaks of "ship-fever" and of "military fever"; and Sir John Pringle's account of what he called "hospital fever." With singular short-sightedness for him, Murchison observes that down to the commencement of the present century no doubt existed on this matter. "All our great physicians of the past—Huxham, Pringle, Cullen, D. Monro, Blane, Stanger, Bateman, &c., re-echoed the opinion of England's greatest Lord Chancellor, Lord Bacon, and most emphatically declared that typhus often originated *de novo* under the circumstances above specified." He sums up the opinions of these authorities in the quaint but expressive language of William Grant, as contained in his essay on the "Pestilential Fever of Sydenham." According to that author, "the seminium of a pestilential fever" is soon bred by overcrowding and defective

ventilation, which "*seminium* once produced is easily spread by contagion."

Surely the fact that the spontaneous generation of typhus began to be questioned at the commencement of the present century, should have suggested to so acute an observer as Murchison the reflection that this shaking of a primeval faith was caused by an increasing knowledge of the nature and ætiology of typhus. He adduces much stronger evidence in support of his views when he quotes Jacquot, the French medical historian of the Crimean War, who wrote[a]:—"Pas une contestation ne s'est élevée au sujet de la cause du typhus; les faits sont clairs et parlants; le typhus spontané est dû aux miasmes humains qui s'exhalent au milieu de l'agglomération, de l'encombrement, etc. *On peut faire naître le typhus à volonté, pour ainsi dire.*"

In support of this doctrine, Murchison also quotes Dr. Alfred Hudson in these words:—"While in Ireland the power of *ochlesis*[b] to generate the poison of typhus without the introduction of extraneous infection is attested by no less authority than that of Dr. Hudson." The original words in Hudson's book are:—"In a few instances these [predisposing] causes are found to act with such intensity as to generate a fever poison without the aid of any exciting or extraneous agency."

These views, curiously enough, receive their death-blow from Murchison himself, when he says:—"Although the nature of the poison of typhus has not yet been demonstrated, *it can hardly be doubted but that it consists of minute particles of living matter.*" It is true that he adds:—"This view, however, is not incompatible with its having an independent origin in overcrowding." Why? Because "the first effect of overcrowding with no ventilation is to cause the respiration of an atmosphere charged with carbonic acid," and "the unrenewed air will become charged, not only with carbonic acid, but also with particles of degraded animal matter, capable, like pus corpuscles, of multiplying in a suitable soil." These, then, are the minute particles of "living matter" which constitute the "contagium vivum" of typhus fever.

[a] *Du Typhus de l'Armée d'Orient.* Paris, 1858.

[b] That is, *overcrowding*, from Gk. ὄχλος, *a crowd.*

I venture to dissent from this doctrine of the *de novo* or spontaneous generation of typhus on the following grounds:—

1. It is **opposed to all analogy**, so far as the specific fevers are concerned. No one suggests that smallpox, or scarlatina, or relapsing fever, arises *de novo*. Why, then, should it be thought that typhus or typhoid does so arise?

2. Notwithstanding what Murchison says on the point, such a doctrine **denies the microbic origin of typhus**, which I am well aware is not yet proved.

3. Typhus **did not arise** in certain **extreme** cases of **overcrowding**, to which Murchison himself refers, such as the "Black Hole of Calcutta," the case of the Irish steamer *Londonderry*, and the tragedy of Ujnala. He explains the failure of typhus to appear in these instances by saying there was not sufficient time for its development.

4. The doctrine is **beset with difficulties** greater than those it is designed to explain. For example, in an account of an outbreak of typhus fever which took place in Bristol during the year 1867, Mr. D. Davies, then Medical Inspector for that city, expresses himself in no uncertain terms on the question under consideration. He remarks[a]:—"I would as soon believe in the spontaneous generation of human beings as I would in the spontaneous generation of typhus fever."

5. The theory is **unnecessary**, if we assume the microbic origin of typhus and remember the vitality of the **resting spores** of pathogenic micro-organisms to which reference has already been made. [See page 16].

Even Dr. Cayley, the able Editor of the posthumous third edition of Murchison's Treatise, dissents from this doctrine, for he comments upon it thus:—"Judging, however, from analogy with relapsing fever and other contagious diseases, it seems probable that typhus is due to a specific microbe which requires conditions of overcrowding and imperfect ventilation to develop its virulent and contagious properties."[b]

[a] *Medical Times and Gazette.* October 19, 1867.

[b] *Loc. cit.* Third Edition. 1884. Page 119.

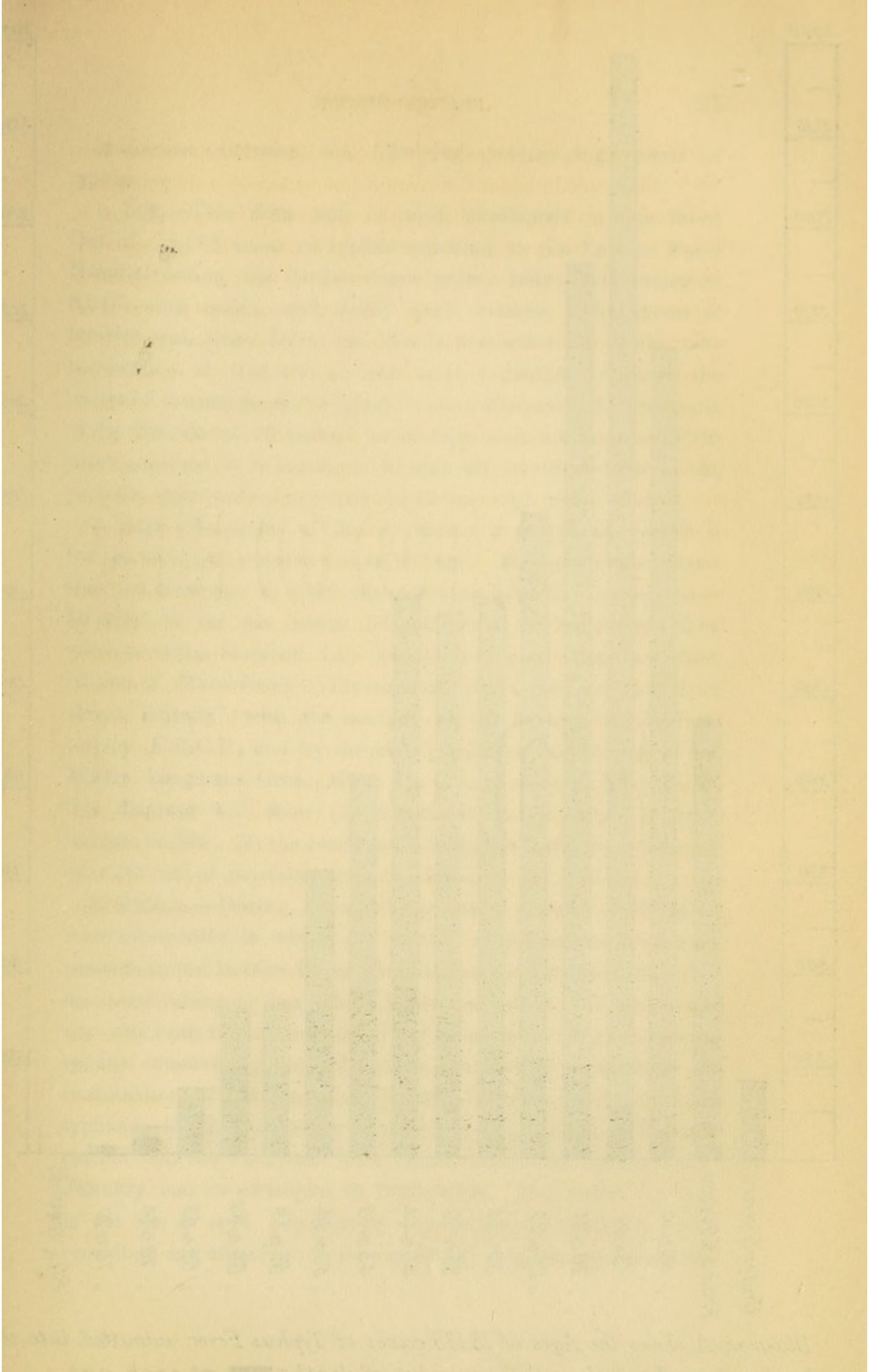
"If we reflect," says Mosler,[a] of Greifswald, "that the contagious matter may remain latent for years in rooms or on furniture, should we not then give up the belief in the spontaneous origin of such a disease? Unsuspected ways in which the poison has successively passed from individual to individual, from the cottage to the palace, are being daily brought to light. Again, every possible means of communication in the whole civilised world must be taken into consideration. Attention is seldom paid to all the dangers of transport. A carrier of the contagium rarely meets with an obstacle in its passage from the sick room to the healthy. Coins and bank tokens never meet with the fate of annihilation. One cannot tell whether the money previously belonged to one who was healthy or diseased. As to paper money, which is composed of organic masses, there can be no doubt but that it might take up infectious matter. Small coins, also, especially those of nickel, which lose their smooth surface when long in circulation, become thickly coated with particles of dirt, and impregnated with infectious matter, particularly as they often serve as playthings for sick children. Having regard to all the possible ways in which transference of the infection may be effected, can we any longer cling to the doctrine of the spontaneous origin of typhus?"

"The hypothesis of a 'spontaneous generation' of infectious diseases," wrote Niemeyer,[b] "in the sense that their cause is a new agent induced by injurious influences is, of course, to be rejected, for in that case we should be inferring a *generatio æquivoca*, which has been disproved."

II. To those circumstances, which in themselves are insufficient to generate the disease, but which render the body more liable or susceptible to the influence of the primary exciting cause—that is, the essential or specific poison, virus, or contagium—and without which the latter would often prove inert, the term "**predisposing causes**" is applied.

[a] *Real-Encyclopädie der gesammten Heilkunde.* Wien und Leipzig: Urban und Schwarzenberg. Vol. VII. Art., "Flecktyphus."

[b] *A Text-book of Practical Medicine.* London: H. K. Lewis. 1880. Revised Edition. Page 615.



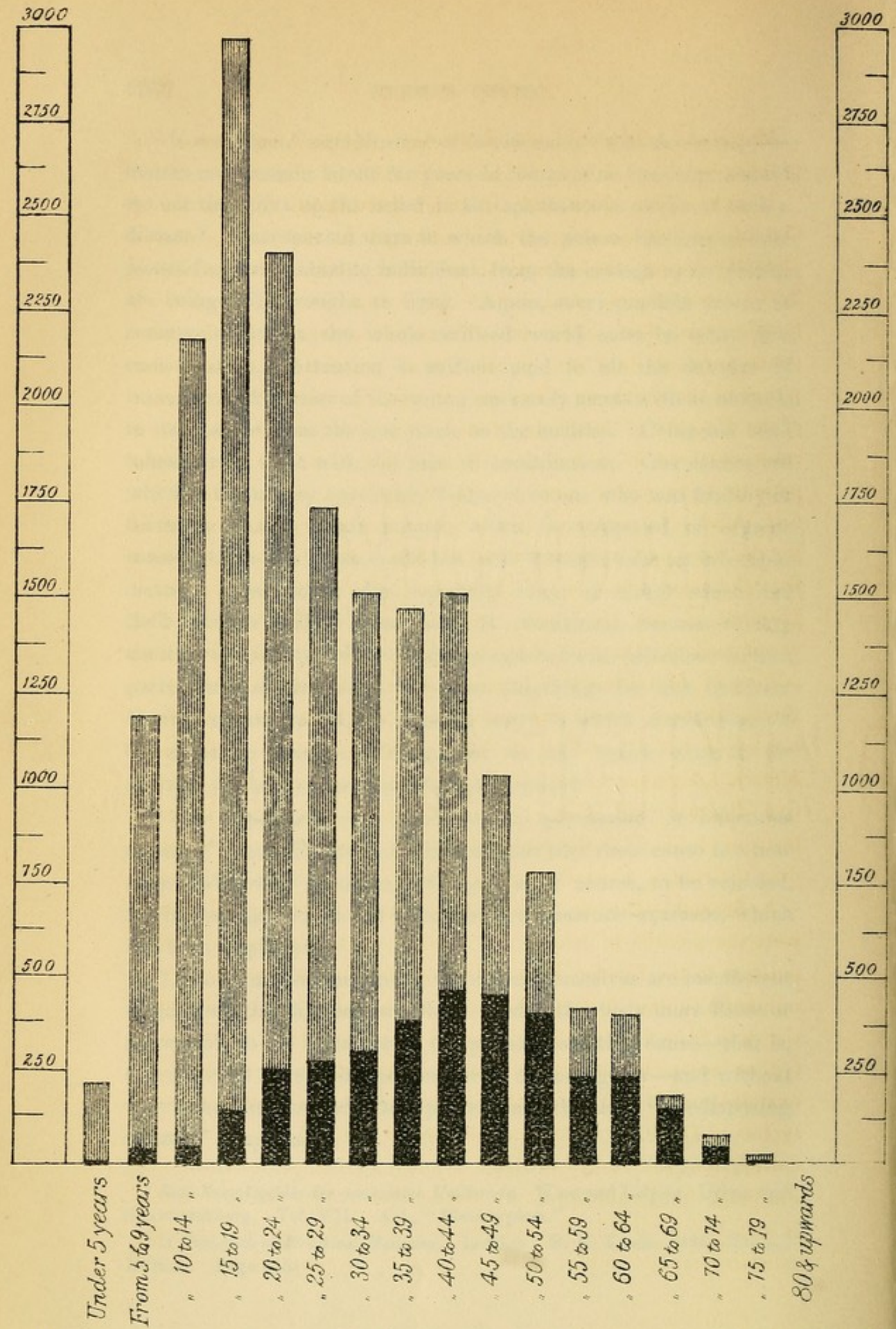


Diagram A, shows the Ages of 18,138 cases of Typhus Fever, admitted into the London Fever Hospital, with the number of deaths ■ *at each age*

Murchison discusses the following predisposing causes of typhus:—

1. **Sex.**—This **does not** in itself **predispose** to this fever. Out of 18,268 cases of typhus admitted to the London Fever Hospital during the twenty-three years, 1848–1870, inclusive, 8,946 were males, and 9,322 were females. The excess of females was, thus, 376; but this is accounted for by the preponderance of that sex in the total population. Lebert, the writer of the article on "Typhus" in von Ziemssen's "Cyclopædia of the Practice of Medicine," found from accurate records of 740 cases observed in epidemics at Breslau that 55·68 per cent. of the patients were males, and only 44·32 per cent. were females.

2. **Age.**—No period of life is exempt from typhus, but it is for the most part a disease of **adult age.** Murchison ascertained that the mean age of 3,456 cases admitted into the London Fever Hospital in the ten years, 1848–57, was 29·33 years. The accompanying Diagram (A) has been copied from the third edition of Murchison's "Treatise on the Continued Fevers of Great Britain," with the sanction of the Editor, Dr. William Cayley, F.R.C.P., and by the kind permission of the Publishers, Messrs. Longmans, Green, & Co. A careful study of this instructive diagram will show (1) the incidence of typhus in each lustrum of life; (2) the remarkable influence which age exercises over the rate of mortality from this fever.

3. **Season.**—During twenty-three years, January and March were the months in which the number of admissions of typhus patients to the London Fever Hospital reached a maximum—the minimum falling in September, August, and July. This distribution was from time to time disturbed by an epidemic, outbreaks of typhus commencing and advancing irrespective of season. An examination of the Registrar-General's Returns of deaths from typhus in Dublin, undertaken many years ago, led me to the conclusion that the death-rate from typhus attains its **maximum in January**, and its **minimum in September.** The reason for this is not far to seek. Typhus is often intimately related to overcrowding, and affections of the respiratory organs are among its

most frequent complications. Hence we should expect to meet with it, especially in the colder seasons of the year. Murchison points out that typhus does not always become more prevalent with the commencement of cold weather, nor does it decline immediately on the advent of summer. He correctly infers from this that the increase of typhus in winter and spring is not so much due to the direct effect of cold as to the **continued overcrowding** and **defective ventilation** of the dwellings of the poor in cold weather.

4. **Temperature and Moisture in the atmosphere** do not seem to have any marked predisposing influence on typhus, notwithstanding the opinion advanced by Dr. T. W. Grimshaw,[a] now Registrar-General for Ireland, in 1866, that a warm moist state of the atmosphere seemed to favour an increase of typhus, whereas dryness with cold had a contrary influence. Murchison was unable to trace any such connection, but points out that exposure to cold and wet, if long continued, depresses the nervous system and so favours the onset of typhus.

5. **Occupation** does **not predispose** to this fever except so far as it involves actual exposure to the poison, as in the case of physicians, medical students, nurses, and laundresses. Dr. Alexander Tweedie considered that **butchers** were particularly exempt from typhus. There is no reason why they should be so, beyond the fact pointed out by Murchison, that they usually have an abundant supply of nourishing food.

6. **Idiosyncrasy.**—By using this term, Murchison wishes to convey that some persons show a complete **natural immunity** from typhus, whereas others have a **peculiar aptitude** for contracting the disease, not once but twice, as happened in his own case.

7. **Intemperance** not only **strongly predisposes** to typhus, but greatly increases its danger. This has been already pointed out.[b] Even a **single act of intoxication** may predispose to this fever. Murchison met with several instances of persons exposed to the

[a] "On Atmospheric Conditions influencing the Prevalence of Typhus." *Dubl. Quar. Journ. of Med, Science*, May, 1866.

[b] See Chapter IV., pages 39 and 42.

poison for months who were not attacked until immediately after a debauch.

8. **Bodily fatigue.**—Whatever lowers vitality and exhausts and debilitates the body, like fatigue, want of sleep, and pain, **predisposes** to typhus.

9. **Mental fatigue and depressing emotions**, such as sorrow, fear, disappointment, and anxiety, have **a like effect.** Cheerfulness and confidence, on the other hand, aid an individual to resist the fever poison.

10. **Previous illnesses** predispose to typhus. A "feverish cold" is sometimes said "to develop into typhus." This is not what really happens; but the catarrh increases the susceptibility of the individual to the fever poison. In hospital practice, convalescents from other diseases are often attacked by typhus if the virus is about. Simple fever and scurvy strongly predispose. Von Hildenbrand's view, put forward in 1811, that typhus rarely attacks phthisical subjects, does not rest on sure ground.

11. **Recent residence in an infected district** greatly increases the risk run by an individual exposed to typhus. Persons continuously so exposed appear to become acclimatised in a greater or less degree. We can imagine that "*typhisation à petite dose*" (to use Jacquot's eloquent phrase), repeated again and again, may in time exhaust the soil for typhus as effectually as one thorough and unequivocal attack of the fever.

12. **Overcrowding** of human beings, **with deficient ventilation, is one of the most powerful** predisponents of typhus. To this fact testimony is borne by all the historians of epidemics of the disease.

13. Lastly, **destitution** and **deficient alimentation** are absolutely **the most powerful** predisposing causes of typhus. It is essentially a poor man's disease. Famine and pestilence "hunt in couples" in this instance, as in many others also.

In conclusion, we may sum up the facts relating to the **ætiology** of Typhus, after Murchison, in the following sentences:—

1. Typhus is due to a specific poison, the entrance of which into the system is the prime exciting cause of the fever.

2. This poison is communicated from the sick to the healthy through the atmosphere, or by fomites; but is rendered inert by free ventilation.

3. The great predisposing causes of typhus are, in the order of their ætiological importance—destitution and defective nutrition, overcrowding, bad ventilation.

4. In the presence of these factors, a small and hitherto inert dose of the fever poison may kindle a serious outbreak of typhus in such a way as to suggest that the fever has arisen spontaneously or *de novo*.

CHAPTER XXVII.

Clinical Description of Typhus Fever.

Stages of Typhus: (1.) Incubation—about twelve days, or less. (2.) Invasion—earliest symptoms referable to the Nervous System. (3.) Nervous Excitement (Earlier Eruptive Stage). Objective Symptoms: typhus rash, maculæ, subcuticular mottling, "Mulberry Rash" (Jenner). Delirium: ferox, tremens, typhomania (Galen). (4.) Nervous Prostration (Later Eruptive Stage)—characterised by ataxia and adynamia. Petechiæ—the "Typhoid State"—its symptoms. (5.) Defervescence or Crisis. Modes of Crisis: sleep, slight diarrhœa, diuresis, perspiration. (6.) Convalescence.—Duration of Typhus.—*Blasting Typhus*, or *T. siderans*.—*Typhus levissimus*.—Relapses.—Temperature in Typhus.—Hyperpyrexia.

We may most fitly consider the **course** of typhus fever as, for convenience, divided into certain stages—those specified by Murchison being the most convenient of all. They are the stages respectively of—(1.) **Incubation**; (2.) **Invasion**; (3.) **Nervous Excitement, Earlier Eruptive Stage**; (4.) **Typhoid State, Later Eruptive Stage**; (5.) **Defervescence**, or **Crisis**; (6) **Convalescence, Nervous Prostration.** "The duration of these stages," says Murchison,[a] "varies in different cases—some may be shortened or altogether absent, and occasionally it may be difficult to say when one stage ends and another begins."

I. **Stage of Incubation.**—In typhus fever there seems to be **no fixed duration** for this stage. In a paper published in the second volume of the "St. Thomas's Hospital Reports" (1871), in which Dr. Murchison collected 31 cases where he had been able to determine the period of incubation, that author arrived at the following conclusions:—

1. The period of incubation of typhus varies in duration in different cases.

2. In a large proportion of cases it is **about twelve days.**

3. In exceptional cases it is longer than twelve days; but it rarely, if ever, exceeds three weeks.

[a] *Loc. cit.* Third Edition. Page 179.

4. In **many cases** (one-third or more) **it is less than twelve days**, and occasionally there is scarcely any latent period, the symptoms commencing almost at the instant of exposure to the poison. It would seem that the poison of typhus may be so concentrated, or the system may be so susceptible to its action, that its effect may be almost instantaneous. Generally, the patients are conscious of the moment when the fever poison enters the system. In my own person, I have often suffered from a "typhus headache" almost immediately after exposure to the poison of this disease.

In one melancholy and tragic instance I had an opportunity of estimating the duration of the stage of incubation in typhus with a certain degree of accuracy. On the night of Wednesday, December 28, 1881, my friend and colleague, Dr. Reuben J. Harvey, Physician to Cork-street Fever Hospital, died on the tenth day of petechial typhus. At the beginning of December Dr. Harvey, then thirty-six years of age, seemed to be in the enjoyment of perfect health. He attended a meeting of the Physiological Society in London on the evening of the 8th, and returned to Dublin on the 9th, travelling all night. On Saturday, the 10th, he attended the wards of Cork-street Fever Hospital, where there was very little typhus at the time. On the morning of the 11th, however, he examined a lad who had been admitted the previous evening, on the eighth day of typhus. This patient he continued to attend daily, until his own illness had lasted at least twenty-four hours.

On December 19, Dr. Harvey did not feel well; next day he suffered from severe headache. On the 22nd, he was too ill to leave his bed. Early on Friday, the 23rd, maculæ appeared (4th–5th day), and ultimately there was a profuse rash, which very soon became petechial. Almost incessant wakefulness was among the first dangerous symptoms; but at a very early stage the heart became weak and its action rapid, and the respirations ran up to 60 in the minute without any pulmonary complication to account for this untoward symptom. The downward progress was swift, and the end came on the evening of the tenth day.

In this case, so pathetic in its sadness, and which lost to Cork-street Fever Hospital the services of a most able, learned, and conscientious physician, the period of incubation could not have exceeded **10 days**, and was, **most probably, 8 or 9 days** in length.

Niemeyer, in his "Text Book of Clinical Medicine," gives two remarkable examples of the contagiousness of typhus, which are also of value as contributions to the evidence as to the duration of its period of incubation. In the year 1854 two typhus patients were received into the Magdeburg Hospital from the prison, which was much crowded. For months previous there had been only a few cases of typhoid fever and not a single case of typhus in his wards. **Eight days** after the reception of these patients, two others who had lain beside them were attacked by the same disease. One of the latter patients had been admitted for intermittent fever, the other for epilepsy. Again, in March, 1855, a tradesman from Heiligenstadt was attacked with typhus while away from home. He was received into Niemeyer's ward, in which no typhus had been treated for almost a year. **Eight days** after the reception of this patient, a blacksmith's apprentice and a mechanic, lying next to him, were attacked by the fever.

In Murchison's second attack of typhus, the incubation stage lasted **exactly 5 days.** Mr. Davies,[a] whom I have already quoted on the subject of the spontaneous generation of typhus, states in the same paper that in 1867 four Norwegian sailors, on the night of the arrival of their ship in Bristol from Onega, visited some typhus fever nests, and all four sickened with typhus **eight days** afterwards.

II. **Stage of Invasion.**—One or two days of slight indisposition, shown by lassitude, vertigo, slight headache, and loss of appetite, may precede the true onset of typhus, which is sudden. The **symptoms**, as usual in specific fevers, are referable to the **nervous system**; they are—*chilliness* or *slight rigors*, *languor*, *frontal headache*, *pains in the back and limbs*, especially the thighs. The chilliness comes and goes, and complete *loss of appetite*, with *constipation* and sometimes *nausea* (but no vomiting), follows. The *tongue* is large, pale, and coated—first with a white, afterwards with a yellowish

[a] *Medical Times and Gazette.* Oct. 19, 1867. Page 428.

brown fur. The *urine* is scanty, high-coloured, and of considerable density—1025–1030. The *pulse* commonly exceeds 100 and is compressible, although rarely it is below the normal rate. The breathing-rate is accelerated in proportion. The *face* is flushed and dusky. The eyelids swell along their edges, the eyes water, and the conjunctivæ are injected. The *expression* at first betokens languor and weariness, but soon becomes dull, heavy, and listless. *Giddiness*, *noises in the ears* (*tinnitus aurium*), and *sleeplessness* are commonly present. If sleep occurs, it is haunted by dreams, the patient talks, and his mind wanders in his sleep. A sense of *complete exhaustion* quickly overwhelms the sufferer, so that by the third day he is fain to take to his bed.

III. **Stage of Nervous Excitement** (**Eruptive Stage.**)—This commonly extends from the appearance of the rash until the commencement of somnolence or stupor. Its leading features are restlessnes, wakefulness, and delirium. During this stage headache gives place to *raving*, and the tongue grows dry and brown, while collections of *sordes* gather on the teeth and gums.

About the fourth or fifth day the **first stage** of the **typhus eruption** may be observed. It is a rash, or exanthem, often closely resembling the rash of measles, and, in consequence, called "morbilliform," "rubeoloid," or "measly." It consists of *spots*, or "**maculæ**," of very irregular size and outline, and of a dirty pink or florid colour, which appear first near the axillæ and on the wrists, then on the sides of the abdomen, afterwards on the chest, back, shoulders, thighs, and arms. They are rarely seen on the face or neck, for two reasons—first, these parts are very vascular, and the general hyperæmia or congestion of the fever conceals the rash; secondly, the rash develops less quickly and less thoroughly in parts freely exposed to the air. When they first appear, and also when they are few in number, as in the milder cases, they are slightly elevated and deleble on pressure, like the velvety papules of measles. They have, however, no defined margin, but merge insensibly into the colour of the surrounding skin. Along with these superficial spots there is a characteristic marbling or mottling of the skin, caused by the presence beneath the cuticle of another crop of

maculæ. Hence the term "**subcuticular mottling.**" The spots and mottling together constitute an eruption to which Sir William Jenner first gave the name of the "**mulberry rash**" of typhus.

Towards the close of the first week, on the 5th day usually, the headache gives place to **delirium**, which is sometimes acute and noisy, like mania (**delirium ferox**); sometimes more like that of excessive alcoholism, being accompanied by muscular agitation and trembling (**delirium tremens**); and sometimes of a low muttering kind (the **typhomania** of Galen.) The nervous excitement is most marked towards evening and at night, prostration taking its place in the morning.

IV. **Stage of Nervous Prostration**—the **Typhoid, Putrid,** or **Malignant Stage** (**Later Eruptive Stage.**)—This is characterised by extreme *nervous prostration* (**ataxia**), and *muscular* and *cardiac weakness* (**adynamia**), defective cerebration, low muttering delirium, stupor, and unconsciousness deepening into coma. The patient lies on his back (*prostrate dorsal decubitus*), moaning or talking to himself incoherently, with a tendency to sink down in the bed. He is utterly indifferent to what goes on around him, looks stupid and unconscious, with injected *ferret-like* eyes, *contracted pupils* (the "**pin-hole pupil**" of Graves), *teeth* covered with *sordes*, and *dry brown-crusted tongue*. *Deafness* is not infrequent. Other symptoms are: *tremors*, *subsultus tendinum*, *spasmodic twitchings* of the face, and even well-marked *choreic convulsions*, or more commonly *picking* or *fumbling* with the bedclothes—the so-called **Floccitatio**[a] or **Carphology.**[b] Obstinate *hiccough* often accompanies these various involuntary movements, and is a very grave sign. Not uncommonly, also, *involuntary evacuations* take place from paresis of the sphincters of the rectum and bladder. The pulse is rapid, small, and soft (112 to 140), and the respirations are shallow, frequent, blowing, and noisy—the "cerebral breathing" of Sir Dominic Corrigan.

Simultaneously with the supervention of the foregoing unfavourable symptoms, the **eruption changes in character,** becoming

[a] Lat. *floccus, a lock* or *flock of wool.*

[b] Greek *κάρφος, a dry stalk*; *λέγω, I gather, pick up, and lay in order.* Galen (A.D. 163) uses the word *καρφολογία, a gathering of chips, &c.*

darker in colour and quite indelible on pressure. The spots are no longer elevated, and in the centre of many of them dark purple or bluish points appear—the **true petechiæ**, which Murchison defines as consisting of an infiltration of dissolved hæmatin into the tissue of the cutis. The peculiarity of typhus, so far as the rash is concerned, consists in an eruption which, in its earlier stage, is a true exanthem due to hyperæmia of the cutaneous capillaries, while in its later stages an escape of blood-pigment into the cutis is substituted for this hyperæmia. The rashes of other diseases may be *accompanied with* the development of petechiæ, but are not, like the macular rash of typhus, *converted into* petechiæ.

The earlier and more marked the "**typhoid state**" just described is, the more severe is the case. The older writers spoke of it as the putrid or malignant state. It is by no means peculiar to typhus. We have seen that it occurs in smallpox and scarlatina, and, indeed, any idiopathic fever, or blood-poisoning, or local inflammation, may pass into this state.

In such a dire strait the patient may lie for many hours, or several days, life trembling in the balance, until the stupor passes into profound and fatal coma, or sudden engorgement of the lungs with asphyxia supervenes, or the heart fails, with coldness and lividity of surface and *profuse sweating*—death ensuing from syncope and coma combined.

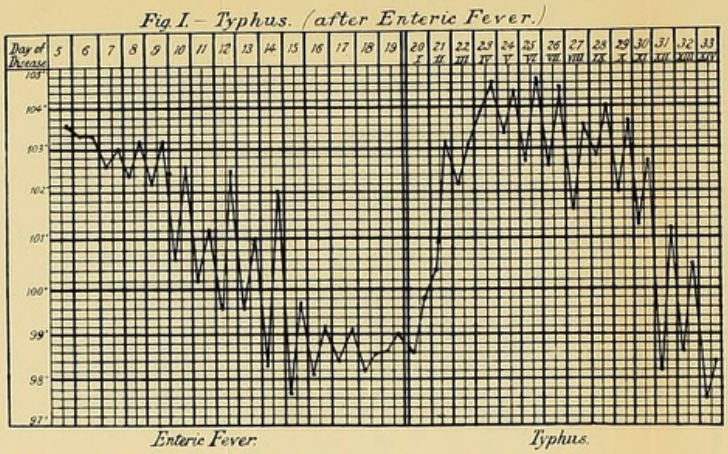
V. **Stage of Defervescence** or **Crisis.**—Happily, such is not always, or even frequently, the end of an attack of typhus. More usually, on or about the fourteenth day there is a more or less sudden improvement. The patient falls into a quiet and prolonged sleep, from which he awakes at first, it may be, bewildered and confused; but he quickly recognises those around him, and for the first time is conscious of his profound weakness. Pulse and temperature fall, the tongue becomes moist and begins to clean, slight perspiration sets in, or the bowels are relaxed, or the urine becomes abundant and deposits lithates (urates) in abundance. In few acute diseases is crisis so marked as in typhus. In 1810, J. von Hildenbrand[a] stated that the disease abated "after a very rapid

[a] *Ueber den ansteckenden Typhus.* Wien. 1810.

Plate V.

CHARTS OF TEMPERATURE RANGES IN TYPHUS.

Fig. I. – Typhus. (after Enteric Fever.)



Enteric Fever. *Typhus.*

Fig. 2 – Adynamic Petechial Typhus.

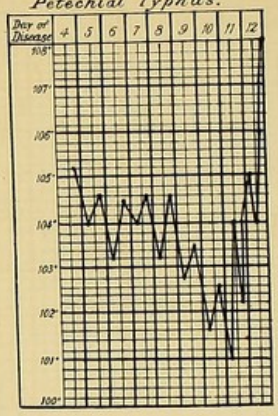


Fig. 3. – Typhus.

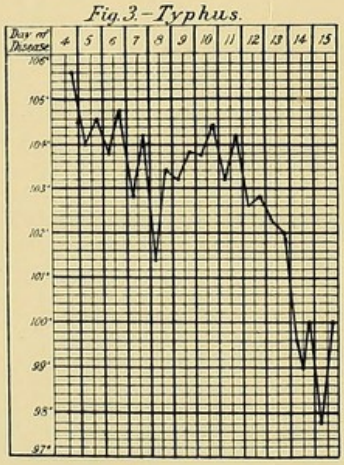


Fig. 4. – Typhus.

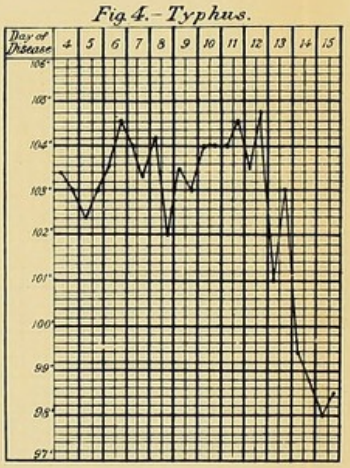
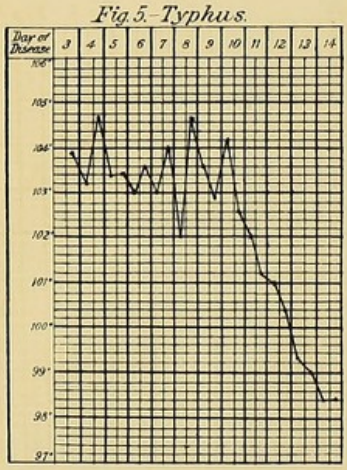


Fig. 5. – Typhus.



fashion." In 1840 Dr. Alexander P. Stewart[a] said that to the *turn* in typhus we might almost apply the Scripture phrase: "At such an hour the fever left him." "La fièvre," said Jacquot,[b] "tombe souvent avec une rapidité étonnante." Lastly, Barrallier[c] observed: "Cette période (de rémission) survient presque brusquement."

The final defervescence, as marked by the thermometer is, to use Wunderlich's expressive phrase, usually "precipitous."[d]

VI. **Stage of Convalescence.**—Once the temperature falls, restoration to health goes on apace. The tongue becomes clean and moist, the appetite is ravenous (**boulimia**)[e] and the bodily powers improve day by day, so that in three or four weeks health and strength are, in uncomplicated cases, fully restored. Typhus but rarely lays the foundation of any permanent organic disease.

Duration of Typhus.—Murchison carefully examined 500 uncomplicated cases which recovered, and 100 fatal cases, with the following results:—The mean duration of the 500 cases was **13·43 days.** In nearly one-half (242) the cases, convalescence commenced on the thirteenth or fourteenth day, and in 384 cases, or in more than three-fourths, on the thirteenth to the sixteenth day inclusive. The mean duration of the 100 fatal cases was **14·6 days,** but when life was prolonged beyond twenty days, the fatal result was due to some complication.

According to Wunderlich,[f] "defervescence most commonly occurs between the thirteenth and seventeenth days, less frequently between the twelfth and thirteenth, and still more seldom at an earlier date."

Dr. T. J. Maclagan[g] investigated 581 uncomplicated cases which recovered in the Dundee Royal Infirmary, and found their mean

[a] *Edin. Med. and Surg. Journal*, October, 1840.

[b] *Du Typhus de l'Armée d'Orient.* Paris. 1858. Page 168.

[c] *Du Typhus epidémique à Toulon.* Paris. 1861. Page 72.

[d] *Medical Thermometry.* New Syd. Soc. 1871.

[e] Gk. *βουλιμία, ravenous hunger;* from *βου-*, used in composition to denote something *huge* and *monstrous* (*βοῦς, an ox*), and *λιμός, hunger.*

[f] *Medical Thermometry.* New Syd. Soc. 1871. Page 330.

[g] *Edin. Med. Journal*, August, 1867.

duration to be **13·39 days**—a value which is practically identical with Murchison's estimate.

Lebert[a] arranges the times of occurrence of the "critical abatement" of the fever of typhus in percentages as follows:

From 6th to 8th day	=	12·3 per cent.	= 71·4 per cent.
„ 10th to 12th day	=	41·9 „	
„ 13th to 14th day	=	29·2 „	
On the fifteenth day	=	8·2 „	= 16·6 per cent.
After „ „	=	8·4 „	

Although the duration of typhus is thus about 14 days, this fever may run a much shorter course. This happens, on the one hand, in malignant cases, which may terminate fatally on the second or third day, or in a few hours. To such cases the name of **Blasting Typhus** or **Typhus Siderans** has been given. This terrible form of the disease is apt to prevail in times of war and destitution. In ordinary times, however, cases of short duration are not uncommon, and are usually mild. Murchison gives the details of cases with eruption which terminated on the tenth, or even as early as the eighth day. In the *Dublin Journal of Medical Science* for July, 1876, I published a series of cases of typhus fever of short duration, all of which terminated favourably. The first case was that of Mary D., a dairymaid, aged twenty-seven, who took ill on Christmas Eve, 1875. She was admitted to the Meath Hospital on December 29 (sixth day), when her skin was dusky, and maculæ were abundant on her back and chest. The lips were covered with sordes. Her tongue was brown at the edges, red and dry in the centre and at the tip. There had been four motions in the preceding 14 hours. P. 119, Resp. 27, Temp. 103·4°. Next day an intense fever odour was observed, the skin was still dusky and maculæ were well marked. Defervescence commenced on this (the seventh) day, the pulse falling from 116 in the morning to 99 in the evening, the respirations from 40 to 35, and the temperature from 103·6° to 103·3°. By January 3 (eleventh day) she was completely fever-free—P. 70, Resp. 20, Temp. 97·8°. The pulse intermitted about twice a minute. The weak state of

[a] Von Ziemssen's *Cyclopædia of the Practice of Medicine*. Art. "Typhus."

Plate VI.

CHARTS OF TEMPERATURE RANGES IN TYPHUS FEVER OF SHORT DURATION.

Fig. 1.

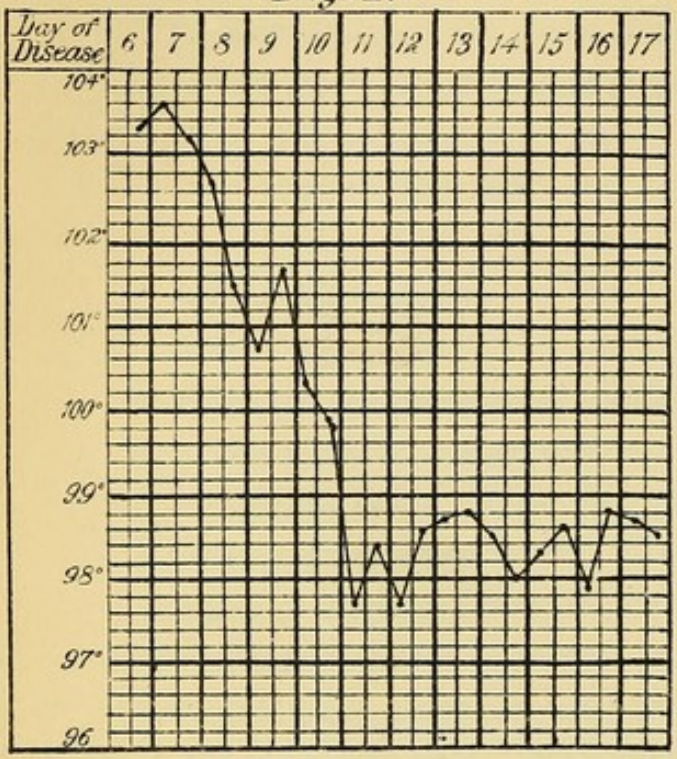


Fig. 2.

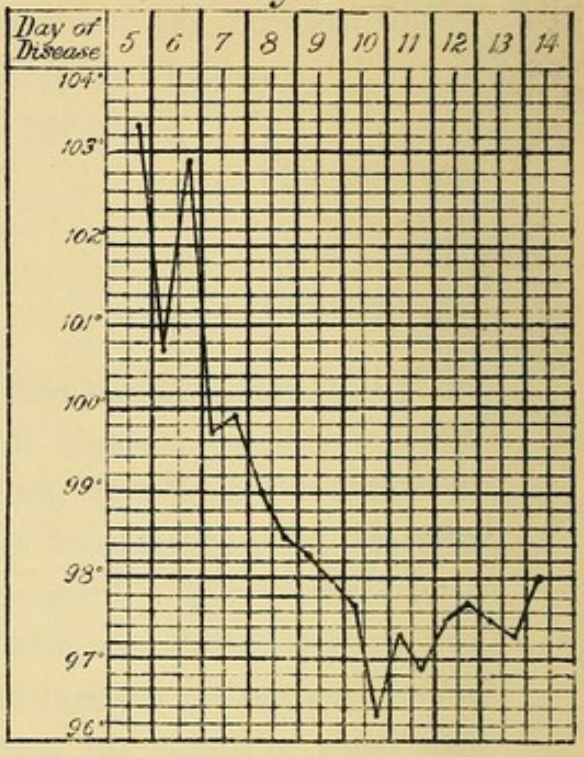


Fig. 3.

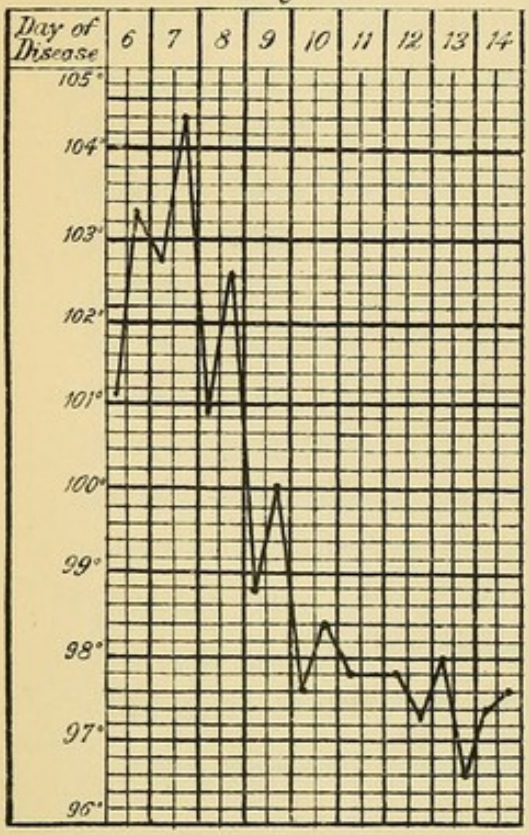
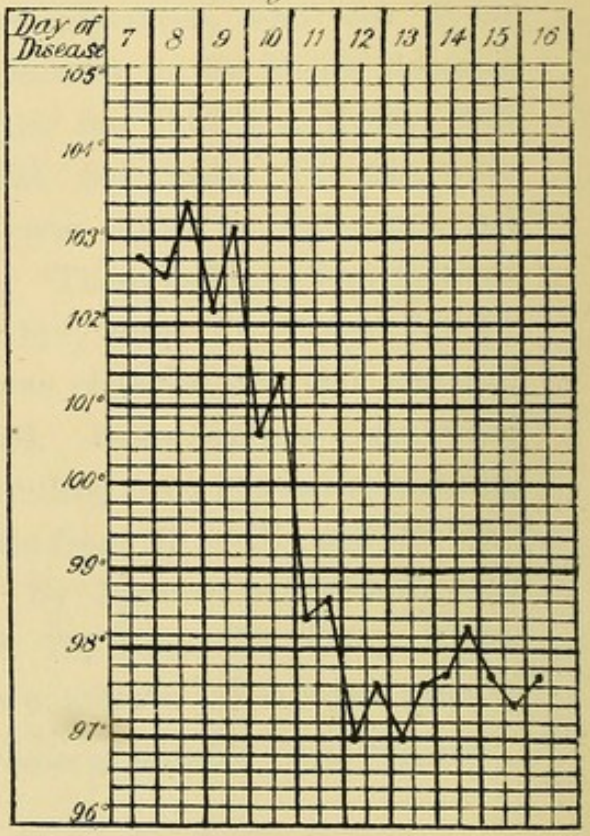


Fig. 4.



the heart required tonics and some stimulants. The temperature charts of this case (Fig. 1) and of three others which were somewhat similar are reproduced in Plate VI.

Traube,[a] of Berlin, in 1853, revived Galen's doctrine of **critical days**, according to which the fever should terminate on one of the *odd* days, and not on the intervening *even* days. Murchison's observations do not support this doctrine in reference to typhus; but both this fever and typhoid or enteric fever tend to terminate at or near the close of a **weekly period**.

Relapses are **extremely rare** in typhus. Neither Sir William Jenner, nor A. P. Stewart, nor Murchison, has ever seen a case of true relapse. Out of 18,268 cases of typhus reported at the London Fever Hospital during 23 years, only one example of a true relapse was observed (by Dr. Buchanan), although in several instances a genuine has been preceded by an abortive attack. Buchanan's case was that of a nurse in the hospital, aged forty-two, who passed through an undoubted typhus fever, lasting two weeks. After a week's interval, a relapse took place, with a recurrence of eruption, lasting upwards of a fortnight. A very similar case is recorded by W. Ebstein,[b] of Breslau, where there was an interval of 25 days between the two attacks.

Temperature.—The facts to be fixed in the memory relative to the behaviour of the temperature in typhus are the following:—

1. A sudden rise of the thermometer takes place at the outset, culminating in a **fastigium** which is generally reached on the evening of the **fourth day**, or earlier, but may be postponed to the seventh day, or later. In this fastigium the thermometer may mark 105°, or upwards, or it may not exceed 103°.

2. A more or less pronounced **remission** of fever or **pseudo-crisis**, occurs at some time between the seventh and tenth days, except in severe cases. In mild cases, this fall of temperature at the beginning of the second week may prove complete and final—a true crisis cutting the fever short, as in the cases of short duration already referred to. On the other hand, in grave cases, a

[a] *Ueber Krisen und kritische Tage.* Berlin. 1852.
[b] *Die Recidive des Typhus.* Breslau. 1869.

gradually rising temperature at this very period may culminate in a **fatal hyperpyrexia.** Of this I will presently quote an example.

3. A renewed rise of temperature in the second week culminates about the eleventh day in a **second fastigium,** which in favourable cases does not attain the height of the initial fastigium of the first week. This second increase of fever may be absent, the temperature gradually falling during the second week, until about the fourteenth day, when it rapidly sinks to or below normal.

4. **Defervescence** is **sudden** in a majority of cases. Preceded very often by a final evening exacerbation on the 12th or 13th day—which Wunderlich calls a "critical perturbation"—defervescence is often "precipitous," the temperature falling 3° to 5° F. or more in a single night, then rising some 2° in the evening, and finally reaching the normal point for the first time next morning. Such is the **crisis** of typhus. It is, in fact, very like the critical fall of temperature in both measles and acute pneumonia (pneumonic fever), except that it occurs at the end of the second week of the fever instead of at the end of the first week, as in the two diseases named.

In severe cases, with cerebral symptoms (**ataxic typhus**), temperature ranges high all through the first week, there is no remission about the seventh day, and, worst of all, the thermometer continues to rise in the second week. This last occurrence often points to the advent of some complication which may postpone defervescence indefinitely, or it may usher in excessive fever or hyperpyrexia as already mentioned.

On the other hand, in **adynamic cases,** with heart failure and pulmonary obstruction, the range of temperature may be moderate—not exceeding 103°, or irregular, with spiking, or the fever may be continuous with absence of morning remissions, or a fall of temperature may be observed with a rise of pulse-rate, without any improvement in the general symptoms. In his "Lectures on Fever," [a] Dr. William Stokes quotes an old and well-founded opinion that all anomalous circumstances in fever are to be feared. The **aphorism** runs: "*Pulsus, vultus, et urina bona : æger moritur.*" And it is

[a] London : Longmans, Green, & Co. 1874. Page 361.

true. **Fatal cases** of typhus are generally preceded by **high temperature from the very beginning**, yet even in such cases it is not so much the intensity as the **continuance** of the fever which determines the mortal result. Just before death and in the death agony, both Wunderlich and Murchison describe a rise of temperature as a very constant phenomenon. According to the former authority, the temperature is seldom as low as 104° during the agony, more usually it ranges between 105·8° and 107·6° F., and once it reached 109·4° F.

In the *Dublin Journal of Medical Science* for February, 1878, I published a case of typhus with hyperpyrexia, in which the exceptionally high reading of 109·1° was recorded during the death-agony of the patient, a woman, aged thirty-eight, who succumbed on the 19th day of a typhus fever of the nervous or ataxic variety.

The following is a brief history of the case:—

Margaret T., aged thirty-eight years, married to a shoemaker, and residing in Cuffe-street, Dublin, was admitted to the Meath Hospital on the afternoon of Wednesday, September 12th, 1877. It is worth noting that a young man from the same house was under treatment for severe typhus in Cork-street Fever Hospital from July 18th to September 8th. There was no special medical history previously to her present illness, which commenced on Tuesday, September 4th, 1877, with a shivering fit, followed by chilliness so great as to oblige her to hang over the fire the greater part of the day. She also suffered from pains in the limbs. She fought against her illness until the following Friday (4th day), when she took to bed, as she was getting much worse. On Saturday, severe pain in the small of the back, and, next day, vomiting of "watery stuff" were superadded to obstinate constipation, which was an early symptom. From the outset also she was very sleepless. She did not come into hospital until the afternoon of the ninth day. In the evening her pulse was 120, her respirations were 28 per minute, and the axillary temperature was 104·9°.

Next morning her face presented a dusky, congested appearance; the eyes were heavy, and the conjunctivæ deeply suffused; her tongue was heavily furred, but moist; both it and the lips were

exceedingly tremulous. There was a marked "nervousness" of manner. A profuse mulberry eruption covered nearly all parts of the body—the original maculæ had become true petechiæ, which were unusually distinct and dark in colour. Her pulse was 124, not strong; respirations, 24; temperature, 104·3° (at 10 a.m.). The heart's action was rapid and weak, but both sounds were audible, and the impulse could be felt in the normal situation.

Treatment.—It was decided to free the bowels by a simple enema of olive oil and warm water, to support the strength by strong beef-tea and abundance of milk, four ounces of port wine and moderate doses of quinine.

September 14th (11th day).—The enema acted twice, bringing away dark, solid fæces in considerable quantity. There was a slight remission of fever, the morning temperature being 103°.

September 15th (12th day).—She spent a sleepless night, being very restless and moaning constantly. The heart was becoming rapidly weaker—the impulse was feeble, the first sound indistinct, the second relatively strong—being thrown into relief by the subsidence of the systolic sound. Strychnin and digitalis were added to the quinine mixture; and the port wine, which had been increased to 8 ounces the previous day, was further increased to 12 ounces, with 4 ounces of whisky. The heart's action was impeded by tympanites, to combat which a turpentine enema was administered. It acted once, with some relief to the patient. The persistent sleeplessness and increasing head symptoms led me to try, cautiously, tartar emetic and opium, as recommended by Dr. Graves. Only two doses, however, were taken—too small a quantity to induce sleep on the one hand, or to be accused of injuring the patient on the other. One-sixth of a grain of tartarated antimony and 10 minims of tincture of opium alone were given.

September 16th (13th day).—She passed a sleepless night, with much muttering delirium. The tongue was moist; but there was progressive failure of the heart. Its action was very weak—quite fœtal in character. Port wine, ℥xii.; whisky, ℥vi. Towards evening the temperature began to fall with an attempt at crisis. At 7.30 p.m. there were marked cerebral symptoms; but in the

night the bowels moved, and both diaphoresis and diuresis occurred, so that an evident effort at crisis was made.

September 17th (14th day).—The morning temperature was moderate—102°; but this one favourable symptom was accompanied by a rise in the rate of both pulse and respirations—a circumstance of evil omen. The rash was on this day evidently fading from the anterior aspect of the body, although it remained very petechial even in this situation.

September 18th (15th day).—The temporary remission of pyrexia was now succeeded by an exacerbation in the symptoms—a morning temperature of 103·7°, constipation, commencing retention of urine, and persistence of the rash. The increased meteorism obliged us to order another turpentine enema, which operated once.

September 19th (16th day).—Another pseudo-crisis occurred on this day, with *profuse diaphoresis*, and a fall of temperature to 101·7°. Again there was a complete want of accordance in the symptoms—the respirations remained as fast as ever (48 per minute), the pulse fell only four beats per minute, there was complete retention of urine. The pulse could scarcely be counted, owing, first, to weakness; secondly, to the intensity of the subsultus which was present. The urine was now slightly albuminous, and the urea was partially decomposed—the fresh urine, drawn off by the catheter, effervescing briskly on the addition of dilute nitric acid.

September 20th (17th day).—In no way better, in some respects worse; temperature rising; bowels costive, with tympanites.

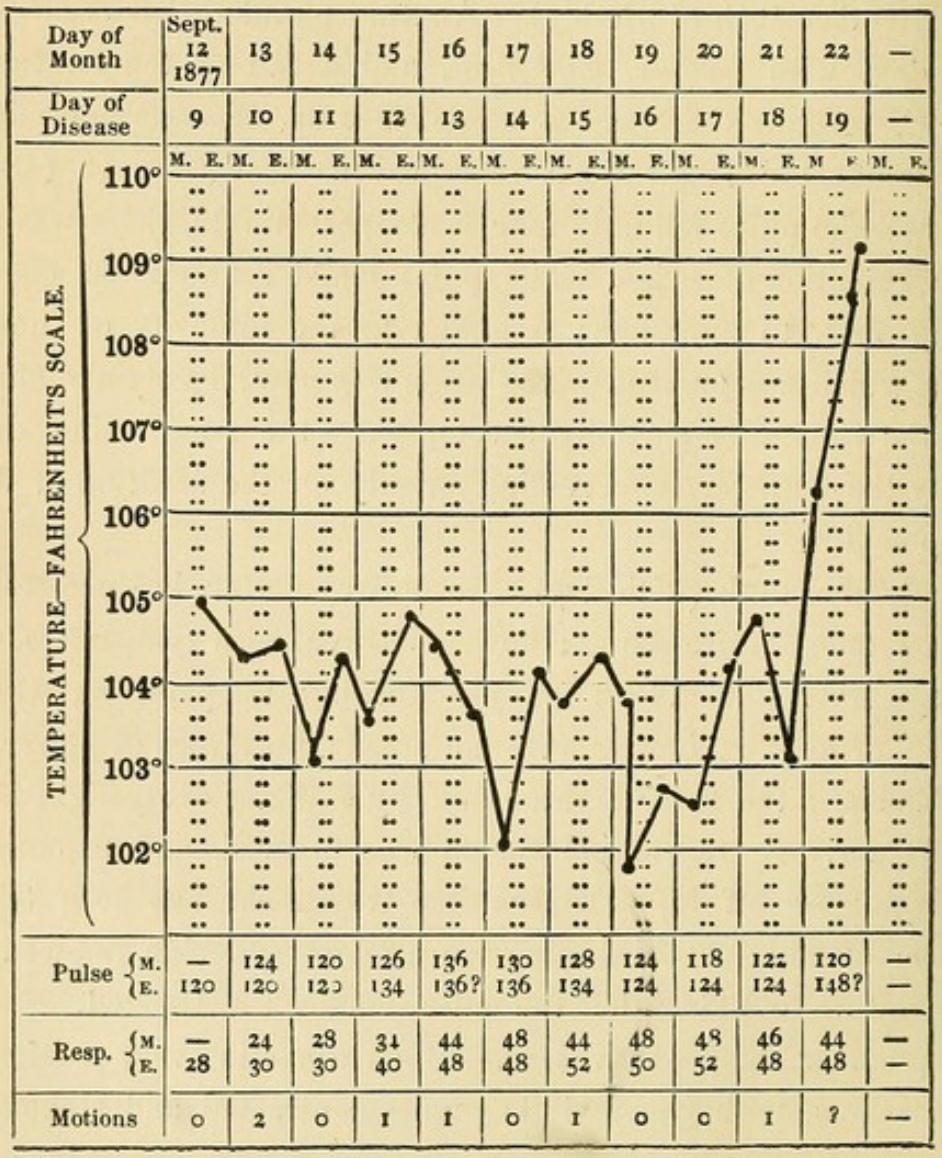
September 21st (18th day).—Morning temperature, 104·8°; pulse, 122 to 126; bowels have not acted; no return of power over the bladder; the urine is drawn off by catheter every eight hours, or oftener. A turpentine enema was ordered, and to omit the 3-minim doses of liquor strychninæ which she has been taking. In the evening a third abortive attempt at crisis reduced the temperature to 103·2°, but the pulse remained unaffected.

September 22nd (19th day).—Much worse in every particular; unconscious, with coma vigil; pupils contracted and sluggish to stimulus of light; considerable albuminuria; morning temperature,

106·4°. Ordered: one grain doses of camphor and musk every second hour. At 6·45 p.m. I visited her again. She was bathed in a cold perspiration; the extremities were cold, her eyes fixed, with coma vigil; bronchial râles heard universally over the chest; axillary temperature (taken twice with two reliable thermometers) was 108·6°. She was manifestly dying. At 7 30 p.m. the last observation on the temperature was made; it was found to be 109·1°. Three hours later she was dead. Unfortunately no observation was made after death. This was partly owing to the late hour at which she died.

Clinical Chart of Temperature, &c.

M. T.; Age, 38; Disease, *Typhus Fever;* Result, *Death on* 19*th day.*



Day of Month	Sept. 12 1877	13	14	15	16	17	18	19	20	21	22	—
Day of Disease	9	10	11	12	13	14	15	16	17	18	19	—
Pulse M.	—	124	120	126	136	130	128	124	118	122	120	—
Pulse E.	120	120	120	134	136?	136	134	124	124	124	148?	—
Resp. M.	—	24	28	34	44	48	44	48	48	46	44	—
Resp. E.	28	30	30	40	48	48	52	50	52	48	48	—
Motions	0	2	0	1	1	0	1	0	0	1	?	—

Authenticated temperatures as high as those noted in this instance—namely, 108·6° and 109·1°—are so rarely recorded that I was induced to bring forward the foregoing "Clinical Record." So far as I am aware, no such temperatures had been observed in the Meath Hospital since the modern introduction of Medical Thermometry. In a valuable appendix to Dr. Stokes' work on Fever, my colleague, Dr. Arthur Wynne Foot, gives a *résumé* of thermometrical observations in the medical wards of that hospital during the three years, 1871, 1872, and 1873. Among 9,248 observations, the highest reading noted was 107·2°. He says:—"On 27 occasions temperatures of 105° Fahr. or upwards were registered in typhoid fever in 15 patients, and of the 15 patients in whom the temperature on one or more occasions reached 105° Fahr. or upwards, five died." The highest temperature recorded was in "a girl aged 16; temperature on 30th morning 107·2° Fahr.; died on the 31st evening. Her mean temperature (51 observations) during the twenty-six days she was in hospital was 103·1° Fahr. The morning temperature, 107·2°, was coincident with severe rigors, preceded by violent pain in the abdomen, ushering in peritonitis, not due to perforation, but to propagation outwards of the irritation arising from numerous and extensive ulcerations of the intestinal glands."

Dr. Murchison,[a] speaking of the temperature in typhus, observes that "a severe case is often characterised, not merely by a high temperature in the first week, but by an anomalous or irregular range in the second; for example, *by an absence of the morning fall, or by a sudden fall with rise of pulse, or with no improvement in the general symptoms. In fatal cases there is usually a rise of two or more degrees just before death or in the death-agony.*"

I have italicised the latter part of this passage, as it bears so directly on the present case, and is so fully illustrated by it.

Wunderlich[b] remarks that "*Fatal cases* of exanthematic typhus generally announce themselves even from the very beginning by

[a] *The Continued Fevers of Great Britain.* Third Edition. 1884.

[b] *Medical Thermometry.* Translated by W. Bathurst Woodman, M.D. The New Sydenham Society. 1871. Page 331.

the enormous height of the temperature (41·2° C.=106·16° Fahr., and even more). The transient remission at the end of the first week is wanting in these cases. Death may occur in the second week with continual high temperatures. If the case enters the third week, *some remission may show itself on the fourteenth day, but this must not be regarded as a favourable symptom, and is very soon compensated.*

"Yet even in fatal cases *the temperatures in the third week are not so high as at the earlier periods, at least till near the death-agony.* The daily maxima do not exceed 40·8° C. (105·44° F.), but are, for the most part, moderate. The danger to life during this third week is indicated not by the height of the fever, but by its continuance.

"*Just before death, and in the death-agony, the temperature constantly rises in exanthematic typhus.* In all my cases in which it was possible to make observations, there was a rise of temperature during the agony of at least 1·25° C.=2·2° F., in one case of even 3·6° (6·48°); and on an average about 1·8° C. (3·24 F.). During the agony the temperature was seldom so low as 40° (104° F.); more usually it was about 41°—42° C. (105·8°—107·6° F.), and once 43° C. (109·4° F.)."

The curves of temperature in this last case are given in Table IV. at the end of Wunderlich's book, and are also to be found in Diagram VII. in Murchison's work, where the case is described as one of "*Typhus fatal on 17th day, with very high temperature* (109·4° *F.*) *before death.*" The chart is strikingly like that which is the subject of the preceding "Clinical Record."

The dread significance of such temperatures is aptly expressed by Wunderlich in his definition of "Hyperpyretic Temperatures"[a]—those "which in every known disease, except relapsing fever, in all probability indicate a fatal termination—107·6° F. (42° C.) or more."

[a] *Loc. cit.* Page 7.

CHAPTER XXVIII.

Analysis of the Chief Symptoms of Typhus.

The Surface: *Facies typhosa.*—Skin: maculæ or petechiæ, sudamina, herpes, poisonous odour, which is most infectious; branny desquamation, atrophy of nails, *taches bleuâtres*, purpura spots, vibices, profuse sweating—an ominous form of crisis.—Circulatory System: Pulse, vital condition of the Heart—Dr. Stokes's views—Question of stimulants.—Respiratory System: Rate of breathing—varieties of respiration—Hypostatic congestion—Breathing offensive.—Digestive System: Anorexia, boulimia in convalescence—Condition of the tongue—"Parrot Tongue"—Sordes—Diarrhœa.—Urinary System: Characters of the urine.—Nervous System: Headache, delirium, mental state, wakefulness, "coma-vigil" (Chomel), "coma-vigil" (Jenner), loss of muscular strength, decubitus, muscular paralysis, agitation, rigidity, general convulsions.—Lesions of Organs of Special Sense: The eye, ear, nose, taste, sensibility of the skin.

We will now briefly consider the different parts or systems of the body as affected by typhus.

I. **The Physiognomy.**—The expression of typhus—the **facies typhosa**—is characteristic. From the outset the patient looks dull and heavy. Afterwards the aspect is vacant and bewildered, sometimes wild and defiant. The face is flushed, with a dusky, earthy, or leaden hue. The conjunctivæ are suffused, the tongne is dry and brown, sordes coat the lips, gums, and teeth. Such is the **physiognomy** of bad typhus.

II. **The Surface.**—The **skin** is covered with maculæ or petechiæ in proportion to the severity of the attack. "The quantity of the eruption," says Murchison, "its depth of colour, and the earliness with which it becomes livid or petechial, are in a direct ratio to the severity of the case." All writers on typhus are agreed as to the ominous significance of a copious, livid, and petechial rash, and as to the mild nature of non-maculated cases. The skin is of a dirty congested hue, deepening into a leaden or livid tint along the sides of the body and over the back. **Sudamina,**[a] or sweat vesicles, and local eruptions of **herpes** are not uncommon.

[a] Lat. *Sudamen, sweat.*

A poisonous odour hangs about the skin of the typhus patient after the first week. It is **most infectious.** Lind compared it to the smell of rotten straw. Gerhard well described it as "pungent, ammoniacal, and offensive." Barrallier likened it to the odour developed by rubbing the leaves of rue between the fingers. Murchison thinks it more correct to speak of it as a smell *sui generis.* This typhus odour is strongest in damp weather and when the ventilation is deficient.

The forms which the true typhus eruption assumes have been already described, as well as the date on which it appears, and the percentage frequency of its presence. It only remains to mention that the rash comes out once and for all, **not in successive crops,** like the rose-spots of typhoid, and that the spots remain visible for a week or ten days. **Desquamation** takes place in fine branny scales, especially when the skin has presented a general erythematous blush. A. Vogel[a] has described and figured a white band and a furrow which often appear at the lunula of the **nails** four to six weeks after the commencement of the fever and gradually advance to the tip. The **hair** usually falls off in convalescence. The occasional appearance of the so-called *taches bleuâtres* is of no clinical importance.

Purpura spots and **vibices** are also seen in certain bad forms of typhus, especially when complicated with scurvy. This often happened in the Crimea, according to Jacquot. The purpura spots are independent of the characteristic petechiæ of typhus, although both are subcutaneous ecchymoses.

The skin is generally dry and hot from an early stage until crisis, which is often accompanied by moderate perspiration. A **profuse sweat** is an **ominous** form of crisis. In several cases, for the most part fatal, Murchison found that, on evaporation, the sweat left a white efflorescence upon the eyelids and face. This consists of rod-shaped and stellate crystals, composed of a free acid, fatty matter, and a large proportion of chlorides. Barrallier made a similar observation in the Toulon epidemic of 1861.

[a] *Die Nägel nach fieberhaften Krankheiten.* Deutsch. Arch. für klin. Med. 1870. Page 333.

III. **Circulatory System.**—The circulation is much disturbed. The pulse is at first full, but soft and compressible, and moderately quickened, to 108, 112, or 120 beats per minute. Occasionally it is abnormally slow—down to 48, 40, and even 30 beats. This is often a sign of debility. The impulse of the heart and the pulse beat do not always correspond in rate, some of the systolic waves failing to reach the radial artery, so that the heart may beat twice for every stroke of the radial pulse. The pulse always becomes more rapid and less full when the patient sits up or attempts to stand, positions which should never be assumed by the typhus patient. In severe cases the pulse becomes undulatory or dicrotous in the second week. This is an indication of very low arterial tension.

The **vital condition** of the **heart** is **profoundly altered** in this fever. It is to Dr. Stokes that we are indebted for a full clinical account of febrile weakening of the heart.[a] According to Louis, the typhous softening of the heart preponderated in the left ventricle. Hence, the first sound was weakened. Sometimes both ventricles were engaged, and then the first sound would disappear. In a case of "malignant fever," observed by Dr. Stokes in the Meath Hospital many years ago, the patient (a woman) became pulseless at the wrist, while the heart beat rapidly and forcibly. Transfusion of human blood was tried, and the patient rallied. The experiment not being repeated, death took place. Dissection showed that the body was absolutely bloodless. From this observation Stokes concluded that the blood passing through the ventricle had very little to say to the making of the first sound.

Weakening of the heart generally commences about the fourth or fifth day of typhus, and begins to subside about the tenth day. The first symptom is a **diminished cardiac impulse**, even when the patient lies partly on the left side, so as to bring the apex of the heart in contact with the chest wall. The **impulse**, in the next place, **fails** altogether, while the **first sound diminishes** in loudness.

[a] Cf. *Researches on the State of the Heart and on the use of Wine in Fever.* Dubl. Jour. of Med. Science. Vol. XII. 1839. Also *Diseases of the Heart and Aorta.* 1854. Page 366. And *Lectures on Fever.* London: Longmans Green & Co. 1874. Pages 185, *et seq.*

These signs are most apparent towards the left side, because the left ventricle is most affected. Occasionally the first sound is accompanied by a temporary bellows-murmur. The third stage is **disappearance of the first sound**, the heart beating with only one, and that **the second sound**, which by contrast may appear to be even **accentuated.** It may, however, be positively increased owing to tension in the pulmonary artery. In a yet more advanced stage of cardiac weakness **both sounds** are **equally diminished** in loudness, and become equi-distant, while the heart beats with great rapidity (140–160 per minute). To this state the term **fœtal heart** is applied, because in its weakness and quickness it resembles the heart-beat of the *fœtus in utero.* This condition indicates extreme debility. The last stage of all is **silence of the heart,** a sign of impending dissolution—as Stokes observes, a condition which is almost invariably fatal. It should be remarked that the influence of a very muscular or a very fat chest-wall must be allowed for, if present, when a physical examination of the heart is made. When loud bronchial râles and rhonchi are audible the heart sounds may also be concealed.

Should the patient recover, the physical signs alter in the inverse order. Coincidently with the return of the first sound, the pulse should fall in rate, otherwise the prognosis is bad. After the fever symptoms have vanished the pulse often falls much below the normal rate—even as low as 30 beats in the minute, and then slowly recovers itself.

Dr. Stokes points out that in fatty degeneration of the heart, or after a bite from the rattlesnake, wine may be taken in almost any quantity without producing head symptoms. From this he adduces an argument for the free use of wine in fever. At the same time he shows that when recovery of the heart coincides with an increasing rate of the pulse, stimulants are almost certainly doing harm—they must be omitted and at once.

IV. **Respiratory System.**—(1). As is well known, the ratio which the respirations bear to the pulse-rate is in health 1 to 4—that is, 18 respirations per minute correspond to a pulse-rate of 72 per minute. In typhus, where there is no pulmonary complication

of account and where the nervous symptoms are not pronounced, this ratio is commonly maintained. Thus a pulse of 120 would mean 30 respirations a minute. In grave cases, however, certain abnormal characters of respiration may arise from **cerebral disturbance** independently of any pulmonary complications. Thus, the breathing in such cases is hurried, **sighing**, irregular, spasmodic, or jerking (Murchison). There is, as Hudson[a] expresses it, a *besoin à respirer*, what the Germans call *Lufthunger*. This irregularity of breathing, independent of any pectoral affection, Graves[b] was in the habit of calling "**cerebral respiration.**" In other cases the breathing is irregular, blowing, or hissing, while the mouth is kept closed, the cheeks puff out, and the nostrils dilate with each expiration—this is the "**nervous** or **cerebral respiration**" of Sir Dominic Corrigan. A third variety is the rising and falling breathing, to which the name of "**Cheyne-Stokes respiration**" has been given.

(2). **Hypostatic Congestion** takes place in the most dependent parts of the lungs in consequence of impaired innervation (paralysis of the pneumogastric nerves), impaired nutrition of the blood vessels, and lessened heart power. Its occurrence is largely determined by the way in which the patient lies—as, for example, on the back (*dorsal decubitus*). At the same time serum is effused into the pulmonary tissue (serous œdema of the lungs), and effusion also takes place into the bronchi and bronchioles. This dangerous state of things, as a rule, betrays its development by neither cough nor expectoration, the chief symptoms being rapid, laboured breathing (30, 40, 50, or even 60 respirations each minute) and cyanosis due to defective aëration of the blood. The surface is livid, cold, and clammy, and the patient passes from stupor into coma.

Physical examination of the chest day by day affords timeliest warning of the stealthy approach of this perilous condition, and should never be neglected in typhus. The patient should not be raised in bed for this examination, but gently and slowly turned

[a] *Lectures on the Study of Fever.* Second Edition. 1868. P. 100.

[b] *Clinical Lectures on the Practice of Medicine.* New Sydenham Society Edition. Vol. I., page 177. 1884.

from side to side, while still in the recumbent position. Further, in order to protect himself from infection, the physician should allow fresh air to play for a moment about the patient's person before applying his ear to the back of the chest. A stethoscope with a long double stem should also be employed for auscultation. The **physical signs** are—First, a few coarse crepitating râles with, it may be, abnormal clearness on percussion probably due to relaxation of lung tissue. The râles are then heard all over the back and front of the chest, and increasing dulness on percussion may be detected with feeble, but not tubular breathing.

The **breath** of a typhus patient is **offensive**, heavy, and pungent, with a smell which has been likened to that of yeast. The proportion of carbon dioxide (CO_2) in the expired air of typhus was shown by Dr. A. Malcolm,[a] of Belfast, in 1843, to be considerably below the normal amount. This anomalous result is explained by Vierordt's observation that, even in health, the proportion of carbonic acid in the expired air diminishes as the respirations increase in frequency. Leyden of Königsberg[b] also, while confirming Malcolm's statement that the percentage of carbon dioxide in the expired air of typhus is diminished, found the absolute quantity increased by one half. In severe cases the breath has often an **ammoniacal** odour, and Murchison's observations led him to think that both in uræmia and in typhus the expired air may contain ammonia independently of the condition of the mouth and pharynx. He figures some very beautiful dendritic (tree-like) crystals of chloride of ammonium obtained by holding a glass rod moistened with hydrochloric acid before the mouth and nostrils of typhus patients.

V. **Digestive System.**—The appetite fails completely from the outset, and this **anorexia**[c] persists until crisis, when a return of appetite is one of the first signs of approaching convalescence. In fact, there is an intense craving for food and an enormous appetite

[a] *Edinb. Monthly Journ. of Med. Sc.* Vol. III. 1843.

[b] E. Leyden. "Ueber die Respiration im Fieber." *Deutsch. Arch. für Klin. Med.* 1870. Page 536.

[c] Gk. ἀνορεξία, *want of appetite.* From ἀν-, *priv.*, and ὄρεξις, *a longing* or *yearning for anything.*

(boulimia)[a] during recovery. The tongue is at first large, pale, and coated with a white fur, which soon becomes yellowish brown; it afterwards shows a dry, brown streak in the centre. At a still later stage of the bad cases it is thickly studded with sordes, becomes black and shrunken, and cannot be protruded, but catches behind the lower teeth. In this condition it is described as the "**parrot tongue**" of typhus. Occasionally it is red, with enlarged papillæ, but scarcely ever is it glazed and fissured like the tongue of typhoid or enteric fever.

The amount of dryness and darkness of the tongue is, according to Murchison, a fair criterion of the severity of the case. When stimulants, however, are given in excess the tongue is apt to become dry and brown. The **sordes** which gather on the lips, gums, and teeth, as well as on the tongue in the second week, consist of proliferated epithelium, which has desquamated quickly, fragments of food, various micro-organisms, dried mucus, saliva, and blood. This mixture becomes black from desiccation.

Nausea and vomiting are rare in typhus, so also is meteorism or tympanites. Gurgling (gargouillement) in the ileo-cæcal region, tenderness on pressure in this situation, intestinal hæmorrhage, and diarrhœa, are all exceptional in typhus. Sometimes, however, the bowels are loose, especially about the time of crisis. Constipation, however, is the rule in typhus. Both liver and spleen may be enlarged, as in other acute infective, or specific, diseases. When **diarrhœa** prevails, the motions become **strongly alkaline** in reaction, and contain many prismatic crystals of **triple** (ammonio-magnesian) **phosphate.**

VI. **Urinary System.**—The renal secretion is much diminished at first. It is acid, dark-coloured, and of high density (1024–1030). The coloration is due partly to concentration but still more to escape of blood-pigment, the result of *typhous dissolution of the blood.* Towards the close of the fever there may be diuresis, with a copious precipitation of urates or lithates. **Urea** is increased in quantity at first, but afterwards falls below normal. This is due, notwithstanding the disintegration of the nitrogenous tissues, to **low diet**

[a] Gk. *Βουλιμία, ravenous hunger.*

and **imperfect elimination.** In a "Note on the Relation between Urea and Fever," communicated to the Tenth International Congress at Berlin, Professor Horatio C. Wood and Professor John Marshall, of the University of Pennsylvania, stated that it seemed to them "not yet definitely decided whether increase of urea elimination is an integrant part of the fever process, or whether it is an indirect outcome, caused by the action of the excessive heat upon certain organs, or other disarrangements of the bodily functions." In their paper, the authors point out one important fallacy which must be guarded against—namely, the tendency to consider temperature as a measure of fever, using the term "Fever" in its proper sense to express "the abnormal nutritive process, the outcome of which is *often* elevated temperature."[a] There is reason to believe that the presence of urea, or some other derivative of albumen, in the blood will give rise to delirium, stupor, and coma, as well as to the ammoniacal odour given off by the skin.

Albuminuria is often present, but this does not necessarily imply renal disease, for in excessive blood-changes such as occur in bad typhus, the blood-serum may find its way into the urine. **Tube-casts,** however, are not uncommonly found on microscopical examination. Murchison suggests that possibly, in some cases, the albuminuria may be due to the presence of albumen in excess in the blood from disintegration of the tissues, which the glandular structures have been unable to convert into urea.

Chlorides gradually diminish from the outset, so that absence of chlorides from the urine cannot be regarded as pathognomonic of acute pneumonia, as has been supposed.

Uric acid is usually increased, and both Frerichs and Murchison have detected **leucin** ($C_6H_{13}NO_2$) and **tyrosin** ($C_9H_{11}NO_3$), two products of the disintegration of albumen or fibrin in the urine of typhus.

VII. **Nervous System.—Frontal headache** is an early and constant symptom. It is a dull, aching, heavy pain—rarely acute, darting, stabbing, throbbing, or bursting. It is the most charac-

[a] Verhandlungen des X. *Internationalen medicinischen Congresses.* Band II. Abtheilung II.: "Physiologie und physiologische Chemie." 40.

teristic nervous symptom during the first week of typhus. **Vertigo** and **rheumatoid pains** in the back and limbs, are also present during the earlier periods of the fever.

Mental confusion or actual **delirium** takes the place of headache towards the close of the first week. Hence typhus has been often called "**brain fever.**" The mind becomes blunted and dull, memory lapses, and cerebration is slow and defective. Hence the term "**typhus.**"[a]

The delirium varies in character. Three types, in particular, are described—(1.) A quiet, listless, low muttering delirium—the **typhomania** of Galen and early writers—well defined as "*affectus ex phrenitide et lethargo mixtus.*" The patient is easily roused from it so as to give either coherent or rambling answers, but soon relapses into a torpid or semi-unconscious state. (2.) A more busy form of raving, with prostration, restlessness, and tremors of the limbs, muscles of the face, and tongue—like the **delirium tremens** of the drunkard. (3.) An acute, noisy and violent or maniacal frenzy, in which the patient's strength appears supernatural for a time, but the outburst is succeeded by profound prostration or fatal collapse. In this form—called **delirium ferox**—the patient may exhibit a homicidal or suicidal tendency.

Of the three foregoing types the first is the most common, the last is the least common in typhus. One form may pass into another, or one form may merge in another.

"The **mental state** of the delirious typhus patient," writes Murchison, "is peculiar, and well worthy the study of the metaphysician. As a rule, the memory is first and most affected; judgment and power of connected reasoning often remain after the memory has entirely gone. The mind may labour under the strangest delusions, and often it appears to revolve obstinately around some fixed idea. The patients rave about objects which have greatly engrossed their attention, either immediately preceding the attack, or years before, and which are now jumbled with persons, scenes, and events, with which they have had no connection." He gives some very graphic illustrations of these mental phenomena from his own experience and that of other authors.

[a] Gk. τῦφος, *smoke*, or *vapour*.

Wakefulness is a common symptom in early typhus. It is apt to be followed by extreme nervous agitation and prostration, or by **somnolence**, deepening into complete **coma**, and terminating in death.

Sometimes a patient awakes from a prolonged sleep and insists that he has never closed his eyes. This condition is the "**coma-vigil**" **of Chomel**[a]—not of necessity a bad sign. But this term is, unfortunately, apt to mislead, for with more propriety it has been applied by Sir William Jenner to another condition of most ominous import. In the "**coma-vigil**" **of Jenner** the patient lies with his eyes wide open, with a vacant gaze, his lips parted, his face pallid and expressionless; the pulse rapid and feeble, or imperceptible; the breathing hardly to be detected; the skin cold and clammy or bathed in sweat. Although awake, the sufferer is insensible and surely dies.

Loss of muscular strength is an early and constant symptom. The patients are not only weak, but from the first complain of a *feeling* of feebleness and lassitude.

The **decubitus** is **dorsal**. In bad cases the patient sinks down in the bed, his head gliding from the pillow.

Various **muscular paralyses** are observed in typhus. Thus, paralysis of the neck of the bladder, coming on about the 10th or 11th day, will lead to involuntary dribbling of urine; paralysis of the *sphincter ani*, to incontinence of fæces. The coats of the bladder may lose their power from over-distension, causing retention of urine. Incontinence and retention may even co-exist. Meteorism is due to paresis—that is, partial paralysis—of the walls of the intestines. The orbiculares palpebrarum may be paralysed, so that the patient cannot close his eyelids—so causing keratitis and sloughing of the cornea from exposure. Aphonia, inability to protrude the tongue, and dysphagia, are other examples of typhous paralysis—the last-named being the worst of all, and usually the forerunner of death.

Muscular agitation indicates great prostration. It is observed in the old and infirm, in the intemperate, and in brain-workers.

[a] *Leçons de Clinique Médicale.* Paris. 1834. Tome I.

The forms it takes are—Tremulousness of the hands, tongue, or whole body; rapid oscillatory movements of the eyeballs (*nystagmus*[a]); choreiform movements of the extremities; twitchings of the tendons (*subsultus tendinum*) of the wrist and facial muscles; choreic convulsions (Murchison, one case; Barrallier, one case); picking at, or fumbling with, the bed-clothes (*floccitatio* or *carphology*), and obstinate hiccough (*singultus*). All these symptoms are of grave import, and are best marked in ataxic typhus.

Muscular rigidity is a much rarer phenomenon than muscular agitation, although equally unfavourable. The fingers may be clenched or the forearms flexed. Tonic spasms of certain muscles have been observed, or even trismus or strabismus. Well-marked opisthotonos, with the head bent back and the limbs rigid, was once observed by Murchison and once by Perry—both cases proving fatal.

General convulsions are most dangerous in typhus. They occur in about one per cent. of the cases, and with rare exceptions are of uræmic origin. In most cases there is albuminuria, with scanty or suppressed secretion of urine. Kidney disease is often present, or there may be an intemperate history. Occasionally, simple retention of urine determines an attack. Uræmic convulsions do not usually appear before the middle or end of the second week. The fit is commonly preceded by unwonted drowsiness or delirium, occasionally the patient may seem to be convalescing. Death may take place during the fit, and in 41 out of 51 cases observed by Murchison it happened in less than twenty-four hours. If life is prolonged, coma or a renewed attack may follow the first fit. Hippocrates[b] regarded convulsions as most dangerous in fever, and all writers agree with this opinion. Occasionally, however, recovery does take place. In Murchison's note-books there were records of 69 cases of typhus with convulsions. Of these 61 were fatal, and 8 recovered.

[a] Gk. *νυσταγμός, nodding*, or *drowsiness*; from *νυστάζω, to nod in sleep.*

[b] *Aphorisms.* Bk. IV. 66, 67. *Ἐν τοῖσι πυρετοῖσιν οἱ ἐκ τῶν ὕπνων φόβοι, ἢ σπασμοὶ, κακόν. Ἐν τυίσι πυρετοῖσι τὸ πνεῦμα προσκόπτον, κακὸν, σπασμὸν γὰρ σημαίνει.*

In Cork-street Fever Hospital during the year ending March 31, 1881, uræmic convulsions proved fatal in three instances, in two at least of which there was a distinct history of alcoholism. It is more than a coincidence that there was a marked tendency to cases of this kind in January, 1881—that is, after the Christmas holidays, and during a period of intense cold.

Retention of urine may give rise to **epileptoid convulsions**, in which case judicious treatment may afford immediate relief. Sir Dominic Corrigan, in his "Lectures on Fever,"[a] gives a striking instance of this; but the following quotation from Dr. Stokes's "Lectures on Fever"[b] will prove even more interesting. He says:—"The most long-continued attack of convulsions I ever witnessed was in the case of a student of this (the Meath) Hospital, who had gone on to the thirteenth day of fever. The distended bladder could be felt, but such was the violence of the convulsions, attended with extraordinary priapism, that all attempts at catheterism were futile. It was also impossible to get the patient to swallow anything, or to use an enema, and under these desperate circumstances we determined to employ chloroform inhalation. The greatest difficulties attended the administration; but, at last, the effect was produced. The convulsions ceased like magic, and suddenly a jet of urine sprang upwards to a great height from the still erect penis, the stream continuing to flow until the bladder was empty, when the priapism disappeared.

"We see, therefore," adds Stokes, "that where the uræmic condition and its accompanying convulsions depend on mere *retention* of urine, we have a ready and efficacious remedy in careful and judicious evacuation of the bladder by the catheter."

VIII. **Lesions of Organs of Special Sense.**—

1. **The Eye.**—The conjunctivæ are generally injected with dark blood, hence the expression, "**the ferret-eyes**" of typhus. The pupils are, as a rule, contracted, sometimes to a mere point—**the pin-hole pupil** of Graves. This is noticed in cases of acute delirium. When stupor is profound or has deepened into coma, the

[a] Dublin: Fannin & Co. 1853. Page 43.

[b] London: Longmans, Green & Co. 1874. Page 421.

pupil dilates, and is insensible to light, and slight strabismus occurs.

Dr. Cayley observes that inequality in the pupils is by no means an infrequent symptom in both typhus and enteric fever. It may occur at any time, and is apparently without prognostic significance. He offers no explanation of the phenomenon.

Photophobia also is not of uncommon occurrence.

2. **The Ear.**—During the first few days, and also in convalescence, tinnitus aurium and noises in the head are often present. After the fifth day, **deafness** of one or both ears may occur, either as part of the general anæsthesia of the fever, or as a result of typhous softening of the intrinsic muscles of the ear, according to Dr. Stokes. In some cases, otitis may be the cause of the deafness, and otorrhœa will solve the question. Fracastori (1546) regarded deafness as a favourable symptom—"*Surditas salutem portendit*," and Alison, of Edinburgh (1849), was of the same opinion. But it is only relatively true, morbid acuteness of hearing, or **intolerance of sound** being distinctly a **bad sign.**

3. **The Nose.**—The sense of smell is blunted, often from catarrh. **Epistaxis**, or nose-bleeding, is **very rare** in typhus. Murchison met with it about a dozen times in 7,000 cases, and even then it was generally scanty. This is a point of diagnostic importance between typhus and typhoid fever.

4. **Taste.**—This sense is perverted from the outset, and in advanced and severe cases is abolished. Acids are longest relished, but after a time cold water is preferred (Murchison).

5. **Sensibility of the Skin.**—This is usually lessened, or may be lost towards the end in grave cases (**anæsthesia**). On the other hand, the opposite condition, **hyperæsthesia**, is occasionally observed.

CHAPTER XXIX.

Complications and Sequelæ of Typhus.

Causes *of Complications* in Typhus. Complications affecting (1) the Respiratory Organs: Bronchitis, pneumonia, gangrene of the lung, pleurisy, tuberculosis, hæmoptysis, laryngitis. (2) The Blood and Circulation: Acute hæmophilia, pyæmia, venous thrombosis, phlegmasia dolens, arterial thrombosis and embolism, heart diseases. (3). The Nervous System: Meningitis, mental disease, paralysis, neuralgic and rheumatoid pains. (4). The Organs of Digestion: Erysipelas of the pharynx, hæmatemesis, diarrhœa, dysentery, intestinal hæmorrhage, jaundice, peritonitis. (5). The Urinary Organs: Nephritis, vesical catarrh, hæmaturia. (6). Diseases of the Integuments and Bones: Œdema, bed sores, gangrene, noma or cancrum oris, "hospital gangrene," buboes. (7). Other Specific Diseases: variola, scarlet fever, diphtheria, erysipelas, typhoid fever.

Complications and Sequelæ of Typhus.—

The chief determining causes of the complications which modify the course of typhus are:—(1). The weakened state of the heart; (2). The impure state of the blood; (3). Constitutional peculiarities and family idiosyncrasies; and (4). The "epidemic constitution" at a given time or place.

I. Diseases affecting the Respiratory Organs.—

"The advent of pulmonary complications in typhus is most insidious, for the ordinary symptoms, cough and expectoration, may be slight or absent, and the patient is unable to complain of pain" (Murchison). Hence the necessity of a daily careful investigation of the chest in this fever.

1. **Bronchitis**, or, as Stokes preferred to call it, the "Bronchial Affection" of typhus, is one of the commonest complications of this fever—hence the names "**Catarrhal Typhus**," given to the disease by Irish writers, and "**Broncho-Typhus**," or "**Pneumo-Typhus**," of Rokitansky.[a]

[a] *A Manual of Pathological Anatomy.* Vol. IV., page 23. Sydenham Society. London. 1852. Translated by Dr. Day.

Bronchial catarrh may usher in, accompany, or succeed an attack of typhus. It is a dangerous complication, particularly in winter—first, because it is almost certain to be associated with more or less hypostatic consolidation in the lungs; secondly, because the bronchial secretion is likely to accumulate in the tubes and asphyxiate the patient in consequence of his inability to cough, coupled with the impaired nutrition and paralysis of the muscular fibres of the bronchi.

Although Stokes[a] denied to the bronchial affection of typhus the name of "bronchitis," he practically admitted that it was bronchitis when he spoke of it as "a special condition of the air-passages, secondary to the fever, the result either of the typhous deposit or *of the vascularity with turgescence* to which I have already alluded." He adds: "I do not know any characteristic difference between the physical signs which may occur in ordinary idiopathic bronchitis and those which present themselves in typhus when the air-tubes are engaged." Surely this is conclusive.

2. **Pneumonia** is a rare complication or rather sequela of typhus. It may end in recovery, or in abscess, gangrene, tubercular phthisis, or fibroid condensation (*cirrhosis*). It may be **distinguished from hypostatic congestion** (with which, however, it is sometimes associated) by the fact that the dulness is unilateral and by the presence of tubular breathing and rusty sputa.

Dr. Stokes,[b] in his "Lectures on Fever," long ago drew attention to a peculiar form of pneumonia which occasionally seemed to replace—as it were—the symptoms of fever, and to which, accordingly, he gave the name of "**aborted**" or "**arrested typhus.**" But his very graphic and striking description exactly corresponds to acute pneumonia of the apex; and, in the light of modern investigations, his cases of supposed arrested or aborted typhus read much more like **pythogenic pneumonia** or **pneumonic fever** than typhus. It should also be borne in mind that this consolidation is described as taking place as early as the fourth or fifth day of the supposed typhus, when as yet there is no typhus rash, and at a

[a] *Lectures on Fever.* London: Longmans, Green, & Co. 1874. Page 131.
[b] *Loc. cit.* Page 157, *et seq.*

time when consolidation and even crisis are not uncommon in acute pneumonia.

3. **Gangrene of the Lung**, easily recognised by the abominable fœtor of the breath and expectoration, and by the ghastly and pinched look of the patient, is a rare and fatal complication. It is apt to occur in the most destitute patients. In one or two cases observed by Murchison it was secondary to extensive bedsores over the sacrum, being evidently of embolic origin.

4. **Pleurisy** is a rare and latent complication. The effusion is generally **purulent** from the beginning as in other specific fevers.

5. **Tuberculosis of the lung** is also a rare sequela of typhus, notwithstanding Stokes's opinion to the contrary, based upon his observations of "fever" cases which were probably typhoid, not typhus.

6. The occurrence of **hæmoptysis** must be considered as exceptional. It results either from tubercular deposits or from *acute hæmophilia* (Murchison).

7. **Laryngitis** is an infrequent but dangerous complication. In Germany, Rokitansky gave to the disease the name of **Laryngo-Typhus.** It may be croupal in character or show itself as acute œdema of the glottis. When the inflammation involves the cartilages, it is called "**perichondritis laryngea.**"

II. **Diseases of the Blood and Circulatory Organs**:—

1. **Acute Hæmophilia.**—Typhus patients, like smallpox patients, may from "dissolution of the blood" become "**bleeders**"—the defibrinated and devitalised blood escaping from the vessels with unwonted ease. Hence the formation of purpura spots and vibices, and the occurrence of epistaxis, hæmoptysis, hæmatemesis, melæna, menorrhagia, and hæmaturia, as well as other hæmorrhages. This tendency to bleed is most marked with coincident scurvy and typhus, as in the French Army during the Crimean War and in the Epidemic of 1847–1848, at Edinburgh and elsewhere.

2. **Pyæmia**, with purulent deposits in the joints, is a rare and very fatal complication about the period of the crisis, or it occurs as a dangerous sequela during convalescence. It is ushered in by

repeated rigors, and is accompanied by extreme prostration, heart-failure, jaundice, and profuse sweating.

3. **Venous Thrombosis** occurs as an occasional sequela, causing **Phlegmasia alba dolens**, or **White Leg.** Stokes held that this lesion was to be expected when the pulse continued quick in convalescence with no pulmonary or abdominal complications to account for its doing so. In ten years only one case per 800 was observed in the London Fever Hospital; but it used to occur much more frequently. It is not always a painful affection. The left leg is most usually affected, perhaps owing to compression of, and consequent slowing of the circulation in, the left iliac vein by the right iliac artery. The swelling is firm and brawny, and at times enormous. The skin is pallid, hence the term "alba."

Besides venous thrombosis, **obstruction of the lymph channels** (J. Warburton Begbie[a]), or **inflammation of the subcutaneous areolar tissue** (diffuse cellulitis) may also cause phlegmasia. The swelling is then rugose and painless, as well as firm and brawny.

4. **Arterial Thrombosis** and **Embolism** are occasional, but serious, complications or sequels of typhus. They cause local gangrene, cancrum oris, osseous necrosis, abscess or gangrene of the lungs, and splenic infarctions.

5. **Heart Diseases.**—Both **pericarditis** and **endocarditis** are **extremely rare** complications. Hence, Dr. Stokes could not believe in the inflammatory nature of the affection which led to weakening of the heart in typhus. Murchison met with only two examples of pericarditis, and one example of endocarditis, in this fever. Notwithstanding the views put forward by Stokes, no doubt is now entertained as to the organic nature of the changes which take place in the **myocardium** in typhus. It is **an acute granular disintegration** of the muscular tissue of the heart, such as has been described above in the account of the pathology of scarlet fever. (See Chapter XVIII., page 178.)

[a] *Edinb. Med. Journal.* September, 1872.

III. Diseases of the Nervous System.—

1. **Meningitis** is, undoubtedly, **very rare** in typhus; but cases have been recorded by Corrigan and Hudson[a] in Dublin, J. B. Russell[b] in Glasgow, Jacquot[c] in the Crimea, Roupell[d] and Murchison[e] in London. The cerebral symptoms of this fever are, however, almost invariably independent of inflammation.

2. **Mental Disease—Temporary Fatuity and Mania** are rare, and very sad, sequelæ of typhus. Both at the Meath Hospital and at Cork-street Fever Hospital I have known mania to occur after this fever, necessitating the removal of the patient to a Lunatic Asylum. Happily, recovery generally follows at last, usually in two or three months.

3. **Paralysis** also is a rare sequela. It is most commonly observed as **hemiplegia**, when it perhaps depends on "arterial thrombosis of the central organs of the nervous system" (Murchison). Hudson[f] mentions the occurrence of **paraplegia**, but gives no illustrative case. About the year 1880, two cases of this sequela occured in the practice of the Meath Hospital—one of dorsal, the other of cervical, paraplegia. Both patients were males, and recovery followed in each instance. Dr. Henry Kennedy[g] observed a case of **general paralysis** after petechial typhus, the patient being a woman aged forty-four.

4. **Muscular Pains** sometimes occasion much distress in convalescence, and protract recovery. They are sometimes of a neuralgic character, but often resemble muscular rheumatism. The former type may be the precursor of phlegmasia, gangrene of the feet, or paralysis (Murchison). In some cases, the pains are probably symptomatic of **peripheral neuritis**, which is now known to occur after specific fevers.

5. An interesting phenomenon sometimes observed in typhus or

[a] *Lectures on the Study of Fever.* By Alfred Hudson, M.D. Dublin. 1868. Second Edition. Pages 252 *et seq.*

[b] *Glasgow Med. Journal.* February, 1869.

[c] *Du Typhus de l'Armée de l'Orient.* Paris. 1858.

[d] *A Treatise on Typhus Fever.* London. 1839. Pp. 108, 217.

[e] *Lancet.* 1865. Vol. I. Pp. 417, 482.

[f] *Loc. cit.* Page 248. [g] *Ibidem.*

during convalescence from this fever is the development of the pathological deep reflex known as "**ankle-clonus**," and the exaggeration of the physiological deep reflex called the "**knee-jerk**" or "**patellar reflex.**"

IV. **Diseases of the Digestive Tract.**—

1. **Erysipelas of the Pharynx** is met with in some cases, causing dysphagia and—it may be—œdema of the glottis, and so endangering life.

2. **Hæmatemesis**, or **vomiting of blood**, is occasionally observed. It may be profuse and so prove fatal. In these cases the rash on the skin is unusually abundant and dark.

3. **Diarrhœa** is sometimes so severe as to constitute a complication. In some epidemics it is more common than in others.

4. **Dysentery** complicates typhus in camps and sieges. It prevailed also in some of the Irish epidemics of typhus. The poisons of the two diseases are, doubtless, developed under somewhat similar circumstances.

5. **Intestinal hæmorrhage** is **very rare** but **very fatal** in typhus. It is due to a **liquefied state of the blood**, and is often associated with hæmatemesis and other hæmorrhages. Its ætiology, therefore, is different from that of intestinal bleeding in typhoid.

6. **Jaundice** also is **rare** but **fatal** in typhus. Sir William Jenner never met with an instance, but 15 cases came under Murchison's notice. In 3 of these the jaundice was due to consecutive congestion of the liver; in a fourth case, it resulted from gastro-duodenal catarrh. In the remaining 11 cases, of which 9 proved fatal, the jaundice co-existed with the rash and was evidently **due to some abnormal state of the blood**, as in pyæmia, yellow fever, snake-bite, and other blood-poisonings. Bile pigment was present in the urine, so there was true jaundice.

7. **Peritonitis** is almost unknown as a complication of typhus. Sir William Jenner and Dr. Alexander Collie have each recorded a single instance of the occurrence of acute idiopathic peritonitis in convalescence. Murchison saw two cases—the causes being, respectively, the bursting of a softened embolic infarct in the spleen and tuberculosis of the peritoneum.

V. **Diseases of the Urinary Organs.**—

1. **Nephritis**—whether primary or secondary, acute or chronic, is a most serious complication of typhus—often inducing uræmic convulsions.

2. **Catarrh of the Bladder (Cystitis)** sometimes occurs in convalescence, especially after retention of urine and over-distension of the bladder.

3. **Hæmaturia** may result from:—(1.) acute hæmophilia, (2.) nephritis or congestion of the kidneys, (3.) cystitis.

VI. **Diseases of the Integuments and Bones.**—

1. **Œdema**, if slight, is generally the result of debility. General anasarca may occur from nephritis. Both appear in convalescence, when the patient begins to walk. In this stage, also, I have repeatedly witnessed extreme suffering from painful feet—the cause apparently being non-removal of the epithelium from the soles of the feet, and consequent obstruction of the sweat ducts.

2. **Bedsores** (gangrene of the integument) occur in protracted cases from :—(1) pressure, as over the sacrum, trochanters, heels, occiput, ears, elbows, and so on; (2) early neglect and want of skilled nursing; (3) impaired innervation.

3. **Spontaneous gangrene**, independently of pressure, is probably brought about by arterial thrombosis. It affects the toes and feet, the nose, penis, scrotum, and pudenda in the female. It occurs in badly-fed patients, and is ushered in by severe shooting pains, numbness, coldness, and lividity. In my "Medical Report" of Cork-street Fever Hospital, for the year ending March 31, 1879, I mentioned the case of a typhus patient who died on the *fourth day* of the fever from gangrene from the fingers.

4. **Noma**, or **Cancrum Oris (gangrenous stomatitis)**, is a very destructive and fatal variety of gangrene, which attacks the cheek, mouth, tongue, and face of delicate, badly-fed children, about the end of the second week of typhus. It is not peculiar to this disease, for it occurs also in smallpox, scarlatina, and measles. According to Rilliet and Barthez, not more than one in twenty cases of noma recover. It is regarded by Hutchinson as allied to, or identical with, "hospital gangrene." The first sign is usually

a brawny swelling in the substance of the cheek. This becomes purple, softens and breaks down, and then the gangrenous ulceration may spread in all directions, causing perforation of the cheek, and involving the lips, tongue, alæ nasi, and eyelids, and even causing exfoliation of the teeth and jaws. I have myself seen two cases of this terrible complication. In one, a large gaping cavity opened through the cheek into the mouth, and the smell of the breath was indescribably fœtid.

5. **Hospital Gangrene** attacks wounds and ulcerated surfaces in persons under the influence of typhus. It is a contagious form of gangrenous inflammation which attacks a raw surface, and is identical with, or closely allied to, acute sloughing phagedæna. The wound, on invasion, ceases to discharge and becomes coated with a gray tenacious slough extending from the centre towards the margin. The patient usually sinks exhausted. The disease is micro-parasitic, and its ultimate extinction may, therefore, be looked for in time.

6. **Inflammatory Swellings** or **Buboes** are not infrequent in typhus, especially in the parotid and submaxillary regions, about, or after the time of crisis. They form rapidly, and are very painful. According to Drs. Craigie[a] and Graves,[b] the inflammation has its seat mainly in the subcutaneous areolar tissue, and not in the substance of the glands. In some cases, they have seemed to Murchison to originate in extravasations of blood. The same author considers that these inflammatory swellings constitute a connecting link between typhus and Oriental plague, or bubonic fever. He goes so far as to say that "typhus is probably the plague of modern times."

These bubonic swellings may terminate in purulent infiltration and abscess, or may recede without suppurating. In the former case especially they are a formidable complication of typhus.

VII. **Other Specific Diseases.—**

1. **Variola**; 2. **Scarlet Fever**; 3. **Diphtheria**; 4. **Erysipelas**;

[a] *Fevers.* Edinburgh. 1837. P. 301.

[b] *Clinical Lectures on the Practice of Medicine.* New Syd. Soc. 1886. Vol. I., page 223.

5. **Typhoid** or **Enteric Fever**—are all known to have complicated or closely followed upon typhus.

Murchison reports the case of a girl aged fifteen, ill of varioloid, who was conveyed to the Smallpox Hospital in a carriage used for the removal of typhus patients. While still going through her illness a typhus rash appeared. In 1862, Dr. Peacock reported in the *Lancet* a case in which the rashes of typhus and scarlatina actually co-existed. Both Murchison and W. T. Gairdner, of Glasgow, have seen typhus complicated with, or followed by, diphtheria. Erysipelas of the face is, according to the former author, an occasional complication of typhus, appearing, perhaps, as early as the third day, but more commonly towards the close of the second week, or during convalescence. It was noticed by Murchison, in 92 out of 14,676 cases, or **once in 159** cases. Lastly, the co-existence of typhus and typhoid fevers admits of no question, and, in my opinion, the frequency of this accident led to the confounding of these two fevers and to the view that they were to be regarded as merely modified forms of the one essential disease.

CHAPTER XXX.

TYPHUS—(*continued*).

VARIETIES OF TYPHUS : Inflammatory, nervous or ataxic, adynamic, ataxo-adynamic or congestive, *Typhus siderans*, *Typhus levissimus*, abortive, catarrhal. DIAGNOSIS : from relapsing fever, enteric fever, "jungle fever," purpura, measles, meningitis, *delirium tremens*, asthenic or "typhoid" pneumonia, uræmia—PROGNOSIS and MORTALITY : Bad signs in typhus ; death-rate influenced by age, sex, condition of life and habits, season, pregnancy, fatigue, privation, late treatment—PATHOLOGICAL LESIONS : (1) General ; (2) Special, affecting the integumentary, respiratory, circulatory, nervous and digestive systems of the body—Pathology of the "*Typhoid State.*"

Varieties of Typhus.—The following, among many other varieties of this fever, have been described :—

1. **Inflammatory Typhus**, characterised by much febrile reaction in the young and robust, and in patients of the upper class—a rare form, having occurred in only 40 out of 1,302 cases observed by Barrallier.

2. **Nervous** or **Ataxic Typhus**, in which nervous symptoms predominate. The eruption is generally copious, dark, and petechial. This form is also described under the names, "Typhus Comatosus" and "**Brain Fever.**" It was observed by Barrallier in 109 out of 1,302 cases.

3. **Adynamic Typhus**, accompanied by great muscular and cardiac prostration, involuntary evacuations, and a tendency to collapse. The skin may be cool and the pulse slow. Barrallier noted this form in 92 out of his 1,302 cases.

4. **Ataxo-adynamic Typhus**, or the **Congestive Typhus** of Armstrong. This is a combination of the ataxic and adynamic forms, and is by far the most common variety, having occurred in 810 out of Barrallier's 1,302 cases.

Typhus Siderans, or "**Blasting Typhus**," very acute, and fatal within a few hours or days.

6. **Typhus Levissimus**, or "**Mild Typhus**," observed by Barrallier in 235 out of 1,302 cases. "Typhus levissimus" was described by von Hildenbrand in 1810 under this name.

7. **Abortive Typhus.** Jacquot gave the name "**Typhisation à petite dose**" to cases in which symptoms occurred in persons exposed to infection without developing into actual typhus. Of this incomplete form Niemeyer observed some cases in the Magdeburg Hospital, and a large number of observations made in the Prague epidemics of 1843 and 1848 exactly correspond with his. The patients—who, in his cases, had always been exposed to typhus poison—complained of rigors, great depression, lightness of the head, frontal headache, weakness of the limbs, loss of appetite, and other symptoms common to the incubation stage of typhus, renewed and stronger chills, slight delirium, and catarrhal symptoms. Towards the close of the first week, the constitutional disturbance, fever, and catarrhal symptoms disappeared, and the patients began to convalesce. They recovered very slowly, suffering much from prostration. Niemeyer called the affection "a rarely-mentioned **abortive form** of typhus."[a]

8. **Catarrhal Typhus**, an Irish appellation for the disease, because it is so often complicated with bronchial catarrh. In Germany, the names "**Broncho-typhus**" and "**Pneumo-typhus**" were introduced by Rokitansky, as already mentioned.

Diagnosis.—The rash is pathognomonic of typhus. Before it appears, we have grounds for a differential diagnosis in a history of exposure to the infection, of such symptoms after exposure as rheumatoid pains, headache, and early prostration.

It may be necessary to distinguish typhus from the following diseases, or diseased conditions, or *vice versâ*:—

1. Spirillum Fever (Relapsing Fever).
2. Typhoid or Enteric Fever.
3. Tropical Remittent Fever ("Jungle Fever").
4. Purpura.
5. Measles.

[a] *Text-Book of Clinical Medicine.* Revised Edition. 1880. Vol. II., page 622.

6. Meningitis: Encephalitis.
7. Delirium Tremens.
8. Asthenic, or Typhoid, Pneumonia.
9. Uræmia.

1 & 2. It will be convenient to postpone the diagnosis of Typhus from **Relapsing Fever** and **Typhoid** or **Enteric Fever** until those fevers have been described.

3. The "**Typhoid-Remittent**" Fever of the tropics—or "**Pernicious Malarial Fever**" (Hirsch)—commonly called "**Jungle-Fever**" in India—is a miasmatic disease, caused by *malaria*. It is non-contagious, independent of overcrowding, prevails in tropical climates, and in warm and rainy seasons. The spleen is much enlarged, and quinine often acts specifically. Petechiæ may be present, but the macular rash of typhus is wanting.

4. Riverius long ago (1648) distinguished the *petechiæ sine febre* (**purpura**) from typhus (**febris petechialis**).

Purpura is non-contagious; as a rule, apyrexial; is unaccompanied by cerebral symptoms, but is attended with hæmorrhages from the mucous membranes. The spots, too, are larger than the petechiæ of typhus.

5. **Measles** is distinguished from typhus by the history of exposure to the poison of measles, the prevalence of an epidemic, the prodromal catarrhal symptoms, the brighter tint and greater abundance of its rash, the presence often of diarrhœa, and the early defervescence. The bronchial catarrh of typhus appears late and in the lighter cases the rash is not abundant.

6. In **Inflammation of the Brain** (**Cerebritis**, or **Encephalitis**) or **its membranes** (**Meningitis**), delirium is present from the very first, and is accompanied by excruciating headache. The senses are morbidly acute, whereas they are dull and blunted in typhus. The pulse is bounding. The eyes have not the heavy, dull look of typhus, nor is the rash of that disease present. Loud cries and screams (*cri cérébral*), as well as strabismus, ptosis, opisthotonos, and partial palsy, characterise meningitis. There is extreme intolerance of light (photophobia) and of sound. Nausea and vomiting

are common. A red streak is left upon the skin after pressure by the finger (*tache cérébrale*, or cerebral stain).

7. The **Delirium Tremens** of the drunkard differs from that of typhus in setting in with sleeplessness and delirium, without shivering, headache, or pains in the limbs. The tongue is moist and coated with a creamy fur, the skin is moist and cool, there is no eruption, and the temperature is not high.

8. In **Asthenic** or **Typhoid Pneumonia** the apex of the lung may be chiefly affected, when the symptoms of the local disease may be masked by the constitutional state (latent pneumonia). The differential diagnosis will, under such circumstances, turn on the presence of physical signs in the lung and the absence of eruption.

9. In **Uræmia** from chronic renal disease, the "typhoid state" may be fully developed, leading to great difficulty in diagnosis. But it is in **chronic interstitial nephritis** (contracted granular kidney—renal cirrhosis) that uræmia chiefly occurs. This form of kidney disease usually occurs in advanced life, especially in gouty subjects or persons suffering under chronic lead-poisoning (plumbism), and the temperature is normal or subnormal. This last circumstance Murchison calls "the grand point of distinction." Finally, the typhus rash is, of course, wanting in uræmia.

Prognosis and Mortality.—

The unfavourable signs in a given case of typhus are:—(1) A presentiment of death, often entertained by physicians when ill of typhus; (2) a soft and compressible pulse in rate above 120 in an adult; (3) absence of cardiac impulse, and lessened or silent first sound of the heart; (4) hurried respirations, particularly if no pulmonary lesion exists to explain this symptom; (5) sleeplessness and delirium; (6) complete coma-vigil of Sir William Jenner; (7) the presence of the *pin-hole* pupil of Graves; (8.) great prostration; (9) convulsions; (10) muscular tremors and hiccough; (11) relaxation of the sphincters before the tenth day; (12) tympanites or meteorism; (13) lividity of the face and surface generally; (14) abundance and darkness of the rash; (15) persistent high temperature; (16) profuse sweating after the tenth or twelfth day; (17) the presence of any serious complication.

When death takes place, it occurs from **asthenia**, with heart-failure; or from **ataxia**, nervous symptoms deepening into coma; or from some **intercurrent complication** or **sequela**.

Mortality.—There is reason to believe that the death-rate from typhus among the community taken all round does not exceed **10 per cent.** of those attacked. Hospital statistics show a much higher rate. Thus, in 23 years ending with 1870, of 18,268 patients admitted in typhus to the London Fever Hospital, 3,457 died, giving a mortality of **18·92 per cent.** The mortality since 1862 has been less than it had been previously—18·22 per cent. compared with 20·89 per cent. As in the case of other epidemic diseases, the mortality is greater immediately after the outbreak than in the later stages.

The last serious epidemic in the Annals of Cork-street Fever Hospital occurred in the years 1880–82, immediately in the wake of the smallpox scourge of the years 1876–80. So far back as January, 1880, typhus showed an epidemic tendency in Dublin, the female department of the North Dublin Union Workhouse being the chief focus from which the disease spread in the first instance. Out of 57 cases of typhus admitted to Cork-street Fever Hospital in the three months ending March 31, 1880, not fewer than 40 came from the female division of the North Dublin Union. The outbreak attained its greatest violence in the last quarter of 1880, during which the admissions rose to 217. It finally died out in the early part of 1882, the admissions falling to 28 in the first three months of that year. In all, 675 patients were treated during the epidemic, and of these 71 died, the resulting death-rate being 10·5 per cent. As usual, the disease was much more malignant at the beginning of the epidemic than it was in its later stages. It is interesting to recall the fact that this—the last serious outbreak of typhus in Dublin—followed not only in the wake of a prolonged epidemic of smallpox, but also in that of the disastrous year 1879, the continuous cold and wet of which led to unparalleled and general distress throughout Ireland.

With the assistance of Dr. John Marshall Day, the Resident Medical Officer of Cork street Fever Hospital, I have tabulated the

number of cases of typhus admitted to the hospital annually in the twenty years ending March 31, 1891, as well as the deaths from this fever which occurred in each of the same twenty years. I find that the total number of cases admitted was 2,895, of which 363 proved fatal. These figures give an average mortality of 12·6 per cent., or *nearly one in eight.* It is worthy of note that the wards of Cork-street Hospital are splendidly ventilated—indeed almost to excess in winter. I have no doubt that this free ventilation contributes largely to so low a death-rate from typhus.

Age influences the mortality from typhus in a most remarkable way. This is shown in the accompanying Diagram (B.), copied from Murchison's "Treatise on the Continued Fevers of Great Britain," by permission of Messrs. Longmans, Green & Co. It shows the variations—according to age—of the percentage death-rate of 18,132 cases of typhus admitted to the London Fever Hospital. According to this diagram, the mortality during the first five years of life was 6·69 per cent.; in the second lustrum, it fell to 3·59; between 10 and 15 it was only 2·28 per cent., between 15 and 20 it rose to 4·46 per cent. After 20, it progressively increased until of those—

Above	30	years of age	35·39	per cent. died.
,,	40	,,	43·48	,,
,,	50	,,	53·87	,,
,,	60	,,	67·04	,,
,,	70	,,	79·00	,,
,,	80	,,	100·00	,,

And yet typhus is not invariably fatal in advanced life. The following is quoted from the "Medical Report of Cork-street Fever Hospital," for the years 1820–21, written by Dr. William Stoker, grandfather of Mr. William Stoker, now one of the Professors of Surgery in the Royal College of Surgeons in Ireland:—"A very remarkable case of malignant typhus fever passed through the wards under my care in the month of January (1821). It was that of a man of the name of Owens, **104 years of age**, husband to the woman of that name, whose death is given in the foregoing tabular obituary. This man's fever continued for three weeks,

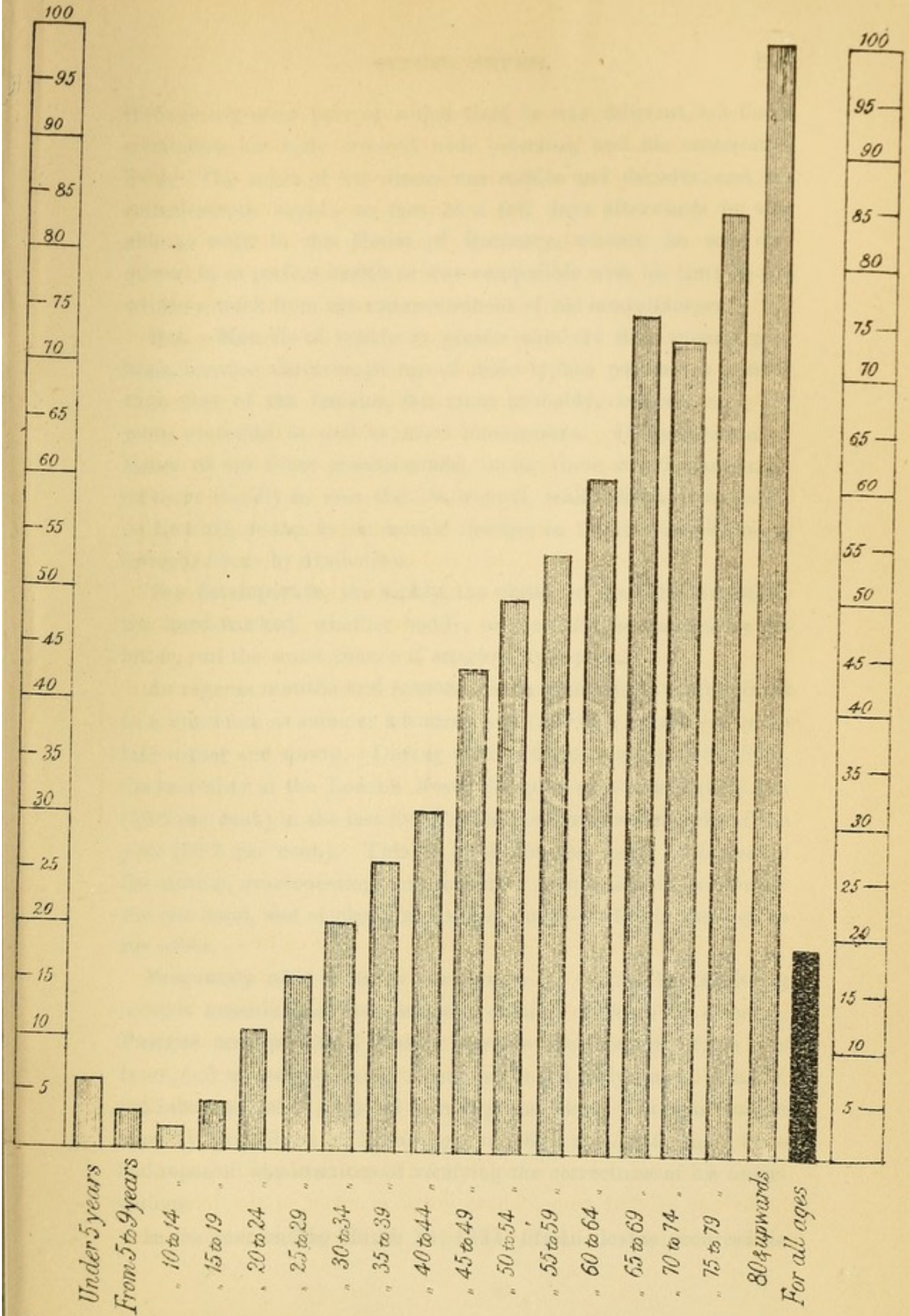
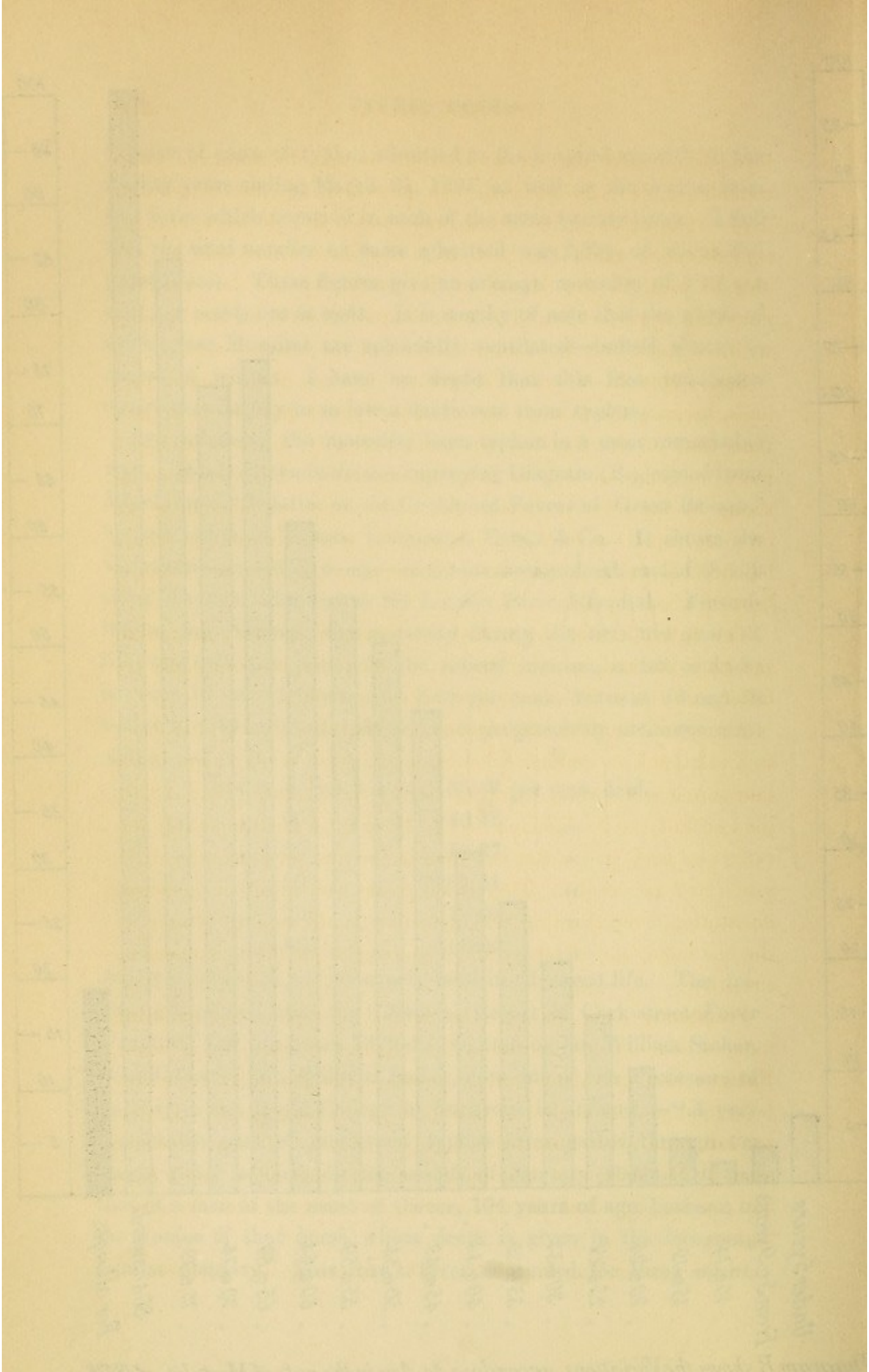


Diagram B shows the Variations according to Age in the rate of Mortality of 1813 cases of Typhus Fever, admitted into the London Fever Hospital. (Compare with Diagr. D.)



through a greater part of which time he was delirious, his limbs tremulous, his body covered with petechiæ, and his extremities livid. The crisis of his illness was sudden and decisive, and his convalescence rapid; so that in a few days afterwards he was able to walk to the House of Recovery, whence he was dismissed in as perfect health as was compatible with his time of life within a week from the commencement of his convalescence."

Sex.—Men die of typhus in greater numbers than women, perhaps, because the average age of male typhus patients is greater than that of the females, but more probably, because men are more muscular as well as more intemperate. Disintegration of tissue by the fever process would, under these circumstances, go on more rapidly in men than in women, while elimination would be checked, owing to the morbid changes in the liver and kidneys, brought about by alcoholism.

The **intemperate**, the **sickly**, the **obese**, or the **very muscular**, the **hard-worked**, whether bodily, or mentally, but especially the latter, run the worst chance if attacked by typhus.

As regards **months and seasons**, the mortality from typhus drops to a minimum as summer advances, and rises to a maximum in the late winter and spring. During twenty-three years (1848–1870), the mortality at the London Fever Hospital was considerably less (16·2 per cent.) in the last five than in the first seven months of the year (20·7 per cent.). This is, no doubt, the result of increased destitution, overcrowding, and defective ventilation in winter on the one hand, and of plenty and a more open-air life in summer on the other.

Pregnancy adds little to the danger of typhus, but **suckling** induces anæmia and increases the chances of death by asthenia. **Fatigue and privation** greatly increase the mortality from this fever, and so does neglect of treatment in the early stages. Lastly, **too late removal to hospital** is most prejudicial. This was insisted upon by Dr. Alison, of Edinburgh, in 1844 and 1849, and I have had repeated opportunities of verifying the correctness of his observations.

In the year ending March 31, 1884, fifteen deaths occurred in

Cork-street Fever Hospital before the deceased patients had been forty-eight hours under treatment in the hospital. Six out of these fifteen cases were suffering from typhus. "Dying on admission" is a grave indictment against those who were responsible for the removal of the unhappy sick to hospital in their last hours. On July 5, 1882, I drew the attention of the Managing Committee of Cork-street Hospital to the circumstances under which the first death which occurred in the hospital within the official year, beginning on April 1, had taken place. On the afternoon of July 1, a married woman, aged forty, was sent in on the *fifteenth day* of bad petechial typhus with bed-sores, and in an extremely prostrate condition. She lived for four and a half days after admission. At the time, I expressed the opinion that a patient, of middle age also, so far advanced in typhus of a severe type, could not be safely moved to hospital, and should have been treated and nursed at home. On August 7, 1883, I again called the attention of the Committee of the Hospital to a very similar case. A married woman, aged forty-nine, was sent in on the tenth day of severe typhus, during a violent thunder-storm. She died twenty-four hours after admission. Can anything more deplorable be imagined than the want of judgment exhibited in this and other "cases sent in when beyond recovery?"

Pathological Lesions.—Cadaveric rigidity is of **short** duration in typhus, and **putrefaction** takes place **rapidly**. **Emaciation** is considerable, but much less than in typhoid fever. Speaking of the anatomical lesions met with in fatal cases of typhus fever, Murchison says that the patient usually dies before the body has had time to become much emaciated. Dr. Cayley, however, quotes Dr. Hermann, of the Obuchow Hospital, St. Petersburg, to prove that a considerable loss of weight takes place during an attack of typhus. That physician found that the average daily loss of weight ranged from ½ lb. to 3 lbs. The greatest total loss was 31 lbs. A widespread **congestion** (passive hyperæmia) is the most constant and noticeable *post-mortem* appearance in typhus.

In connection with the several systems of the body, the following additional pathological appearances may present themselves:—

1. **The Integumentary System.**—The petechiæ of typhus remain visible after death. Bedsores may occasionally be observed, and less frequently spontaneous gangrene. Rarely, lymphatic and bubonic swellings may be present. There is much livid discoloration of the surface, and the green tinge of decomposition sets in very early after death.

2. **The Respiratory System.**—The chief features are traces of a widespread catarrhal inflammation of the air passages (catarrhal laryngitis, tracheitis, and bronchitis), hypostatic consolidations in the lungs, rarely pneumonic consolidations, and pleuritis with purulent effusion.

3. **The Circulatory System.**—The blood is profoundly altered, being feebly coagulable, often staining the endocardium and the intima vasorum. Lebert[a] says that the blood found in the heart and larger veins usually forms a black pultaceous, soft clot, in which there is but little distinctly coagulated fibrin. Examined under the microscope, rouleaux are found wanting, and the red blood-corpuscles are crenated and misshapen. The muscular tissue of the heart is softened and friable. Rokitansky[b] held that this softening was "a simple diminution of consistence, not depending upon any disturbance of texture." But cloudy swelling and granular fatty degeneration have been observed by all modern pathologists. The appearances have been already described (see Chapter XVIII., page 178).

4. **The Nervous System.**—The **membranes of the brain** are **highly vascular**, but this is not to be regarded as a token of inflammation—the congestion is, in fact, passive or mechanical, and depends on heart-failure or pulmonary obstruction. In rare instances deposits of lymph or pus, no doubt, indicate the presence of recent inflammation. Murchison, Stokes, and other authorities rightly hold that no relation exists between the vascularity of the cerebral membranes and the cerebral symptoms observed during life.

"The assumption of inflammation of the brain in the presence of

[a] Von Ziemssen's *Cyclopædia of the Practice of Medicine.* Art. "Typhus Fever." Vol. I., page 335.

[b] *Pathological Anatomy.* Sydenham Society. 1852. Vol. IV., page 171.

violent nervous symptoms in fever," wrote Dr. Stokes,[a] "constitutes one of the greatest dangers to which young physicians are exposed, when they come to deal with the most formidable complication of the disease—aye, and old physicians, too, whose clinical education has been imperfect. I have known of the application of leeches to the head in an advanced case of cerebral fever with delirium ferox to be followed by sudden sinking and death."

Increased **effusion of serum** within the cranium is, according to Murchison, one of the most frequent morbid appearances in typhus. The most common seats of this effusion are beneath the arachnoid and in the lateral ventricles, and sometimes in the cavity of the arachnoid. The serum is transparent or sometimes apparently opalescent, owing to slight opacity of the superposed membrane. It *may be* colourless or straw-coloured. It does not contain flakes of lymph or exudation-corpuscles.

There is no relation between the amount of effusion and the intensity of the symptoms of brain trouble, nor is the increased serosity a sign of inflammation. Murchison thinks that in typhus the brain wastes from malnutrition and the effusion takes place to fill up space. It would be more correct to say that from the congested vessels, with badly-nourished walls, and owing to lessened pressure upon them by the shrunken brain, effusion takes place.

The **cerebrum** and **cerebellum** are generally **healthy.** They may be highly vascular, a condition shown by diminished consistence, a darker tint of the grey matter, and numerous blood-points on section of the white matter. Softening of the brain has been occasionally observed, and Barrallier[b] has called attention to a remarkable indistinctness of the *arbor vitæ* of the cerebellum in some cases.

Similar appearances to those described in connection with the brain are less frequently seen in the spinal cord.

Nothing definite has yet been ascertained as to the condition of the **sympathetic system** of nerves in typhus. Marmy found many of the ganglia softened, and Beveridge found the cervical ganglia

[a] *Lectures on Fever.* London: Longmans, Green & Co. 1874. Page 279.
[b] *Du Typhus épidémique à Toulon.* Paris. 1861. Page 106.

enlarged and dense from the deposit of an amorphous granular matter.

5. **The abdominal viscera.**—The characteristic lesions of enteric fever are wanting. The kidneys are not uncommonly hyperæmic and enlarged, while the tubes are engorged with blood and stuffed with granular epithelium. **Acute nephritis** is thus unmistakably present in not a few instances.

The pathology of the "**Typhoid State**" is not yet clearly made out. Murchison thinks it is very possible that the condition to which this name is given may have a common origin in all the diseases in which it shows itself, or may be due to the **accumulation** in the blood of the **products of disintegrated tissue** as the result of the primary malady. The connection between the typhoid state and the presence of urea, carbonate of ammonia, or other nitrogenous detritus in the blood is, according to Murchison, a subject which deserves further investigation.

James Andrew[a] considers that the chief primary cause of the condition may be the **injurious influence** of a **high internal temperature** upon the central nervous organs, but this same temperature acts directly also upon the parenchyma of glandular organs, and upon the muscular fibre both of the heart (Stokes) and of the voluntary muscles (Zenker). The disorder of the nervous centres must be greatly increased by, if not sometimes decidedly due to, the changes in the composition of the blood, and in the forces of the circulation.

Murchison admirably sums up the **pathological appearances** in typhus as follows:—

"1. There is no lesion constant in, or peculiar to, typhus.

"2. The intestines never exhibit the peculiar lesions [almost] invariably present in typhoid fever, and the mesenteric glands are not enlarged.

"3. No evidence of acute inflammation is found in the brain or its membranes, to account for the cerebral symptoms.

"4. The chief morbid appearances are: a fluid condition of the

[a] *Quain's Dictionary of Medicine.* 1883. Art. "Typhoid State."

blood, atrophy of the brain, with increase of intra-cranial fluid, granular degeneration of the sympathetic nerves; atrophy, with granular or waxy degeneration of the muscles and heart; enlargement and congestion of the liver, spleen, pancreas, and kidneys, with a swollen, granular state of the gland-cells; bronchial catarrh and pulmonary hypostasis. The relative frequency of these lesions varies at different times and places; none are of constant occurrence, or peculiar to typhus."

CHAPTER XXXI.

The Treatment of Typhus.

Prophylactic and Curative Treatment (or "Management"). Preventive Measures: Personal cleanliness, good food and air-space, ventilation. Management: hygienic measures adopted at Cork-street Fever Hospital, Dublin. Nursing. Medicinal treatment must be purely symptomatic. Water Treatment. Quinine. Alcoholic Stimulants. Food.—Treatment of Complications and Sequelæ: Pulmonary congestion, bronchitis, paresis, incontinence of urine, convulsions, bedsores, phlegmasia and thrombosis, œdema. Convalescence: costive bowels. Tonics. Change of Air.

This topic has already been dealt with in detail in the Chapters on the General Principles of Treatment.

"**It is easier to prevent Typhus than to cure it,**" said Murchison, and if thorough effect could be given to all the preventive measures described in Chapter IV., typhus fever would, in all probability, soon cease to exist. Nothing is more striking than the diminution of this fever of late years—even in those countries (such as Ireland), and towns (such as Cork, Dublin, Glasgow, and Liverpool), where the repeated epidemics in the past caused it to rank as an endemic disease.

In the recognition of the facts that destitution, overcrowding, and deficient ventilation, enormously predispose to typhus, lies the key to the **prophylaxis** of the disease. Personal cleanliness, an abundant supply of good, wholesome, food, a sufficient cubical air-space per head of the population (500 cubic feet, at least), and free ventilation, which means the supply of 3,000 cubic feet of fresh air per head every hour, are the best preventives.

Those sick of typhus should be treated in large airy wards or rooms—1,500 to 2,000 cubic feet being allowed to each patient—and the beds should be at least six feet apart.

We have already seen that, if a single fact is well established in the life-history of the contagium of typhus, it is that the virus is most readily destroyed or rendered inert by contact with atmo-

spherical air, presumably through oxidation. If this is so, what vast magazines of typhus-poison might be rendered powerless for mischief in every gentle breeze, not to speak of a more violent tempest, provided only doors and windows are not too closely barred.

After thus referring to the destruction of the virus of typhus fever by oxidation, through the natural agency of the currents of the atmosphere and of strong winds, it may be of interest to describe the therapeutical means adopted by my colleagues and myself during the last epidemic in Cork-street Fever Hospital, with a view of neutralising the fever poison, when it was actually at work in the patient's system.

First, attention was constantly directed to the **ventilation** of the fever wards. In severe cases, where the eruption was copious and dark (petechial), a strong current of fresh air was allowed to blow over and around the patient whenever the state of the weather at all permitted it.

Lebert[a] says on this point that in a recent epidemic at Breslau he found it to be an excellent plan, even during the severest cold of winter, to keep the windows open during part of the day and night. The patients bore it well so long as the fever lasted, though they proved very sensitive to cold after the defervescence.

The weight of bed-clothes was reduced to a minimum, so that the fresh air might easily reach the surface of the whole body. It might be supposed that the exposure would give the patients cold; but experience proves that this is not so, and I am convinced that in a vast majority of cases the bronchial catarrh and other pulmonary affections of typhus are not the result of cold, but of passive hyperæmia of the bronchial mucous membrane and of the parenchyma of the lungs—that, in fact, these lesions belong to the essential pathology of the disease.

Secondly, directions were given to the nurses in all bad cases, with profuse eruption, **to sponge** the entire surface of the body twice or three times a day **with vinegar and warm water.**

[a] Art. "Typhus Fever," in von Ziemssen's *Cyclopædia of the Practice of Medicine.*

Thirdly, solution or tincture of the **perchloride of iron, chlorate of potassium** in moderate doses, or **quinine**, was given in nearly all such cases, either separately or in combination, and with remarkably good results.

Under this hygienic treatment several very grave cases progressed favourably, and it is an interesting fact connected with my own practice in the hospital, that I seldom felt called upon to order for these patients any large quantity of alcoholic stimulants.

Bearing in mind the early and great prostration of typhus, the patient should take to bed as soon as possible in a cheerful, large, airy apartment, with two bedsteads in it—one for day and one for night.

The best form of bed for a typhus patient is a hair-mattress laid upon a woven-wire spring mattress. The bed-clothes should be light. The patients' head should be kept as cool as possible, but his feet should be warm.

There is no disease in which the services of a trained, experienced, strong, and judicious **nurse** are more needed than in typhus. In the ninth of his "Clinical Lectures on the Practice of Medicine," Dr. Graves gives excellent advice on this point. He says:—"You should never, if possible, undertake the treatment of a case of fever where the friends or relations of the patient supply the place of a regular fever nurse. The mistaken tenderness of relatives and their want of due firmness, presence of mind, and experience, will frequently counteract your exertions and mar your best efforts. Affection and sorrow cloud the judgment, and hence it is that very few medical men ever undertake the treatment of dangerous illness in the members of their own families. . . . Again, it will not do to have a nurse who has been usually employed in other diseases; your assistant must be a regular fever nurse, and the man who undertakes the treatment of a long and dangerous case of fever without such an assistant will often have cause to regret it. . . A fever nurse has a vast deal in her power; if an enema is to be administered, the patient will be much less disturbed and annoyed than if it were given by an unskilful person. The mere handling of a patient—the moving of him from one bed to another—the

simple act of giving him medicine and drink—the changing of his sheets and linen—the dressing of his blisters—and a thousand other offices, can be performed with advantage only by an experienced nurse. Always bear in mind that it is of the utmost importance to economise the patient's strength in fever. The very act of lifting him up, or removing him from one side to another, tends to produce exhaustion. In the advanced stages of fever, the services of a properly qualified nurse are inestimable. Then there is the moral management of the patient, and this is an office which no one can undertake unless qualified by experience, and a correct knowledge of the habits of persons labouring under such forms of disease. Everyone admits the value of moral superintendence in the treatment of the insane. Now there are very few patients who are not in a state analogous to insanity, for a longer or shorter period, during a course of typhus fever. There is a necessity for moral management in fever as well as in insanity, and this is understood only by an experienced nurse."

Both in hospital and private practice, the nurse, or nurses—for there should be a day nurse and a night nurse—should keep a written record, for the information of the physician, of the times at which food and stimulants have been given, the bowels have moved or water has been passed; of the changes in the nature and character of the symptoms from visit to visit of the physician; and of the behaviour of the temperature and the rate of the pulse and respirations at stated intervals already agreed on.

So far as the physician is himself concerned, every case of fever should be **visited** at least **twice a day**—morning and evening—during the acute and critical stages, for a few hours may mean life or death to the sufferer.

The **medicinal treatment** of typhus is purely **symptomatic**, for as yet we possess no specific for this disease, if we except **fresh air.**

"Although," says Murchison, "many practitioners have at different times proposed to cut short an attack of typhus by such heroic remedies as blood-letting, the cold affusion, emetics, and quinine, we possess as yet no such specific" as will arrest its progress or shorten its duration.

Von Hildenbrand, early in the nineteenth century, observed—"No method yet known, whether rational or empirical, can *cure* the contagious typhus, either in a direct or in an indirect manner, or even abridge its ordinary and natural course, which is about fourteen days."

To the same effect Stokes[a] declared that "the treatment of fever, whether it be typhus or typhoid, is reducible to a simple formula, and is essentially the same in both types of disease. We know of no cure for fever; no man has ever cured it. It is, however, curable spontaneously. If you leave it to its own course, it is capable of curing itself. It will spontaneously subside. Remembering the law of periodicity, the great object of the physician should be to gain time, preserving the patient from the dangers which threaten him, which belong to this special state of life. If he can be kept alive to the 14th day, the 21st, the 36th, or even the 60th day, recovery will probably ensue. Every day, every hour of existence preserved and sustained is a great gain. The risks that he runs are due to debility or to the influence of the secondary affections. We, so to speak, cure the patient by preventing him from dying. . . . Herein lies the secret of the treatment of fever. We watch the progress of the disease throughout its varying phases; we meet by judicious treatment, as they arise, the symptoms of secondary and local malady; we sustain the system as far as practicable; we preserve the sufferer at the least expense to the constitution; and we wait patiently until the hour shall strike when, in accordance with the mysterious law of periodicity, the fever shall have departed and convalescence shall have begun."

Murchison employs a striking metaphor when, to the same effect, he says—"A patient with typhus is like a ship in a storm; neither the physician nor the pilot can quell the storm; but by tact, knowledge, and able assistance, they may save the ship."

Mosler, of Greifswald, says that he has up to the present tried the methods mentioned by Murchison for the treatment of typhus: **the water treatment**, the use of large doses of **quinine, alcoholic**

[a] *Lectures on Fever.* 1874. Pages 83 and 84.

stimulants, and **nourishing food.** Suitable combinations of these measures are recommended by him as the best way of dealing with typhus, and by means of them he obtained brilliant results in an epidemic observed by him in 1867, when only 8 deaths occurred among 94 patients, the mortality being 8·7 per cent. compared with the average laid down by Griesinger as ranging between 15 and 20 per cent. In the same epidemic, before suitable treatment had as yet come into use, of 36 patients 20 died, which gave a death-rate of 55 per cent. We are, therefore, justified in concluding that even the mortality in typhus may be reduced to an inconsiderable figure by a persistent water-treatment, carried out with the aid of the thermometer, especially when combined with the use of quinine and of the other remedies already mentioned.

The administration of alcoholic stimulants in typhus **should not be a matter of routine.** The sagacious physician owes it to his patient and to his own reputation to decide, in each case after full consideration, whether these remedies—potent for evil no less than for good—are to be prescribed or withheld. Stokes's views on this subject, contained in the twenty-seventh of his *Lectures on Fever* (Edition of 1874,) are thoroughly sound and philosophical. After pointing out that, in his practice, cases had occurred in which either no wine was used, or it was sparingly employed, or it was not ordered until after the middle period of the fever, he goes on to speak of the **anticipative treatment** by stimulants—that is, their administration at an early stage of fever, when the sagacity of the physician enables him to foretell the occurrence of great prostration of nervous energy. Under such circumstances he gives stimulants *by anticipation.* In adopting this practice we follow the old maxim, "venienti occurrite morbo," and we take into account the character of the prevailing epidemic, and the previous medical history of the patient. The effect of each dose of alcohol should be carefully watched and noted.

The management of some of the **complications** and **sequelæ** of typhus remains to be discussed.

1. **Pulmonary Congestion** and **Bronchitis** are best treated by poulticing, dry cupping, and the application of stimulating liniments

to the chest by rubbing—for example, compound camphor liniment, acetic turpentine liniment, and so on. Iodine may also be applied in the form of an oleate, or in combination with water and glycerine—the stronger preparations destroy the absorbent power of the skin. Internally, the most useful remedies are iodide of potassium and bark (in convalescence), quinine and iron, quinine and arsenic in pill, digitalis. In gangrene of the lung, free stimulation, chlorate of potassium and borax, large doses of quinine, guaiacol (which is obtained by the destructive distillation of guaiacum resin and is contained in beech creasote to the amount of 60 to 90 per cent.), and antiseptic inhalations of carbolic acid, creasote, tar vapour, &c., are the most suitable remedies.

2. **Paresis** after the fever requires a generous diet, mineral tonics, strychnin, and massage, with galvanism, shower-baths, and sea-bathing in summer. Tincture of perchloride of iron is an excellent remedy in **incontinence of urine**, and in the female this infirmity is often at once relieved by cauterising the orifice of the urethra with nitrate of silver.

3. In addition to the measures suggested in Chapter V. (page 63) for the relief of **convulsions**, it is right to mention that Hudson, in 1837, recorded a case of recovery after two severe fits of convulsions in typhus, the treatment adopted consisting in the abstraction of ten ounces of blood by wet cupping from the neck and purging with calomel. In a case of typhoid fever in a lady, complicated with epileptoid convulsions, the treatment adopted by Hudson consisted in withholding wine, cutting off the hair, applying cold to the head and leeches behind the ears; also, in the exhibition of grain doses of calomel at short intervals. This lady recovered perfectly. She never had epilepsy before her fever, nor did she afterwards suffer from it.

4. **Bedsores** may be avoided to a large extent by careful nursing and by the use of an annular air-cushion, a water bed, or a spring bed. The threatened parts should be protected by being kept dry, and painted twice a day with a solution of one part of sheet gutta-percha in eight parts of pure chloroform, or equal parts of white of egg and rectified spirit, or equal parts of collodion and castor-oil.

When the sores have formed, they should be washed and dressed antiseptically. An excellent stimulating application is composed of two parts of castor-oil and one of balsam of Peru spread on lint, or carbolised-oil (1 in 40, that is, $2\frac{1}{2}$ per cent.). These dressings should be protected by a layer of oiled silk, or else covered with some poultice less septic than linseed meal, such as the yeast, carrot, chlorine, or charcoal poultice.

5. When **phlegmasia** or **thrombosis of the femoral veins** occurs, the foot and leg of the affected side must be raised, and a long wide flannel bandage should be applied from the feet upwards. If thrombosis is present, denoted by a hard painful cord in the situation of the femoral vein, strips of lint smeared with equal parts of glycerine and extract of belladonna may be laid along the affected part, and covered with the flannel swathe as before.

6. **Œdema of the lower extremities** generally yields to a generous diet, and tonics like iron and quinine and strychnin.

The **dieting** of a typhus fever patient should be conducted on precisely the same principles as those which guide us in managing an enteric fever patient. The reader is, therefore, referred to the Chapter on the "Curative Treatment of Enteric Fever," where the subject is considered in detail.

During **convalescence**, the patient recovering from typhus should be warned against assuming the upright position too soon, or exposing himself to cold. The returning appetite should be held in check for the first two or three days. On the third day, with a clean tongue and a quiet pulse, a piece of boiled white fish, or chicken, or the hollow part of a tender mutton chop, may be allowed. **Costive bowels** should be relieved by sipping water in mouthfuls frequently, or by the administration of enemata of cold water. **Tonics** may be given with advantage; but of these the most potent are exercise in the open air, and change of residence to the country, the seaside, or the mountain slope.

CHAPTER XXXII.

Relapsing, Famine, or Spirillum Fever.

Nomenclature—Definition—Ætiology (historical sketch)—Mode of Prevalence—Predisposing Causes—Geographical Distribution—Exciting Cause—Bacteriology—*Spirillum Obermeieri*—Inoculation Experiments—One Attack of Relapsing Fever confers no immunity against a second.

In describing this form of Continued Fever, I am at the serious disadvantage of never having seen a case of the disease in my hospital or private practice. This fact, after well-nigh twenty years' experience in two hospitals—one of them, Cork-street Fever Hospital, the great epidemic hospital of the south side of Dublin—leads me to dispute in the strongest manner the accuracy of Hirsch's unqualified and dogmatic statement that "there can be no doubt that the disease is endemic in Ireland."[a] Although this disease is personally unknown to me, the descriptions of relapsing fever by Murchison, H. Lebert, C. A. Ewald, and others, are so admirable that it becomes possible to picture to oneself what this form of continued fever must be in its clinical aspect.

Nomenclature—Relapsing Fever, Famine Fever, Spirillum Fever (Vandyke Carter, 1882), Typhinia (Farr, 1859), Bilious Typhoid Fever, Yellow Fever of the British Islands (Graves and Stokes, 1826), "A Five Days' Fever with Relapses" (Rutty, 1770), Synocha (Cullen, 1769), Miliary Fever (Ormerod and Watson, 1848).

Germ.—Recurrens (Ewald), Rückfallsfieber, recurrirendes Fieber Die Hungerpest, Armentyphus, Febris, *vel* Typhus Recurrens (Hirsch, 1859), Rückfallstyphus (Lebert, 1870), Das wiederkehrende Fieber (Obermeier, 1869).

French—Fièvre à rechute, Typhus à rechute, Fièvre recurrente, Fièvre à récidive.

[a] *Handbook of Geograph. and Historical Pathology.* New Syd. Soc. Vol. I., page 614.

Ital.—Febbre a recidive, Febbre ricorrente.

Span.—Fiebre recidiva.

Swedish.—Recurrenta Feber (Cf. *Förhandlingar vid Svenska Läkare-Sallskapets*, 1865, p. 110, and 1868, p. 73).

Norwegian or *Danish.*—Recidiv-Feber, Galbefeber.

Dutch.—Galkoorts (i.e., *Biliary Fever*).

Definition.—A specific contagious febrile disease, characterised by the presence in the blood during the febrile paroxysm of a spiral bacterium, called after its discoverer the *Spirillum Obermeieri;* by a critical defervescence after 5 or 7 days, which is followed by a remission lasting 7 or 8 days, and by a relapse on or about the fourteenth day, lasting some 3 days. This fever prevails chiefly in the form of an epidemic during seasons of scarcity and famine. It is unaccompanied by an eruption, but jaundice is present in some 20 per cent. of the cases. The death-rate is not high, being from 5 to 7 per cent. After death, the liver and spleen are commonly found enlarged. A second, or even a third relapse may occur.

Ætiology.—The first minute account of an epidemic of Relapsing Fever dates from the years 1739 and 1741, and we owe it to Dr. Rutty.[a] He described it as "a fever of six or seven days' duration, terminating in a critical sweat. . . .; here the patients were subject to a relapse even to a third or fourth time, and yet recovered." It followed hard upon the scarcity caused by seasons of unexampled severity in the years 1739–40. "It was far from being mortal," adds Rutty, who goes on to say—"I was assured of seventy of the poorer sort at the same time in this fever, *abandoned to the use of whey and God's good providence*, who all recovered, while those who had generous cordials and great plenty of sack perished."

In an historical sketch of the fevers which were "epidemical" in Ireland during the last and at the commencement of the present century, Drs. Barker and Cheyne, writing in 1821, observed: "Certain it is, that the fever in 1800 and 1801 very generally

[a] *A Chronological History of the Weather and Seasons, and of the Prevailing Diseases in Dublin, with their various Periods, Successions and Revolutions, during the space of forty years.* Dublin. 8vo. 1770.

terminated on the 5th or 7th day by perspiration ; that the disease was then very liable to recur ; that the poor were the chief sufferers by it ; and that it was much more fatal amongst the middling and upper classes in proportion to the number attacked."

Barker and Cheyne[a] were the historians of the great epidemic of 1817–1818 ; Graves and Stokes of that of 1826–1828. Other outbreaks occurred in Great Britain and Ireland in 1842–1843 and 1847–1848 ; as well as in Silesia in the last-named years (Dümmler and von Bärensprung). A very full and excellent account of the epidemic of 1847 in Ireland will be found in the 7th and 8th Vols. of the New Series of the *Dublin Quarterly Journal of Medical Science* (1849). There was a great epidemic at St. Petersburg and Odessa in 1863, and a second epidemic occurred in Silesia in 1867 and 1868. In the latter year relapsing fever reached Berlin for the first time, frequently recurring up to 1873. In 1868 relapsing fever re-appeared in Great Britain also, an epidemic beginning in London in July of that year, and lasting until June, 1871. In Liverpool relapsing fever caused 355 deaths in 1870, 207 deaths in 1871, and 25 deaths in 1872.

Seventy-seven deaths from relapsing fever were registered in England in the ten years 1880–89. Of these, 33 occurred in males and 44 in females. A few deaths occurred every year in Scotland up to 1879.

So far as Ireland is concerned, the Registrar-General, Dr. T. W. Grimshaw, has kindly furnished me with the following Return of the Number of Deaths from Relapsing Fever, registered in Ireland during the ten years, 1881–90 :—

1881	...	1	1886	...	3
1882	...	1	1887	...	0
1883	...	1	1888	...	5
1884	...	2	1889	...	2
1885	...	4	1890	...	3

In reference to this table, it is to be remembered that the Regis-

[a] *An Account of the Rise, Progress, and Decline of the Fever lately Epidemical in Ireland.* Dublin : Hodges & M'Arthur. 1821.

trar-General is not responsible for the diagnosis, and the eminently contagious nature of relapsing fever suggests grave doubts as to its accuracy in the cases embodied in the return.

As regards its **mode of prevalence**, Murchison draws the following conclusions from the history of the disease:—

1. Relapsing fever is an **epidemic disease** in a stricter sense than even typhus. It may disappear entirely for years from those places where at other times it rages most fiercely.

2. **Epidemics of relapsing fever** have usually **co-existed with epidemics of typhus**, and have always appeared under circumstances of distress or famine.

3. In mixed epidemics the relative proportion of typhus and relapsing cases has varied at different times and places; but, as a rule, the proportion of relapsing cases has been much greater at the commencement than towards the close of the epidemic, and with the advance of the epidemic typhus has taken the place of relapsing fever.

Predisposing Causes.—Hirsch[a] shows clearly that the prevalence of relapsing fever is not dependent upon climate, is little influenced by weather or season, and seems to bear no relation to the soil, as regards altitude, configuration, geological formation, and the like.

The disease, further, shows no preference for race or nationality.

As regards **sex**, of 2,115 cases admitted into the London Fever Hospital in twenty-three years (1848–70), 1,279 were males, and only 836 were females. Murchison thinks that this curious result is due to the fact that far more males than females belong to the class of tramps and vagrants, who constitute a large proportion of the cases of relapsing fever.

This disease attacks **all ages**—in the London Fever Hospital the youngest patients were two boys aged 5 months, the oldest was a man aged 75. The percentages of all the cases were—under 15 years, 18·9; from 15 to 25 years, 35·9; 25 years and upwards, 45·2. The mean age of typhus patients was (as already men-

[a] *Handbook of Geographical and Historical Pathology.* New Syd. Soc. Vol. I., pages 606, *et seq.*

tioned) 29·33 years, or about 3 years above the mean age of the whole population; but the **mean age** of relapsing fever patients was only **24·41 years**, or two years under that of the population at large.

While its area of distribution, as we will show presently, is much more limited than that of typhus, it is an undoubted fact that **relapsing fever and typhus often coincide** remarkably in time and place, relapsing fever appearing to be associated in a very conspicuous manner with epidemics of typhus. But, as Hirsch points out, the most striking analogy to typhus which relapsing fever manifests, comes out in the relation of the origin of the disease to all those conditions of social misery which play so decided a part in the history of typhus outbreaks. As Engel[a] says of it in his account of "the Bukowina Fever," relapsing fever is peculiarly a "**morbus pauperum.**"

As regards the **Geographical Distribution** of relapsing fever, in Europe, the British Islands (and particularly Ireland), Russia, and Germany (with the exception of the south), Austria, the Levantine States, and the islands of the Eastern Mediterranean, have been most frequently and most severely visited. On the other hand, Scandinavia, Switzerland, France, Italy, and the Iberian Peninsula, seem to have been hitherto exempt, or nearly so, from visitations of the disease. From Norway, however, Danchersen reports (*Norsk Magazin for Lægevidenskaben*, 1865, xix., p. 76) the prevalence of epidemics from 1858 to 1861, and again in 1865, in the medical or dispensary district of Vadsö, inhabited by a poor fishing population.

In Ireland, during the epidemic of 1826–28, relapsing fever often assumed the character of bilious typhoid, so much so that Graves and O'Brien[b] were constrained to explain cases of that kind as yellow fever.

Outside Europe, one of the most extensive centres of relapsing fever and of bilious typhoid is **India.**

[a] *Oest. med. Jahrb.* 1847. Vol. III., p. 249.

[b] *Transactions of the Med. Assoc. of the King and Queen's College of Physicians in Ireland.* 1828.

An extensive epidemic, described by Dr. Vandyke Carter,[a] who gave the disease the name of "**Spirillum Fever**," occurred in 1877–78–79 in Western India, coincident with a period of famine due to prolonged drought and complete failure of the rice-crop.

The disease also occurs along the North African Coast, including Egypt and Algeria, as well as in Abyssinia. In 1864–65 it prevailed in certain parts of China. Since 1844, it has occasionally appeared in some of the Eastern States of the North American Union. Central and South America, Australia, and Polynesia, have apparently escaped the disease up to the present.

Exciting Cause.—That there is a specific poison in relapsing fever, communicable from the sick to the healthy, is proved beyond all doubt by evidence similar to that which has been adduced in the case of typhus. At one time (in 1847) Virchow held that the disease was not contagious, but he must have abandoned this heresy, for Hirsch[b] says: "There exists no doubt whatever in the experience of *all* observers, and according to the experimental inoculations, that relapsing fever (or bilious typhoid) is *contagious*."

Bacteriology.—Since Obermeier's discovery in 1873,[c] no doubt remains that for relapsing fever, at all events, there exists a specific germ, or (as Lebert calls it) a **protomyces**.[d] Obermeier's spirilla differ only in size from similar filaments originally discovered in stagnant water by Ehrenberg in 1838, and named by him **Spirochæte plicatilis** (*σπεῖρα, a coil; χαίτη, a hair*). Another spirochæte was found in the mucus of the gums by Steinberg in 1862. This he named **Spirillum buccale**. It has since been described by F. Cohn under the name **Spirochæte denticola**. The three forms are closely allied and may be regarded as different species of one genus or different varieties of one species (Murchison). The following excellent account of what is now known as the **Spirillum**

[a] *Spirillum Fever of Bombay.* 1877.

[b] *Loc. cit.* Page 614.

[c] *Vorkommen feinster, eine Eigenbewegung zeigender Fäden im Blute von Recurrenzkranken. Ueber Pilzparasiten im Blute bei Recurrens*, x., 1873. *Loc. cit. Centralblatt für die med. Wissenschaften*, xi., 145. 1873.

[d] Gk. *πρῶτος, first; μύκης, a fungus.*

of Relapsing Fever, or the **Spirochæte Obermeieri** (Cohn) is given by von Jaksch :[a]—

The **spirillum** was first noticed by Obermeier in the blood of a patient suffering from relapsing fever. It has since been seen by many observers, but most authorities are agreed that it is to be found **only during the paroxysms** of the disease, and that as the temperature falls the bacilli disappear.[b] When a specimen of blood containing them is placed under the microscope, the bacilli appear as long and very delicate unsegmented threads twisted into spirals. Their average length is about six or seven times the diameter of a red blood-corpuscle. They have a brisk vibratile movement in the direction of their long axis. This motion, when the blood is examined with a low power, gives to the eye a peculiar impression of disturbance, and will immediately lead the practised observer to look for the presence of spirilla. If he then increases the power, and still better, if he employs an oil immersion lens with Abbe's condenser and a small diaphragm, the spirilla come clearly into view. These bodies are extremely sensitive to reagents of all kinds. Even the addition of distilled water will cause them to disappear.

The number of spirilla which are to be seen together in a specimen of blood varies greatly, and often bears no relation to the intensity of the fever.

If the blood is examined in the intervals of the disease, provided another paroxysm be impending, it displays peculiar refractive bodies resembling diplococci, which are especially numerous when the paroxysm sets in; and just as it begins, they even seemed to von Jaksch, in certain cases to grow out, as it were, into short thick rods, from which the spirilla were finally evolved. Before those of von Jaksch similar observations had been made by Sarnow[c]; and, pending further confirmation, it seems probable that these bodies are the **spores of spirilla** which have so long been sought for.

[a] *Clinical Diagnosis.* By Dr. Rudolf v. Jaksch. Translated from the second German edition by James Cagney, M.D. London : Charles Griffin & Co. 1890. Pages 30 and 31.

[b] In opposition to this view, see Naunyn. *Centralblatt für Bacteriologie und Parasitenkunde.* IV. 376. 1888.

[c] *Der Rückfallstyphus in Halle a. S. im Jahre, 1879–81.* Inaugural Dissertat. Leipzig. 1882.

Since both the spirilla and the forms just mentioned have been met with as yet only in the blood of persons suffering from relapsing fever, their great importance as a **clinical test** is apparent.

Monkeys have been inoculated with success from man,[a] but inoculations of mice, rabbits, sheep, and pigs gave negative results. The spirilla were found in the blood of the inoculated monkeys in great numbers, and also in the brain, lungs, liver, kidneys, spleen, and skin. They are believed to be the cause of the disease (Crookshank).

Moschutkovsky,[b] of Odessa, was the first to attempt to produce infection by inoculating the blood of relapsing fever patients on healthy men, and he proved not only the communicability of the disease, but also the specific pathogenetic significance of the parasite. A further result of his researches was to prove the identity of relapsing fever and the so-called "bilious typhoid," a view already maintained by Griesinger and the Russian physicians on anatomical and pathological grounds.

The period of incubation in the inoculated cases was never more than 8 or less than 5 days. In monkeys, Vandyke Carter found it to vary from 30 hours to 5 days. When the disease is taken in the natural way, its latent period is on the whole shorter than that of typhus. According to Lebert, it varied at Breslau from 3 to 7 days—it was, however, oftener over than under 5 days. Murchison analysed 32 cases, and found that in not one instance did the stage of incubation exceed 12 days, while in 13 it did not exceed 5 days. In 3 cases the attack was immediate on exposure to the infection.

One attack of relapsing fever confers little or **no immunity** from a subsequent visitation. This was proved in all the great epidemics of the present century both at home and abroad, but a classical observation is that of Sir Robert Christison, Bart., who in the outbreak of 1817–19 experienced no fewer than three separate attacks within fifteen month sin his own person.[c]

[a] H. Vandyke Carter. *Lancet*, 1879. Vol. I., page 84; and 1880, Vol. I., page 662. See also Koch. *Deutsche med. Wochenschrift.* 1879. Nos. 16, 27, 30.

[b] *Centralblatt für die med. Wissenschr.* 1876. No. 11. *Petersb. med. Wochenschrift.* 1878. No. 27. *Archiv. für klin. Med.* 1879. XXIV., page 80.

[c] *Edin. Med. Journal.* January, 1858. Page 583.

CHAPTER XXXIII.

Clinical Description of Relapsing Fever.

Sudden onset.—High Temperature.—Abrupt crisis. Intermission.—Relapse.—Low mortality.—"Bilious Typhoid" (Griesinger).—"Yellow Fever of the British Islands" (Graves).—Heart murmurs.—Stages and duration.—Temperature.—Complications and Sequelæ.

Relapsing Fever usually begins with **great suddenness.** In some cases, indeed, the actual outburst of the disease may be preceded by such prodromata as *malaise*, weariness, pain and heaviness in the head, flying pains in the extremities, and thirst.

Whether such prodromata have occurred or not, the patients are suddenly seized with chills or rigors, frontal headache, and pains in the back, neck, and limbs. They complain of giddiness and noises in the ears (*tinnitus*). The vertigo rather than their weakness compels them to take to bed at once. This cold stage is quickly followed by a reaction, characterised by flushing of the face, epistaxis in many cases, a dry, hot skin, increase of pains, throbbing headache, and burning thirst. The appetite is, in most cases, completely lost; but there may be voracious appetite (*boulimia*). This was noticed in the epidemic of 1843 in the practice of the London Fever Hospital, and also in that of 1847, in Ireland, especially by Mr. Kelly,[a] of Mullingar, Co. Westmeath, The pulse becomes very quick, reaching 120 as a rule, or far exceeding even this rate. It is generally full and firm, and its rapidity is not of evil omen. On this point, Niemeyer says that the pulse is more frequent in relapsing fever than in almost any other disease.

The tongue is large and moist, rarely becoming dry, cracked and brown. The bowels are constipated; tenderness at the epigastrium is complained of. Nausea and vomiting are not uncommon

[a] *Dubl. Journ. of Med. Science.* New Series. Vol. VIII., page 64. 1848.

symptoms. A slight catarrhal jaundice may be present, but this is to be distinguished from the much more serious hæmatogenous jaundice which not infrequently renders the disease malignant (**bilious typhoid**). The vomited matter may consist of green bile, or else of blood like coffee-grounds (black vomit). Sleeplessness is a common and distressing symptom; but delirium is much less usual than in typhus.

The urine is scanty and high-coloured, and, according to Obermeier, shows that parenchymatous nephritis exists. When jaundice is present, the urine contains bile-pigment and bile-acids. Physical examination reveals considerable enlargement of both liver and spleen even at an early stage. There is **no eruption** of a specific kind, but a roseolar rash has not infrequently been observed, as well as true petechiæ in certain cases and crops of sudamina in or after the sweating stage.

The **fever** runs **very high** in relapsing fever—temperatures between 105° and 108·7° being quite common shortly before the crisis. The thermometer at the beginning rises so abruptly that within twelve or twenty-four hours of the earliest symptoms it may reach 104° to 106° F.

Towards the close of the first week—sometimes as early as the third, or as late as the tenth day—more usually on the fifth, sixth, or seventh day—all the **symptoms subside like magic.** In a few hours—eight or nine—the pulse sinks to 70, 60, 50, or even lower, and the temperature *runs* down from its acme of 106° to 108° to several degrees, it may be, below normal. This sudden crisis is ushered in by profuse sweating—the patients being literally bathed in perspiration for some hours; more rarely by diarrhœa, epistaxis, menstruation, or intestinal hæmorrhage. The perspiration which accompanies crisis has an acid reaction and "a characteristic disagreeable smell" (Cormack, 1843). Occasionally a brief spell of frenzied delirium immediately precedes the critical defervescence.

During the next few days, while the pulse remains slow, the temperature gradually rises towards normal, the tongue cleans, appetite returns, and the patients feel and declare themselves well.

In most cases, however, on or about the **seventh day** from the

crisis, without warning or assignable cause, **the relapse** sets in. This is, in fact, a repetition of the initial symptoms of the first attack. Sometimes this secondary paroxysm is even more severe than the first, so far, at all events, as high temperature is concerned; but the febrile movement is generally of shorter duration, lasting only three or four days, and terminating by **another crisis**, with its attendant phenomena.

Now and again, there is a **second relapse**, coming on about the 21st day, and lasting two or three days. Rarely, even a third or a fourth relapse may occur; but, on the other hand, there may be no relapse at all in exceptional cases, the passing off of the first paroxysm terminating in convalescence without further suffering.

By far the most common **ending** of relapsing fever is **in recovery.** The **death rate** in most epidemics is only 2 or 3 per cent., and it rarely exeeeds 6 or 8 per cent. When death does occur, it is the outcome of—(1) **collapse** and general paralysis during the paroxysm; (2) **exhaustion** during the interval; (3) **some complication** or secondary affection—such, for example, as acute pneumonia or nephritis with uræmia.

Pregnant females invariably **abort**, and **often die**, in relapsing fever.

Bilious Typhoid (Griesinger).—"Under certain influences, still unknown," writes Niemeyer,[a] "possibly, merely as a result of the action of a particularly intense contagion, relapsing fever assumes a very malignant character. The appearance of the disease is especially modified by excessive participation of the biliary apparatus, and in most cases death appears with severe symptoms. Griesinger describes this malignant form of relapsing fever from his observations made in the East, and terms it 'Bilious Typhoid.' The St. Petersburg epidemic of 1864 to 1866, where, besides simple recurrent fever, there were numerous cases of bilious typhoid, especially at its commencement, fully confirmed Griesinger's description of the disease, as well as his opinion that it was a severe form of recurrent fever." We have already seen above

[a] *Text-Book of Practical Medicine.* Revised Edition. London: H. K. Lewis. 1880. Vol. II., page 662.

that this view is altogether borne out by the inoculation experiments of Moschutkovsky and others.

According to Zorn,[a] the **fever does not run so high** in this bilious form as it does in the simple variety. It is not uncommon for peripheral parts to feel even cold, a very dangerous symptom. The same writer also states that the second attack or relapse occurs in only about one half of the bilious cases.

Under the heading, "**Yellow Fever of the British Islands,**" Graves[b] detailed a number of cases of the severe form of Relapsing Fever, just now described. Nearly twenty patients died at the Meath Hospital of the disease, prominent symptoms of which were hardness and tenderness of the abdomen about the epigastrium and hypochondria, a knotted feel of the abdominal muscles, general jaundice of a bright yellow colour, uneasiness and anxiety of countenance, a very quick and hurried pulse, coldness of the extremities, and deep purple coloration of the tip of the nose, the cheeks, and sometimes of the toes (local asphyxia). Another notable symptom was abdominal spasm, to which the hospital nurses gave the expressive and (as it proved) appropriate name of "**twisting of the guts,**" for recent intussusceptions or invaginations of the intestines were among the most constant pathological conditions noticed after death.

The **purple coloration of the nose** superadded to the jaundice imparts a weird and frightfully repulsive appearance to the patient sinking under this malignant form of relapsing fever.

In the epidemic of relapsing fever of 1847, Dr. Stokes made a number of observations in the Meath Hospital on the condition of the heart in the disease. He found little evidence of softening of the heart, but "**a bellows murmur,** or, in some cases, **a prolongation of the systolic sound,** was common, especially in the relapse; but this did not result from carditis." Dr. Stokes's observations were borne out by his colleague, Dr. Cathcart Lees. The murmur was systolic in time, basic, and travelled not infrequently into the

[a] *Petersburg Zeitschrift.* IX. 16.

[b] *Clinical Lectures on the Practice of Medicine.* New Syd. Soc. Edition. Vol. I., page 323. 1884.

great vessels. It generally *diminished in intensity when the patient sat up*, and Dr. Stokes regarded it as of anæmic origin. Stokes's researches on the subject have been amply confirmed by R. D. Lyons, of Dublin (in 1861); G. P. Tennent, of Glasgow (in 1871); W. Zuelzer, of Breslau (in 1867); and Obermeier, of Berlin (in 1869).

When **cerebral symptoms** occur in relapsing fever, they are generally, as in typhus, **independent of inflammation** of the brain or its membranes, and depend either on cholæmia, or uræmia, or some other form of blood-poisoning.

Stages and **Duration.**—Four well-defined stages may be recognised in the course of relapsing fever: they are those of—(1) the **primary paroxysm**, (2) the **intermission**, (3) the **relapse**, and (4) **convalescence.**

In 100 consecutive cases under Murchison's care, and accurately noted by him in his case-books, the average duration of the primary paroxysm was 5·96 days; that of the intermission was 7·82 days; that of the relapse was 3·45 days—total duration, 17·23 days. Under ordinary circumstances, when there are but two paroxysms, the duration of relapsing fever to the commencement of permanent convalescence is, according to Murchison, about **18 days.**

Temperature.—The thermometer begins to rise before the initial rigor and while the pulse is still normal. It reaches, or may reach, 104°–106° F. within from twelve to twenty-four hours. An acme, or fastigium, is attained shortly before the crisis (105°–108·7°). During the paroxysm there are usually daily remissions of one or two degrees Fahrenheit—mostly in the morning, and best marked in children. Crisis is sometimes ushered in with a rigor, when the temperature falls to or below normal—a fall of 8°, 9°, 10°, or even 13°, occurring in a few hours. In one case the thermometer fell 14·4° in twelve hours! For two or three days after the crisis the temperature is commonly sub-normal (96°, 94°, or even as low as 92°). So constantly is this the case, that this low temperature is a useful diagnostic sign. In the relapse the maximal temperature of the whole attack is generally recorded, but the hyperpyrexia is of shorter duration than in the primary paroxysm.

The high temperatures in relapsing fever entail little or no danger to the patient, nor do they produce serious cerebral symptoms.

In Plate VI., are two charts of the temperature ranges in relapsing fever—one copied from Wunderlich's "Medical Thermometry," the other from Murchison's "Treatise on the Continued Fevers of Great Britain."

Complications and Sequelæ.—

1. Pneumonia is said to be more common in relapsing fever than in typhus, but Murchison observed this complication in only 4 or 5 out of 600 cases.

2. Sudden collapse may prove rapidly fatal, and has been known to occur at any stage of the disease. The pulse becomes small, irregular, or imperceptible, the heart's impulse fails, and its sounds are blurred or obliterated, the whole surface is cold and livid, and the patient often becomes quite insensible. This fatal syncope is, in some cases, due to hæmorrhage; in others, to pre-existing organic disease.

3. Hæmorrhages from various parts are not uncommon, and may occur at any stage. The most usual kind of hæmorrhage is epistaxis; but bleeding may also take place from the womb, stomach, bowels, kidneys, and ears.

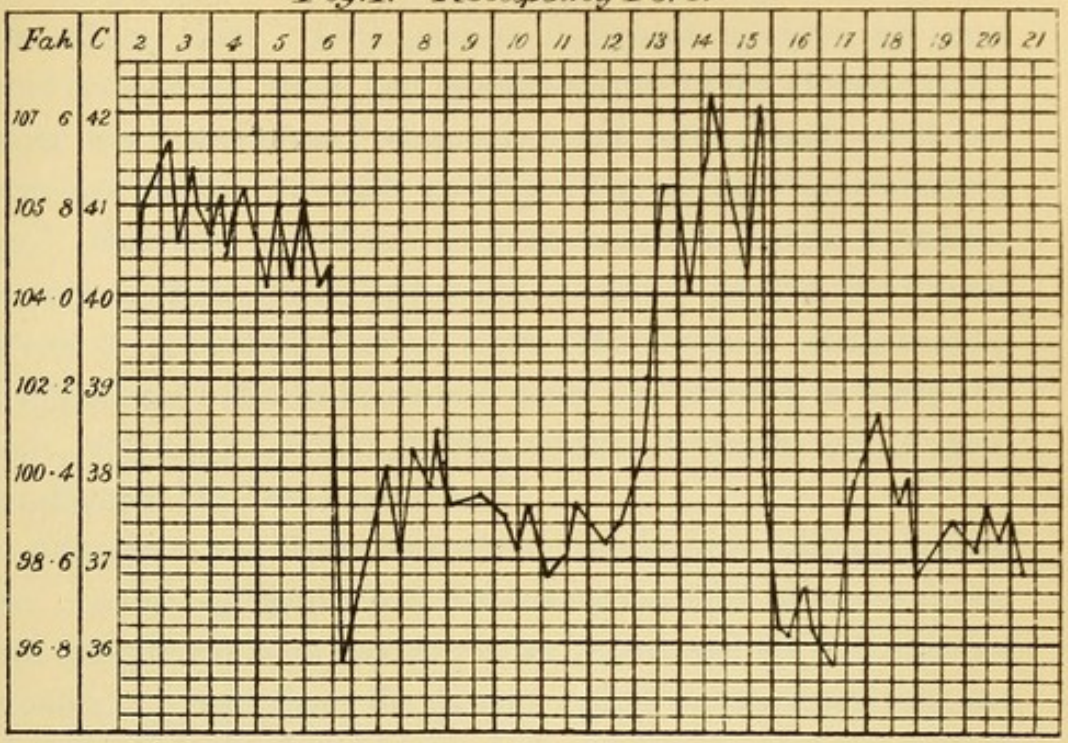
4. The spleen is commonly much enlarged. It may rupture or become the seat of thrombotic abscesses.

5. "One of the most remarkable features of relapsing fever," says Murchison, "is the frequent occurrence during convalescence of a peculiar disease of the eyes. This sequela has been observed in almost all epidemics, but is never met with after typhus or enteric fever." Among other writers on the subject we find Dr. Arthur Jacob, of Dublin, who contributed to the fifth volume (that for 1828) of the "Transactions of the Association of Fellows and Licentiates of the King and Queen's College of Physicians" a paper entitled "On internal Inflammation of the Eye following Typhus Fever." The disease, as described by Dr. Mackenzie, of Glasgow (1843) presents two distinct stages—the amaurotic and the inflammatory. J. A. Estlander, in a paper published in 1869—

Plate VII

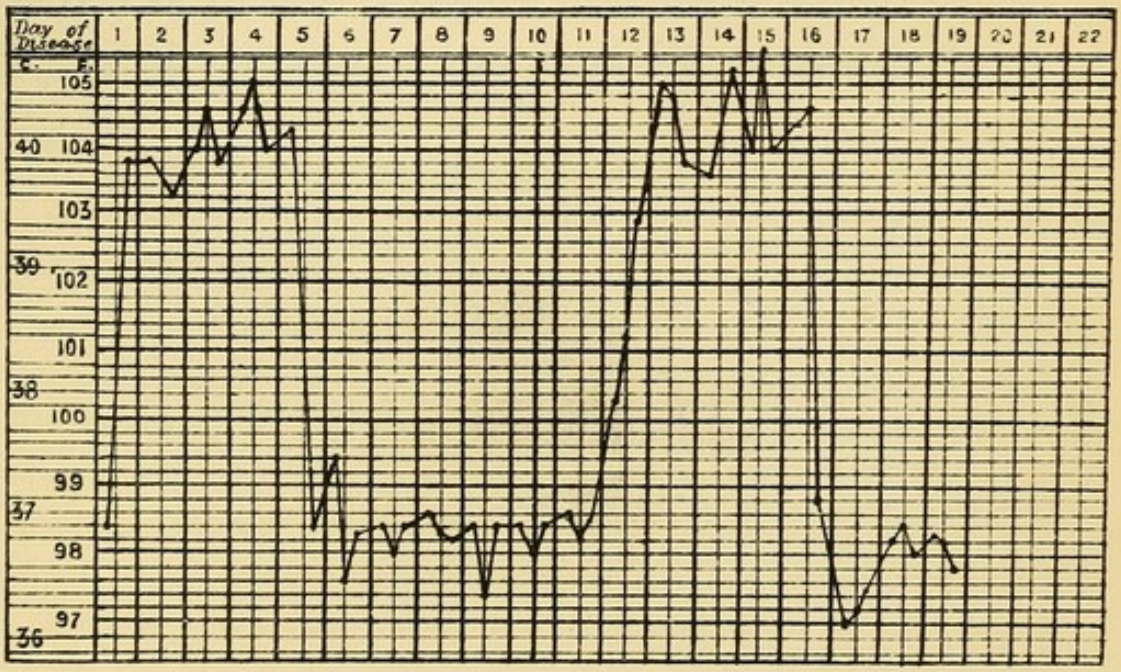
CHARTS OF TEMPERATURE RANGES IN RELAPSING FEVER

Fig. I.—Relapsing Fever



(Copied from Wunderlich's "Temperature in Diseases")

Fig. 2—Temperature in Relapsing Fever, from 1st day of attack Emma D. aged 23. admitted into London Fever Hospital, March 16th 1870



Three months before, this patient had passed through a similar attack of Relapsing Fever in the London Fever Hospital.

(Copied from Murchison's "Continued Fevers of Great Britain")

"Ueber Chorioiditis nach Febris typhosa recurrens"[a]—showed that the starting point of the disease is in the chorioid, and especially in the ciliary body. Inflammation is lighted up here by some morbid state of the blood, and spreads thence to the vitreous body, causing the "amaurotic" symptoms, and subsequently—but not always—to the iris, the iritis corresponding to the "inflammatory stage." Recovery is tedious—in most cases two months have been necessary to effect a cure, and unless carefully treated, the disease may end in permanent loss of sight (Murchison.) Reference on this subject may also be made to a paper by Luchhan, in Virchow's *Archiv.*, volume LXXXII (1880), entitled "Ohren- und Augenerkrankungen bei *Febris recurrens.*"

6. **Diarrhœa** and **Dysentery** often complicate or follow relapsing fever. In some epidemics they are among the chief causes of death. These troubles come on suddenly after rigors, and are accompanied by vomiting. Sometimes the patient suffers much from pain and tenderness over the lower part of the abdomen, or from griping tenesmus.

7. **Peritonitis** is a rare, but very fatal complication. In the epidemic of 1847–48, of 2,846 cases of relapsing fever in the Glasgow Infirmary, 7 died from peritonitis. It is usually secondary, depending on dysentery, rupture of the spleen, or splenic abscess.

[a] *Archiv. für Ophthalmologie.* 1869. Band, XV. Abtheilung II., page 108.

CHAPTER XXXIV.

The Diagnosis, Prognosis, Pathology and Treatment of Relapsing Fever.

Diagnosis from: Typhus, enteric fever, simple continued fever, remittent fever ("jungle fever"), yellow fever.—Presence of the spirochæte in the blood is pathognomonic.—Prognosis and Mortality: Unfavourable symptoms. Pathological Anatomy.—Treatment: no specific for Relapsing Fever. Hygienic and Expectant Treatment.—Nitre.—Treatment of symptoms and complications.—"Bilious Typhoid" (Griesinger.)—Convalescence.

Diagnosis.—Relapsing Fever may be confounded with:—

1. **Typhus.**—In forming an opinion, regard should be paid to the nature of other cases occurring in the same house or family, the presence or absence of eruption, the presence of epistaxis, jaundice, vomiting, and cardiac murmur, and the whole clinical history, which is quite different in the two fevers.

2. **Typhoid** or **Enteric Fever.**—The differential diagnosis again turns on the clinical history, the occurrence of epidemics of relapsing fever in times of famine.

3. **Simple Continued Fever** in its acute form. But in simple fever no relapse occurs.

4. **Remittent Fever.**—This, however arises from malaria, affects all classes of the community alike, and is not contagious. Besides, there is no resemblance between the intermissions of relapsing fever and the remissions of remittent fever. Remittent fever, also, is a tropical disease.

5. **Yellow Fever,** because jaundice may be present in relapsing fever, particularly in that form now known as "bilious typhoid" (Griesinger.) The points of diagnosis are: yellow fever attacks all classes alike, but the same individual once only in his lifetime. It is a most mortal disease. Jaundice is nearly always a prominent symptom in yellow fever, which does not cause great swelling of the spleen. Death in yellow fever is usually preceded

by "**black vomit**," which is rare in relapsing fever. The diseases differ completely in their clinical course, and, lastly, yellow fever is checked, almost annihilated, by air-temperatures below 70° F.

Dr. William Cayley[a] very properly points out that the **demonstration of the spirochæte in the blood** will now distinguish relapsing fever from all other febrile diseases.

Prognosis and Mortality.—Out of 2,115 cases of relapsing fever admitted to the London Fever Hospital from 1848 to 1870, inclusive, only 39 proved fatal—that is a death-rate of 1·84 per cent., or about 1 in 54. From statistics of 18,859 cases with 761 deaths, Murchison estimates the **death-rate** of relapsing fever in the United Kingdom at **4·03 per cent.**, or 1 in 24·78. In the epidemic of 1877–79 in Bombay, however, Vandyke Carter observed a mortality of 18·02 per cent.

The **rate of mortality** increases with advancing age, but not at all to the same extent as in typhus. Almost all published statistics agree in assigning a higher death-toll to the male sex. The mortality also is greatest in winter. Lastly, the rate of mortality is higher at the beginning and height of an epidemic than at its close. In mixed epidemics of typhus and relapsing fever, an apparent exception to this rule is observed, owing to the increasing ratio which the typhus cases, with their higher death-rate, bear to relapsing fever in the later stages of the outbreak.

The **unfavourable symptoms** in relapsing fever are: jaundice with cerebral symptoms, purpura-spots and vibices, copious hæmorrhages, suppression of urine with head symptoms, ataxic symptoms (the "typhoid state"). Again, the prognosis is, of course, unfavourably influenced by the presence of a complication or of a chronic organic disease, particularly fatty degeneration of the heart.

Pathological Anatomy—The *post-mortem* appearances of relapsing fever may, according to Murchison, be summed up as follows:—

1. There is **no specific** or constant **lesion.**
2. The most common lesions are enlargement and infarction of

[a] Murchison's *Treatise on the Continued Fevers.* Third Edition. 1884. Page 394.

the spleen, slight leukæmia, congestion of the liver and kidneys, jaundice, dysentery, and pneumonia.

3. In most cases nothing can be discovered in the liver, or in the bile-ducts, to account for the jaundice. In exceptional cases only is there acute atrophy or catarrh of the ducts. The icterus, or jaundice has, however, been characterised by Bock and Wyss as of hepatic origin (hepatogenous), because of the presence of the bile-acids in the urine.[a]

4. No lesion can be discovered in the brain or its membranes, even when cerebral symptoms have been most marked.

Treatment.—"The chief promoting causes of the disease being famine and contagion," writes Dr. T. W. Grimshaw,[b] "the **means for prophylaxis are obvious.**" "Relapsing fever," says Murchison, "is the appanage of poverty and destitution; and the more completely we succeed in ameliorating the condition of the poor, particularly in times of famine, the more successful shall we be in averting the disease."

The details of Preventive Treatment already specified in Chapter IV., as well as those given under the head of typhus, apply perfectly well to relapsing fever.

No specific for this disease has yet been discovered. Quinine, digitalis, and the salicylates have all been tried and found wanting.

Riess[c] considered that he obtained satisfactory results from salicylate of sodium in a single dose of rather more than 90 grains ("in Einzelgaben von 6·0 Grm."), but Ewald was not able to confirm his results with even larger doses.

As there is no specific, and as the malady is not very dangerous to life, our treatment of it should be **hygienic** and **expectant** to a large extent. Lebert puts this very well when he says[d]:—"Rest in bed, fresh air, cleanliness, fever diet, milk, soups, meat broths, cooling drinks, are the principal things to be attended to. If the

[a] *Studien über Febris recurrens.* Berlin. 1869.

[b] *Quain's Dictionary of Medicine.* 1883. Art. "Relapsing Fever."

[c] *Typhus recurrens in Berlin.* Berl. klin. Wochenschr. 1868. No. 22. 1869. No. 31.

[d] Von Ziemssen's *Cyclopædia of the Practice of Medicine.* Vol. I. Art. "Relapsing Fever." 1875.

patient has an appetite, if he can take nourishment, he should have not only more but better fare—vegetables, stronger soups, meat, and particularly wine. Clear pure water and carbonic acid water are the beverages best borne."

Murchison gives advice to the same effect when he says:—

"1. That a larger quantity of nourishment is usually required after the cessation of the febrile paroxysms than in typhus, and that many patients during the fever, and especially in the relapse, will take a considerable quantity of nutriment with relish, and apparently with benefit.

"2. That alcoholic and other stimulants will often be required about the time of crisis, to counteract the tendency to collapse. They are especially indicated in persons over 45 years of age, and where there is evidence of a weak heart."

The same author recommends that the treatment should be begun in the early stage with an emetic of ipecacuanha and antimony, or of mustard. Constipation should be counteracted by small doses of castor oil (a teaspoonful, with one of glycerine, in milk). Murchison and many writers, from Baglivi downwards, speak highly of the value of **nitre** in relapsing fever. It acts as a mild aperient, an efficient diuretic, and a preventive of uræmia, and (consequently) of cerebral symptoms also. The formula for the administration of the salt runs thus:—

℞. Potassi Nitratis, gr. 60–120;
Acid. Nitric. dilut., ʒj.;
Tincturæ Digitalis, ʒss.;
Aquæ, ad ℥vi.

M. ft. mist. *Signa:* "One sixth part every fourth hour."

To fulfil the same indications as the foregoing, Dr. Cheyne's "mild anti-febrile and gently stimulant diaphoretic" (as Graves[a] calls it) may be prescribed:—

℞. Ammonii Carbonatis, gr. 60;
Succi Limonis, ʒii.;
Syrupi Aurantii, ℥ss.;
Aquæ, ℥iiiss.

M. ft. mist. *Signa:* "One-sixth part every third or fourth hour."

[a] *Clinical Lectures.* New Syd. Soc. 1884. Vol. I., page 318.

Graves improved Cheyne's prescription by substituting carbonate of sodium for carbonate of ammonium—thus, in a modern form :—

℞. Sodii Carbonatis, gr. 60;
Succi Limonis, ℥i., ℥vi.;
Misce et adde—
Tinct. Aurantii Recentis, ℥ii.;
Syrupi Aurantii, ℥ss.;
Aquæ, ad ℥vi.

M. ft. mist. *Signa :* "One sixth part every third or fourth hour."

Graves says nothing can be more agreeable in flavour than this mixture. The citrate of sodium which is formed "determines gently to the kidneys, tends to keep up a soluble state of the bowels, and forms a most grateful and refreshing beverage."

If a weaker solution is preferred, a drachm of the carbonate of sodium may be dissolved in five, instead of four, ounces of water.

As a *placebo*, Lebert gives small doses of **phosphoric acid.** This formula would, probably, meet his approbation :—

℞. Acidi phosphorici diluti, ℥iss.;
Elixir. Glusidi (Saccharini), ℥ss.;
Aquæ, ad ℥iv.

M. ft. mist. *Signa :* "A tablespoonful every second or third hour."

Bleeding (*venesection*) is **inadmissible** in relapsing fever for **reducing temperature** or any other purpose. Niemeyer sets his face even against cold baths, on the ground that relapsing fever, in spite of the high temperature, has a low mortality. He thinks it well to limit ourselves to **sponging the body** with cold lotions, and—if the **cerebral symptoms** are severe—to the **application of ice to the head.** Lebert is quite in accord with Niemeyer on this point and recommends the very same measures. Should they fail to relieve headache and procure sleep, Murchison says recourse must be had to opium or hydrate of chloral.

When **collapse** threatens, Lebert orders an ammoniated tincture of musk as follows :—

℞. Moschi, gr. 60;
Ammonii Carbonat., gr. 30;
Olei Menthæ, min. v.;
Alcoholis, ʒii.;
Aquæ Destillatæ, ʒv.

Of this mixture, thirty drops are to be taken in a tablespoonful of sugar and water, or wine, every hour.

Obstinate diarrhœa is to be combated by alum, tannin, nitrate of silver, and opium (Lebert); or by small doses of laudanum in decoction of logwood, or an astringent mixture containing kino or catechu with opium (Murchison).

For **dysentery** the best remedies are **ipecacuanha** and **opium**. They may be prescribed in the form of "Twining's Pill"[a] (ipecacuanha, blue pill, and extract of gentian), or as follows (Murchison):—

℞. Pulv. Ipecac., gr. 2;
Pulv. Ipecac. Comp., gr. 5;
Hydrargyri cum Cretâ, gr. 3.

Misce, Fiat Fulvis. *Signa:* "One four times a day."

An enema of starch and opium should be used from time to time, if there is tenesmus; and occasional doses of castor-oil are useful if the stools are scanty and the abdomen is distended.

Should dysentery not yield to these means, the pharmacopœial solution of the **pernitrate of iron** with small doses of opium will be found an excellent remedy.

Severe epistaxis should be controlled by plugging the nostrils and posterior nares.

For the **post-febrile ophthalmia**, in its earlier stages, a liberal diet, tonics (such as quinine and iron), and blisters behind the ears, will do good. When **iritis** declares itself, Mackenzie[b] recommends leeching the temples, continued dilatation of the pupils with solution of belladonna or atropin, blisters behind the ears, a nutri-

[a] Cf. Twining. *Diseases of Bengal.* 1832.
[b] *Loc. cit.* 1843.

tious diet, and one of the following powders every four or six hours until the gums are touched :—

℞. Hydrargyri Subchloridi, gr. 12 ;
Quininæ Sulphatis, gr. 12–24 ;
Pulv. Opii, gr. 3 ;
Sacchari Albi, q.s. ut fiant pulv. xij.

In "**bilious typhoid**," Griesinger recommends large doses of **quinine** (10 to 30 grains daily)—a mild purgative of salts, castor-oil, or cream of tartar (acid tartrate of potassium) having been first given at the beginning of the attack.

During the **convalescence** of patients who have become exceedingly exhausted, Lebert considers that a good invigorating fare is best supplemented by **tincture of cinchona**, or mild preparations of **iron**, particularly of the **lactate**. This last may be given to the amount of three to eight grains daily, in pill form, with extract of gentian.

CHAPTER XXXV.

Enteric, or Typhoid, Fever.

General Considerations.—Essential difference between typhus and enteric fevers not recognised in the past—reasons for this.—Evils of not differentiating between typhus and enteric fevers.—Fundamental distinctions.—Liebermeister's views.

The day has now long passed by since the doctrine of the identity of typhus and enteric fever attracted any number of adherents among the great thinkers of the Medical Profession. But there was a time when this doctrine did obtain credence, and even the commanding genius of William Stokes clung to this faith with what in one less distinguished would, perhaps, be regarded as obstinate infatuation. Writing so recently as 1874,[a] he used such expressions as these: "The inexpediency of drawing hard and fast lines of distinction between *what are termed* typhus fever and typhoid fever."[b] "Fever has been *somewhat arbitrarily* divided into two classes, or placed under two great headings—typhus and typhoid."[c] "Do not these facts point to the conclusion that *there is but a slight tension*, so to speak, *in the individuality or separate characters of the various forms of fever*, and that in their essence and from a practical point of view, they may be looked on *as species rather than genera*—the genus being fever, that condition on which anatomical investigations, in the words of Graves, throw but a negative light?"[d]

I think there are three chief reasons why in the past the essential differences between typhus and enteric fever were not recognised.

[a] Lectures on Fever. London: Longmans, Green & Co. 1874.
[b] *Loc. cit.* Page 44.
[c] *Loc. cit.* Page 78.
[d] *Loc. cit.* Page 89.

First, in presence of the defective hygiene of former days the "typhoid state," of which mention has so often been made in these pages, was much more commonly observed than it is now. It *complicated* enteric fever no less than typhus.

Secondly, there can be no reasonable doubt that enteric fever was of much less frequent occurrence before the days of water closets and of water carriage of "night soil." Murchison especially alludes to the fact that enteric fever has much increased in Edinburgh of late years,[a] and his explanation is that this increase of the fever, "followed the introduction of new sanitary arrangements—the substitution for the scavenger and nightmen of drains opening into the interior of the houses, but with a water supply insufficient to prevent the escape of sewer emanations."[b]

Thirdly, from the non-recognition of the separate identity of typhus and typhoid, it followed almost as a matter of course that cases of both these fevers were treated in the same wards—nay, in adjoining beds, perhaps, sometimes in the same bed. The natural consequence was that the typhoid patients contracted typhus, which soon declared itself with its stupor and maculæ—thus rendering confusion worse confounded."

The evils which accrue from non-recognition of the essential difference of typhus and typhoid as regards their origin are clearly shown by Mr. J. Spear, in his report to the Local Government Board on appearances of typhus fever in various parts of England during the year 1886–87.[c] In Leeds, Hartlepool, Carlisle, Middlesborough, Oldham, Newcastle-on-Tyne, and Flint, typhus gained a footing through the neglect of unrecognised cases; and in Liverpool this is the common experience with respect to constantly recurring localised outbreaks. In a previous year (1885), Mr. Spear met with an outbreak in Workington. The disease spread by a demonstrable chain of cases to several adjoining villages and

[a] Treatise on the Continued Fevers. Third Edition. 1884. Pp. 443 and 444.

[b] Cf. W. T. Gairdner, M.D. *Public Health in relation to Air and Water*, Edinburgh. 1862.

[c] *Sixteenth Annual Report of the Local Government Board.* 1886–87. Page 269.

towns, localised epidemics of considerable importance resulting. In each one of these several invasions the disease had been regarded as *typhoid* or enteric fever, with the result that inefficient measures for preventing its spread had been applied. In 1883, Mr. Spear investigated a notable outbreak of typhus in a large charitable institution at Hammersmith. The disease had been spreading for three months amongst the children, infecting at least nineteen of them, as well as several of the Sisters of Mercy and two priests from outside, before its true nature was recognised. The priests were supposed to be suffering from "typhoid."

From his experiences Mr. Spear was led to conclude that considerable outbreaks of typhus might occur without medical recognition, the disease being mistaken for typhoid fever or (in children) for measles, influenza, &c., and that the result of such diagnostic failure was not merely official mis-information, but often the unchecked spread of the disease. He observes—"It is a matter the extreme importance of which to the general community has not yet been sufficiently appreciated, or *practical instruction in the diagnosis of fever would occupy a more prominent place in medical education.*"[a]

Liebermeister[b] puts the matter very well when he says, "In fact, the only likeness between typhus and typhoid fever, except that they are both infectious diseases, is that in both diseases many of the cases are attended with severe and long-continued fever. In every other respect they are different. Typhus fever has many more analogies with smallpox, measles, and scarlet fever, than with typhoid fever. Typhoid fever, in its ætiology and mode of propagation, resembles dysentery and cholera much more than it does typhus.

"The real fundamental difference between the two diseases is this: Typhus fever is a purely contagious disease; typhoid fever belongs to the miasmatic-contagious diseases. Typhus can be transmitted directly from person to person; its contagion is as

[a] *Loc. cit.* Page 287.

[b] Von Ziemssen's *Cyclopædia of the Practice of Medicine.* Vol. I., page 41. Art. "Typhoid Fever."

intense and evident as is that of the acute exanthemata. Typhoid fever, on the contrary, is never [directly] transmitted from person to person. There can be no doubt that this profound difference in the mode of propagation depends upon an essential difference in the poison producing the two diseases. Every classification, therefore, which is grounded on scientific principles, must separate these two diseases widely from each other."

As we shall see in the sequel, Liebermeister is too dogmatic in absolutely denying the transference of typhoid or enteric fever from person to person, but there can be no doubt that the disease belongs, as he supposed, to the miasmatic-contagious rather than to the contagious group. That is, an affected body furnishes, indeed, the morbific germs of typhoid fever, but these germs must undergo a further development after they leave that body, most probably in immediate relation to sewer gas or the products of decomposition (putrefaction), before they attain their complete infecting power and become capable of reproducing the disease in another body to which they have gained access by one or other of the recognised channels of infection.

CHAPTER XXXVI.

Enteric Fever (*continued*).

Nomenclature.—Synonyms.—Definition.—Literature and History.—Geographical Distribution.—Ætiology: Predisposing causes: sex, age, defective sewerage and drainage, season, temperature and moisture, soil and underground water.—Immunity.—Exciting cause.—Bacteriology: *Bacillus typhosus* (Eberth).—Supposed spontaneous origin of enteric fever.—This doctrine is now untenable.—Resting spores.—Paths of Infection: currents of air, drinking water, milk, meat.—Mode of invasion.

Nomenclature.—Probably there is no other disease which has so many, and at the same time such unsuitable, names. Objections can be raised on etymological, pathological, and ætiological grounds respectively to each of the three names by which this fever is best known to English authorities. It is *not like typhus*, and therefore the term "**Typhoid**" in its literal sense is inapplicable to it. *The fever is not* in any sense *the result of the characteristic intestinal lesions* of the disease, and so the term "**Enteric**" is unsuitable from a pathological point of view. Lastly, mere *putrefaction is* certainly *not the proximate cause* of the fever, and so Murchison's ingenious name of "**Pythogenic Fever,**" derived from what he endeavoured to show was the cause of the fever (πυθογενὴς, from πύθων-πύθομαι, *putresco*—and γεννάω, *to beget, to engender, to produce*) is ætiologically incorrect. Putrefaction does not engender typhoid or enteric fever, however much its presence may contribute to the potency of the specific poison of the disease.

Having regard, however, to their general acceptance in English-speaking communities, and waiving their strict propriety from a pathological and an etymological standpoint respectively, the terms "enteric fever" and "typhoid fever" are probably the best names for the disease we can adopt.

Since 1871 the term "Enteric Fever" has been used officially in the Reports of the Army Medical Department.

It is also adopted in the Reports of the Registrars-General for England, Scotland, and Ireland; and under it such classical writers as Murchison and Hilton Fagge have described the disease. To it, therefore, I will give the preference in the succeeding account of this form of Continued Fever.

The name "typhoid" we owe to Louis, who in 1829 wrote of the fever as "Fièvre typhoïde." It was adopted by Chomel in 1834, and by Alex. Stewart in 1840.

I select the following additional names for this disease, as they refer to its characteristics, its symptoms, or its course:—

From its **supposed resemblance to typhus** it was called Typhus Nervosus (de Sauvages, 1760); Typhus Mitior and Synochus (Cullen, 1760); Typhus gangliaris vel entericus (Ebel, 1836, Schönlein, 1839).

From its **seasonal prevalence** it is known in America as Autumnal or Fall Fever (Austin Flint, 1852).

From its **remittent character in children** it was called Infantile Remittent Fever (Evans and Maunsell, 1836).

From its **long duration** it was described as Febris lenta (Forestus, 1591); Slow, or Lent, Fever (Strother, 1729).

From its **supposed nervous** or **hysteric character** it was called Low Fever, Nervous Fever (Gilchrist, 1734); Slow Nervous Fever (Huxham, 1739).

From its **septic character** it was called Febris putrida (Riverius, 1623); Fièvre ataxique (Pinel, 1798); Entérite septicémique (Piorry, 1841).

From its **gastric and intestinal symptoms and lesions** it became known as Febris gastrica (Bellonius, 1640); gastrisches Fieber (Richter, 1813); Fièvre gastrique (*Dict. des Sciences Médicales*, 1816); Epidemic Gastric Fever (Cheyne, 1833); Gastric Fever (Craigie, 1837); Bilious Fever (Sir John Pringle, 1750, and Rutty, 1770); Schleimfieber (Kanz, 1795); Fièvre muqueuse; Mucous or Pituitous Fever (Copland, 1844); Gastro-entérite (Broussais, 1816); Dothiénentérite (Bretonneau, 1826); Entérite folliculeuse (Cruveilhier, 1835); Enteric Fever (Ritchie, 1846); Intestinal Fever (W. Budd, 1856); Enterica (*sc.* Febris) (Samuel Wilks).

The German names under this heading are Typhus-abdominalis (Autenrieth, 1822); Darm-typhus (same authority): Ileo-typhus (Griesinger, 1857).

From its **supposed mode of origin** it was called Nightsoil Fever (Brown, 1855); Cesspool Fever; and, as before mentioned, Pythogenic Fever (Murchison, 1858). Also "Worm Fever" is a trivial term for the disease.

The **synonyms** for Typhoid Fever in common use in the chief European tongues are the following:—*Germ.* Abdominal-typhus, Darm-typhus; *French*, Fièvre typhoïde, Dothiénénterie; *Ital.* Febbre tifoide, Tifo enterico, Tifo intestinale, Tifo addominale, Ileotifo; *Span.*, Fiebre tifoidea, Dotinenteria; *Dutch*, Slependekoorts (*i.e.*, slow fever), Darmkoorts, Zenuwzinkingkoorts[a] (Becking); *Danish* and *Norwegian*, Tyfoidfeber; *Swedish*, Tyfoidfeber.

Definition.—An acute, specific, infective fever prevailing usually as an endemic rather than an epidemic disease. The virus of this fever develops in the dejections of the sick and in presence of the decomposition of certain organic substances. The most important and prominent symptoms are: a gradual onset, which is often insidious or marked by slight chills, a feeling of lassitude, or diarrhœa and a gradually rising temperature, with an "easel-like" range on the chart, culminating in a high fever. The fever, at its fastigium or acme runs a continuous, in the beginning and closing stages, a remittent, course. Prostration of the nervous system and headache; later on, but only in the severer cases, dulling of the intellect and often delirium, of a low muttering kind; dilatation of the pupils; a tendency to heart failure. Continuous diarrhœa, frequently intestinal hæmorrhages, enlargement of the spleen, meteorism, tenderness and gurgling (gargouillement) in the ileo-cæcal region. An inconstant roseolar rash appears from the end of the first week in successive crops, but never becomes petechial. A tendency to relapse is often observed. The anatomical characters are: an inflammatory tumefaction, ulceration, and sloughing of the glands of the small intestine, and less frequently of those of the large intestine also—the solitary and agminate glands (Peyer's patches) of

[a] *I.e., Nervous catarrhal fever.*

the ileum being especially affected—as well as tumefaction of the mesenteric glands and spleen, the presence of specific bacilli in these organs—the *Bacilli typhosi* of Eberth (1880). In certain rare cases the intestinal lesions may recede or fail to appear. The duration of the illness amounts on an average to 3 or 4 weeks—according to Murchison to 24·3 days. (After Zuelzer[a] and W. Cayley[b]).

Literature and History.—Hippocrates[c] is supposed to have referred to Enteric Fever under the name πυρετός ἡμιτριταῖος. ("Semitertian Fever"—a form of ague described by Galen).

In Germany, von Hildenbrand,[d] in 1810, first clearly distinguished between typhus and enteric fevers. Early in the present century Broussais[e] and others in France maintained that a "gastro-entérite" was the essential cause of "fever." They were led to the false theory of the *symptomatic* nature of fever by the discovery that the intestines of those who died in Paris of what we now call enteric fever were inflamed and ulcerated.

Serres and Petit[f] considered that the intestinal lesions were specific, depending on the introduction of a poison into the system, and of an eruptive nature like the pustules of smallpox.

In 1818 Bretonneau of Tours undertook certain anatomical researches, the result of which led him to propose for this disease the name of **dothiénentérie**, or **dothiénentérite**, to indicate the special nature of the intestinal lesion which accompanies it—from the Greek δοθιην, *a small abscess* or *boil* (Lat. *furunculus*) and ἔντερον, *the intestine*. The second form of the word contains the French equivalent "*-ite*" of the terminal *-itis*, signifying inflammation. This cumbrous term was adopted by Bretonneau's illustrious pupil, Trousseau,[g] and under it the disease is described in the *Clinique médicale de l'Hôtel Dieu de Paris*.

[a] *Real-Encyclopädie der Gesammten Heilkunde.* Wien und Leipzig: Urban und Schwarzenberg. 1885. Erster Band. Art. "Abdominal Typhus."

[b] Fowler's *Dictionary of Practical Medicine.* London: J. & A. Churchill. 1890. Art. "Typhoid Fever."

[c] Editio Foesii. 1624. Page 961.

[d] *Loc. cit.* Wien. 1810.

[e] *Examen des Systèmes modernes de Nosologie.* Paris. 1816.

[f] *Traité de la Fièvre Entéro mésentérique.* Paris. 1813.

[g] "*De la maladie à laquelle M. Bretonneau a donné le nom de dothiénentérie, ou de dothiénentérite.*" *Archives Gén. de Méd.* Série I., tom. x., 169. 1826.

In 1829, P. C. A. Louis published in Paris, in two volumes, his "Recherches sur la maladie connue sur les noms de gastro-entérite, fièvre putride, adynamique, &c." and in that work he first proposed the name of "Fièvre typhoïde" for the disease.

It was not until the year 1836 that accumulating clinical and pathological evidence led to the conclusion that two distinct diseases—typhus and enteric fever—were being confounded with each other. The credit of establishing the differential diagnosis of the two fevers is divided by Murchison among a number of observers—R. Perry,[a] of Glasgow (1836); H. C. Lombard,[b] of Geneva (1836); Gerhard and Pennock,[c] of Philadelphia, U.S.A. (1836); Shattuck,[d] of Boston (1838); H. C. Barlow,[e] of London (1840); and A. P. Stewart,[f] of London (1840).

In a series of admirable papers published between 1849 and 1853, Sir William Jenner maintained that typhus and the so-called "typhoid fever" were as distinct as any two of the exanthemata. His writings did much to set the vexed question at rest, although many years elapsed before the specific distinctness of the two fevers was finally and universally recognised. Barrallier, of Toulon, in 1861, put the question in an almost epigrammatic form as follows: "Elles sont séparées l'une de l'autre par leurs causes, leurs symptômes, leur marche, leur durée, leurs caractères anatomiques; elles appartiennent réellement à la même classe de maladies, les fièvres essentielles spécifiques, mais elles constituent des genres à part, comme la rougeole et la scarlatine dans le groupe des fièvres éruptives."

The principal modern writers on the fascinating subject of enteric fever are: Charles Murchison,[g] W. Cayley,[h] and C. Hilton

[a] *Edin. Med. and Surg. Journ.* Vol. XLV. 1836.
[b] *Dubl. Journ of Med. Science.* Vol. X. 1836.
[c] *American Journ. of Med. Sci.* Vols. XIX. and XX. 1836. February and August.
[d] *American Med. Examiner,* Feb. and March. 1840.
[e] *Lancet.* February 20, 1840.
[f] *Edin. Med. and Surg. Journ.* Oct., 1840.
[g] *Treatise on the Continued Fevers of Great Britain.* Third Edition. 1884.
[h] *Croonian Lectures.* 1880,

Fagge,[a] in our own country; August Hirsch,[b] Professor of Medicine in the University of Berlin; Karl Liebermeister,[c] Professor of Clinical Medicine in Tübingen; and Wilhelm Zuelzer,[d] of the University of Berlin, in Germany; and Charles Smart,[e] Major and Surgeon, U. S. Army, in America. In the work of the last-named author, the section on Pathological Anatomy is profusely illustrated with a series of most beautiful photo-reliefs, heliotypes, photographs on steel, and chromo-lithographs. In these the pathological appearances of the intestines in typhoid or enteric fever are shown in perfection. I venture to say that such another collection of drawings does not exist.

Geographical Distribution.—Enteric fever **prevails all over the world.** The sketch of its geographical distribution given by Hirsch justifies the designation of **an ubiquitous disease.** Its incidence is as wide as the globe. Until recently, the **Tropics** were supposed to enjoy an immunity from it, but this is not so, although the disease is, no doubt, less prevalent in lower latitudes than in higher. This fever is met with in India—often obviously lying hidden behind the "continued and remittent fever of the Anglo-Indian physicians—in Burma, Cochin China, Réunion, Mauritius, Madagascar, Tahiti, Senegambia, Bermudas, the West Indies, Cayenne, and Brazil—all tropical or sub-tropical localities. It would seem, then, that climate does not, *in and by itself*, exert a determining influence on the occurrence of enteric fever (Hirsch).

This disease is endemic in the British Islands. It is apparently most common in England, more common in Ireland than in Scotland, and in the last-named country more common on the west than on the east coast (Murchison). It is also endemic in nearly all parts of the Continent. In Iceland, where it is known by the name of "Landfarsot" (*sickness of the country*), it is met with every year—

[a] *The Principles and Practice of Medicine.* Art. "Enteric Fever." 1886 and 1891.

[b] *Handbook of Geographical and Historical Pathology.* New Syd. Soc. Translation. 1883–1886.

[c] Von Ziemssen's *Cyclopædia of the Practice of Medicine.* Vol I. 1875.

[d] *Real-Encyclopädie der gesammen Heilkunde.* Art. "Abdominal Typhus."

[e] *The Medical and Surgical History of the War of the Rebellion.* Part. III. Volume I. Medical History. Washington, 1888.

not infrequently in an epidemic and malignant form. In Africa it is not wanting. It prevailed extensively among the British troops in the Zulu, Egyptian, and Soudan campaigns, and among the French troops in Tunis. Brigade-Surgeon Albert A. Gore, M.D., contributed to the *Dublin Journal of Medical Science* a series of papers on enteric fever in Egypt since the British occupation of that country.

In North America the disease is endemic from Greenland to Mexico. It has been observed throughout Central and South America also, as well as in most parts of Australasia.

Ætiology.—General considerations lead us—for the most part with Murchison—to the following conclusions:—

1. Enteric fever is either an **endemic** disease, or its epidemics are circumscribed (and local).

2. It is most **prevalent in autumn** and after warm weather.

3. It is **independent of overcrowding**, and attacks rich and poor indiscriminately.

4. It sometimes **arises apparently independently** of a previous case, in the presence of fermentation of fæcal and perhaps other forms of organic matter, in which presumably the "resting spores" of its *contagium vivum* have lurked.

5. It **may be communicated** by the sick to persons in health, but even then the poison is not like smallpox, given off from the body in a virulent form, but is developed by the decomposition of the excreta after their discharge.

Dr. Cayley expresses the opinion—based upon observations made at the Middlesex Hospital—that the period required for the development of the infectious properties of the stools in this fever does not exceed twelve hours, and may be shorter. He holds that the fresh stools are incapable of communicating the disease, a point which may well be regarded as settled, notwithstanding the opinion to the contrary put forward, in 1880, by Dr. Collie, of the Homerton Fever Hospital.

6. Consequently, **an outbreak** of enteric fever **implies poisoning** of air, drinking water, milk, or other ingesta, with decomposing excrement.

I have ventured to change the wording of the fourth of these propositions, because I cannot assent to the doctrine of the *de novo* or spontaneous origin of enteric fever which is implied in Murchison's original words—viz., "It may be generated independently of a previous case by fermentation of fæcal, and, perhaps, other forms of organic matter." [a]

While we admit the propriety of using the term "endemic" in connection with enteric fever, we agree with Dr. Hilton Fagge that "there could be no greater mistake than to suppose that its diffusion is, like that of ague, independent of the movements of human beings and of their intercourse." The analogy is rather between enteric fever and cholera.

Of late years enteric fever has shown a greater tendency to an epidemic prevalence than was formerly the case. This is notably so in Dublin, where an epidemic of this fever has prevailed every autumn since 1887. The Registrar-General for Ireland, Dr. Grimshaw, has furnished me with accurate statistical facts bearing upon the prevalence of both typhus and enteric fever in Dublin during the past twelve years. From Table II., in which this information is embodied, the rapid decline of typhus and the still more rapid increase of enteric fever in the Irish metropolis are clearly apparent. The Table includes the number of cases of typhus and of enteric fever admitted to hospital each quarter and of deaths from these diseases in the Dublin Registration District during the twelve years, 1879–1890, inclusive.

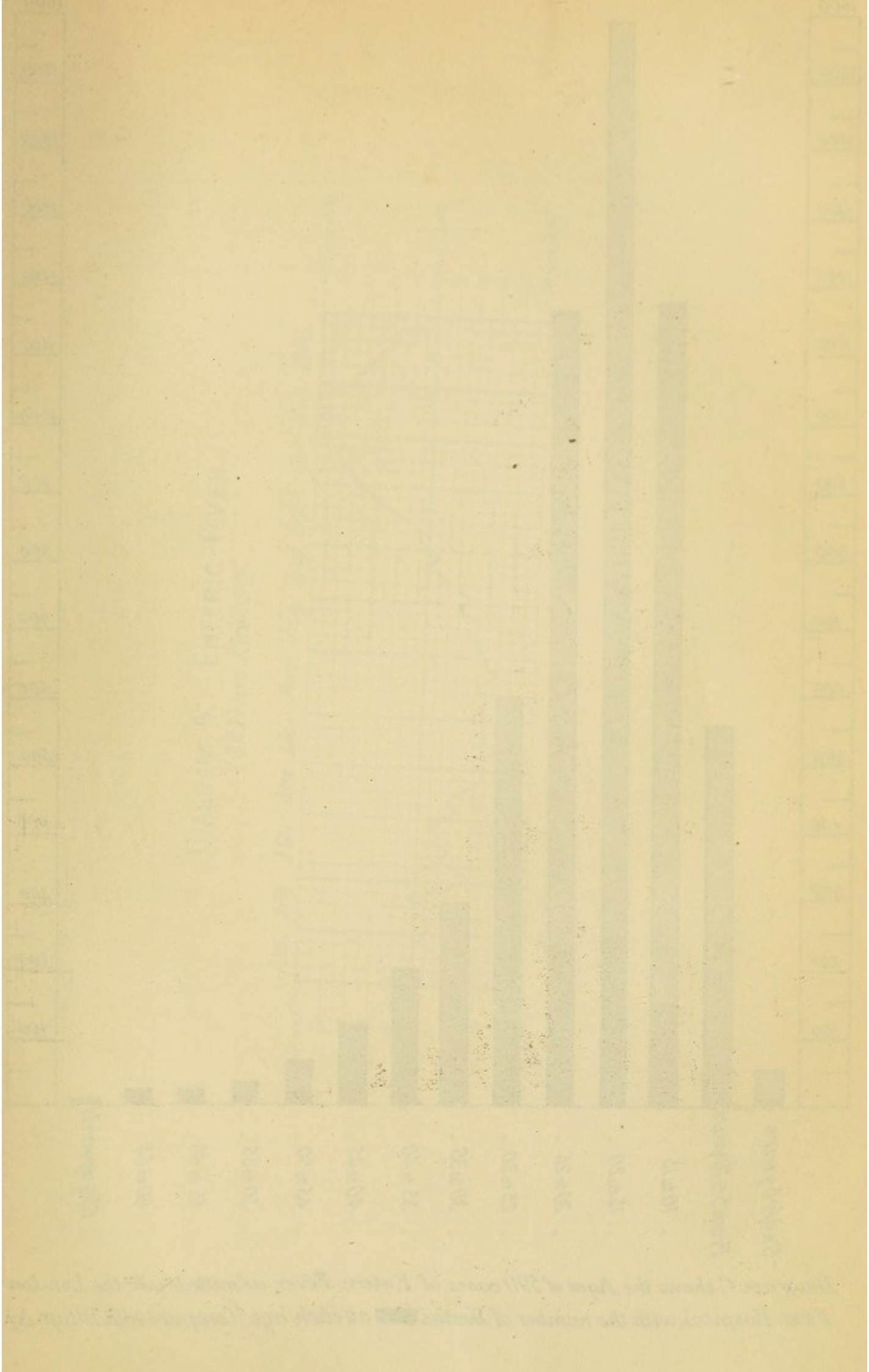
[a] Murchison. *Loc cit.* 1884. Page 499.

TABLE II.—*Showing for each Quarter in the 12 years, 1879–90, the number of cases of Typhus and of Enteric Fever admitted into the principal Dublin Hospitals; the number of Deaths in these Institutions from each of the diseases named, and the number of Deaths from those causes registered in the whole of the Dublin Registration District.*

Years	Quarters	Typhus			Enteric Fever		
		No. of Admissions to Hospitals	Deaths in Hospitals	Total Deaths Registered in Dublin Registration District	No. of Admissions to Hospitals	Deaths in Hospitals	Total Deaths Registered in Dublin Registration District
1879	March	70	16	25	143	21	83
	June	57	13	20	72	8	51
	September	43	9	21	71	2	34
	December	49	6	23	81	5	37
	Total	**219**	**44**	**89**	**367**	**36**	**205**
1880	March	159	19	26	94	11	47
	June	129	32	44	67	4	63
	September	141	21	32	107	4	32
	December	470	44	55	109	4	46
	Total	**899**	**116**	**157**	**377**	**23**	**188**
1881	March	470	72	85	51	7	42
	June	357	42	54	35	2	28
	September	246	31	36	31	6	31
	December	147	18	19	52	7	22
	Total	**1,220**	**163**	**194**	**169**	**22**	**123**
1882	March	126	19	25	34	3	28
	June	96	16	19	44	2	33
	September	124	11	17	49	2	29
	December	245	18	23	75	2	45
	Total	**591**	**64**	**84**	**202**	**9**	**135**
1883	March	351	51	57	42	3	46
	June	253	28	48	30	3	27
	September	100	17	23	34	3	26
	December	125	9	13	76	9	33
	Total	**829**	**105**	**141**	**182**	**18**	**132**
1884	March	141	22	30	64	8	29
	June	122	20	21	48	3	29
	September	103	9	12	34	6	30
	December	103	14	20	72	12	46
	Total	**469**	**65**	**83**	**218**	**29**	**134**

TABLE II.—*continued.*

YEARS	QUARTERS	TYPHUS			ENTERIC FEVER		
		No. of Admissions to Hospitals	Deaths in Hospitals	Total Deaths Registered in Dublin Registration District	No. of Admissions to Hospitals	Deaths in Hospitals	Total Deaths Registered in Dublin Registration District
1885	March	78	15	17	58	3	33
	June	70	14	17	48	5	40
	September	54	6	14	73	5	24
	December	45	4	6	86	9	47
	Total	**247**	**39**	**54**	**265**	**22**	**144**
1886	March	38	9	18	75	9	33
	June	16	3	4	32	4	19
	September	38	7	11	55	7	25
	December	31	3	6	101	15	52
	Total	**123**	**22**	**39**	**263**	**35**	**129**
1887	March	33	7	5	65	5	37
	June	32	2	10	57	9	24
	September	28	2	5	75	5	29
	December	42	3	4	79	9	45
	Total	**135**	**14**	**24**	**276**	**28**	**135**
1888	March	80	7	9	65	6	37
	June	51	3	7	39	8	31
	September	27	5	5	91	5	33
	December	21	5	10	170	25	67
	Total	**179**	**20**	**31**	**365**	**44**	**168**
1889	March	46	8	7	126	12	48
	June	24	4	5	127	13	38
	September	27	3	3	176	16	41
	December	35	3	3	354	38	101
	Total	**132**	**18**	**18**	**783**	**79**	**228**
1890	March	48	5	4	155	23	58
	June	36	5	7	135	8	32
	September	14	1	2	106	16	32
	December	61	9	12	240	19	63
	Total	**159**	**20**	**25**	**636**	**66**	**185**



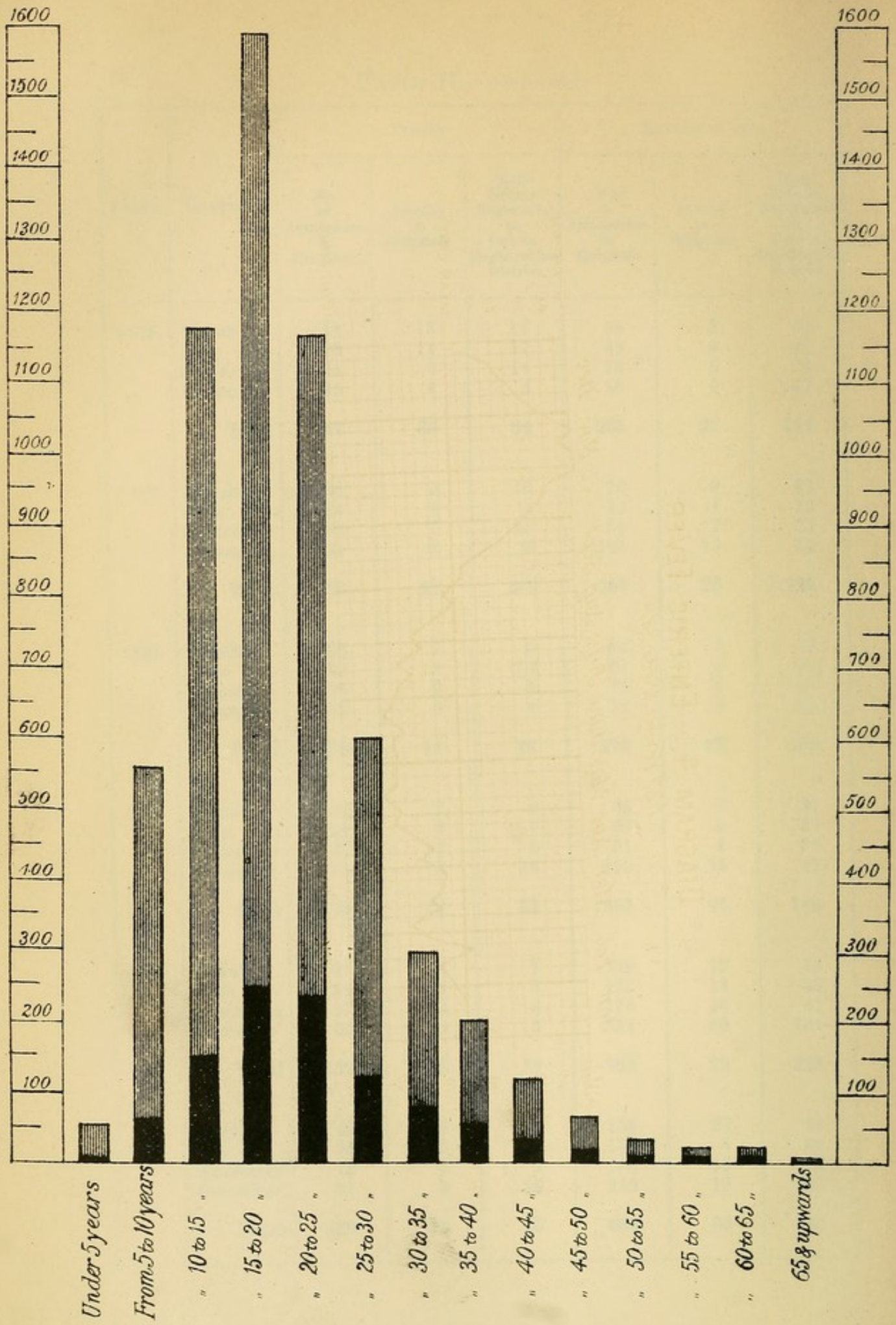
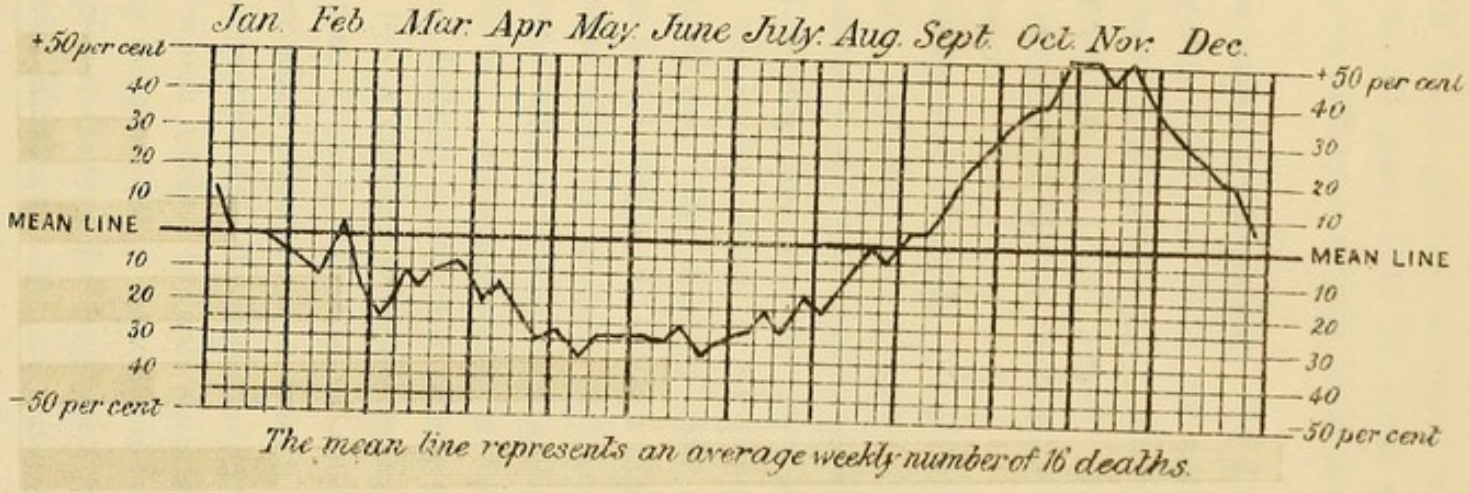


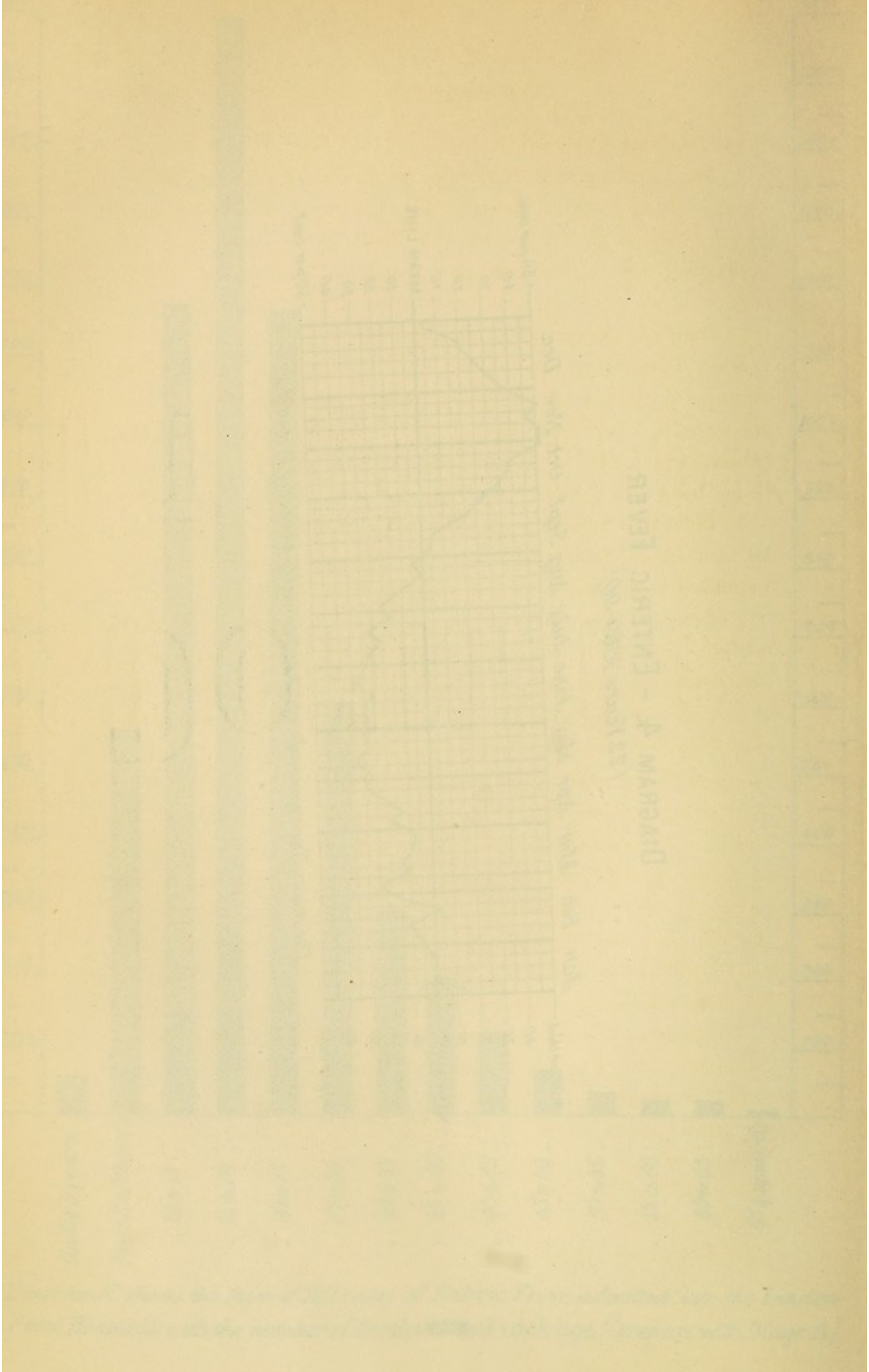
Diagram C shows the Ages of 5911 cases of Enteric Fever, admitted into the London Fever Hospital, with the number of Deaths ■ at each age. (Compare with Diagr. A)

DIAGRAM 4. – ENTERIC FEVER.

(22 Years 1869-90)



The mean line represents an average weekly number of 16 deaths.



Predisposing Causes :—

1. **Sex** has little or **no influence** in determining the occurrence of this disease. Of 5,988 patients admitted into the London Fever Hospital in the 23 years, 1848–70, 3,001 were males and 2,987 were females—a difference of only 14 in favour of the male sex.

2. **Age** is a **more potent predisponent**. Of 5,911 cases in the London Fever Hospital, 3,926, or 64·71 per cent. were between 10 and 24 years of age. Enteric fever is a disease of youth and adolescence, the mean age of 1,772 cases being 21·25 years (Murchison). The influence of age is illustrated in Diagram C., from Murchison's work.

3. **Defective Sewerage and Drainage.**—In the United Kingdom, as well as in France, Germany and America, enteric fever has **increased** in the presence of **shortcomings in sanitation.**

4. **Season.**—This fever is most prevalent in **autumn** and **early winter**—hence the names by which it is often described in America—"**Autumnal**," or "**Fall Fever.**" The exciting cause of the disease seems to be called into action only, as Murchison says, "by the *protracted* heat of summer and autumn, while it required the protracted cold of winter and spring to impair its activity or to destroy it." An examination of the returns of the Registrar-General for Ireland shows that enteric fever exhibits — as the summer rolls by—a decided tendency to increase in Dublin at an earlier period than typhus. This is, no doubt, partly due to the fact that the secondary phenomena of enteric fever are generally developed in connection with the digestive system, acute and infective diseases of which increase towards autumn.

The accompanying Diagram (4) is reproduced from the Annual Summary of the Registrar-General for England for 1890.

5. **Temperature and Moisture.**—*Hot, dry, calm* summers increase the prevalence of enteric fever, which is less frequent in *cold, wet, stormy* seasons. *Warm, damp* weather, however, predisposes to the disease. Floods occurring in badly-drained localities may impregnate sources of drinking water with the germs of enteric fever, and so lead to its outbreak.

6. **Soil** and **Underground Water.**—Professor von Pettenkofer and Professor Buhl,[a] of Munich, have shown that when the subsoil water in that city (as measured by the depth of water in the surface wells) is falling, the number of cases of enteric fever increases; when the water level is rising, the number of cases diminishes. Liebermeister and Buchanan suppose that these observations simply illustrate the mode in which the disease is communicated by means of drinking water. When the subsoil water is low any noxious matters in it accumulate and acquire a greater virulence.

In the case of an outbreak of enteric fever at Terling, Essex, in December, 1867, Dr. Thorne Thorne, Inspector of the Local Government Board of England, found that the disease had broken out with great severity precisely when the wells were high.[b]

7. **Idiosyncrasy.**—Many facts, says Murchison, "seem to show that certain **peculiarities of constitution** favour or avert an attack" of enteric fever.

8. **Recent Residence in an infected locality** is universally admitted to be a **powerful predisponent** to enteric fever. It would seem that the system through constant exposure becomes habituated to the effects of the poison, and in this respect enteric fever resembles dysentery, ague, and other malarious fevers.

Besides Age—Occupation, Station in Life, Overcrowding and Defective Ventilation, Previous Illness, Intemperance, Fatigue, and Mental Emotions—all seem to have little or no determining influence in increasing the liability to enteric fever.

Immunity.—As a general rule, one attack of enteric fever protects its subject from a second. At the same time **well-authenticated instances of persons contracting this fever a second time** are on record. Several have come under Murchison's notice, in which both attacks have occurred subsequent to puberty. Trousseau records two examples of a second attack—one patient, a woman,

[a] An excellent *resumé* of various papers on this subject in the *Zeitschrift für Biologie* will be found in the *Ugeskrift for Læger*, Copenhagen, January 30, 1869. A translation by my father, Dr. W. D. Moore, appeared in the *Dubl. Journ. of Med. Science*. Vol. XLVII., p. 497. May, 1869.

[b] *Tenth Report of the Medical Officer of the Privy Council.* 1868. Page 51.

suffered again after an interval of four years; the other, a girl, had a severe attack at the age of twelve, and another equally severe a year afterwards. Similar instances of unequivocal second attacks have been recorded by Piedvache, Michel, Bartlett, Paul, and William Budd.[a]

In April, 1877, I attended with the late Dr. Alfred Hudson, a lad of fifteen years, who passed through a typical attack of enteric fever lasting 23 days—in which, however, constipation and not diarrhœa was the rule. In October, 1891, the same gentleman, at the age of twenty-nine, sickened of a fever which proved to be undoubted enteric fever. The Charts in both illnesses are included in Plate IX.

Exciting Cause.—This is the introduction into a susceptible body of the specific virus of Enteric Fever.

Bacteriology.—That parasitic organisms, capable of self-reproduction, lie at the root of the toxic agent or that they are essentially related thereto, has long been conjectured, for that toxic agent is capable of reproduction to an extraordinary degree both within and without the human body. Furthermore, the disease shows a course which is strikingly typical, appearing as it does to be dependent upon the phases of development of such a *Contagium vivum* (Zuelzer).

The fullest accounts of recent researches into the micro-parasitic origin of enteric fever will be found in Dr. Gaffky's article on the "Etiology of Enteric Fever," which appeared in the second volume of *Mittheilungen aus dem Gesundheitsamte* (Berlin, 1884), and was translated for the New Sydenham Society, by Mr. J. J. Pringle, M.B., Assistant Physician to the Middlesex Hospital;[b] and in Mr. Watson Cheyne's translation for the same Society of *Micro-organisms with Special Reference to the Etiology of the Infective Diseases*, by Dr. C. Flügge, O. O. Professor and Director of the Hygienic Institute at Göttingen.[c]

[a] See Murchison. *Loc. cit.* Third Edition. 1884. Page 471.

[b] The New Sydenham Society. Vol. CXV., page 205, *et seq.* London: H. K. Lewis. 1886.

[c] The New Sydenham Society. Vol. CXXXII., page 248, *et seq.* London: H. K. Lewis. 1890.

So long ago as the year 1870, a communication was made by Coze and Feltz that they had found in the blood of typhoid patients an organised ferment, the shape of which recalled that of *Bacterium catenula.*

The earliest fruitful investigations on the subject were those by von Recklinghausen in 1871, Eberth in 1872, and Klein[a] in 1875. The first real advance was made, however, in 1880, when Eberth discovered a characteristic micro-parasite in the implicated viscera of enteric fever.[b] To this the name **Bacillus typhosus** (Eberth) was given. About the same time similar observations were made by Klebs and Eppinger,[c] and these have been confirmed by the researches of Robert Koch, Meyer, and Friedländer, and more recently by Gaffky and very many others (von Jaksch)—in our own country, by Coats and Crooke.[d]

Gaffky conducted a series of most accurate investigations on the subject in the laboratory of the Imperial Office of Public Health, Berlin, with the result that, in twenty-six out of twenty-eight fatal cases of enteric fever examined, the typical bacillar masses were present in the mesenteric glands, or in the spleen, liver, or kidneys. From this fact Eberth considers that the view that these organisms are in reality the cause of the typhoid process, has, without question, increased in probability to a high degree. Moreover, the bacilli do not apparently proliferate after death, as they would do had they anything to say to putrefaction. When animals have been infected by inoculation with these bacilli, they manifest symptoms of enteric fever, and the researches of E. Fränkel, M. Simmonds,[e] and C. Seitz,[f] seem to leave no doubt as to their pathogenic character, although Beumer and Peiper[g] have come to a different

[a] *Reports of the Medical Officer of the Privy Council and Local Government Board.* London. 1875.

[b] *Virchow's Archiv.* LXXXIII., 486. 1881.

[c] Klebs' *Handbuch der pathol. Anatomie,* 7 Leiferung, bearbeitet von Prof. Eppinger.

[d] *Brit. Med. Journal,* March 12 and July 1, 1882.

[e] *Centralbl. für klin. Med.* VI., 737. 1885.

[f] *Bacteriol. Studien z. Typhus-ätiologie.* Munich. 1886.

[g] *Zeitschrift für Hygiene,* I., 489, 1886, and II., 110, 1887.

conclusion. Gaffky has not succeeded in producing the disease in animals with his cultivations.

Bacilli have of late years repeatedly been found in the blood of typhoid patients, and von Jaksch[a] says "they are **doubtless the exciting cause of the disease.**" Rütimeyer[b] and Neuhaus[c] experimented upon the blood taken from the rose-spots, and succeeded in cultivating bacilli from it.

Bozzolo,[d] in three atypical cases, was able to demonstrate the presence of Eberth's bacillus in the blood, and thus determine that he was dealing with typhoid septicæmia. He considers bacteriological examination of the blood **of great diagnostic importance.**

Neumann,[e] again, has found the *Bacillus typhosus* in the urine in six out of twenty-three cases investigated. Karlinski, of Cracow, examined the kidneys and urine in six fatal cases of enteric fever and the urine in thirty-eight other cases of the same disease. Bacilli were found in all the kidneys examined. In twenty-one specimens of urine Eberth's bacilli were found, all these urines being albuminous. When **albuminuria was transient** or absent, **no bacilli** were found. Colonies rapidly increased in albuminous urine, and preserved their vitality for a long period; in urine containing bile, the bacilli died in five days.

An important question arises as to the **passage to the fœtus from the mother** of the micro-organisms of enteric fever. Eberth in particular has raised an objection to the experiments made by Reher, Neuhaus, Chantemesse, and Vidal,[f] which went to demonstrate the presence of the *Bacilli typhosi* in the blood of fœtuses derived from typhoid mothers. Eberth is of opinion that a confusion must have arisen between the bacteria of typhoid and the bacteria of putrefaction. In one of his later communications,[g]

[a] *Clinical Diagnosis.* Translated by James Cagney, M.D. London: Charles Griffin & Co. 1890. Page 33.

[b] *Centralbl. für klin. Med.* VIII., 145. 1887.

[c] *Berlin. med. Wochensch.* XXIII., 89 and 389. 1886.

[d] *Deutsche med. Zeitung.* Berlin. Aug. 28, 1890. Sajous' *Annual of the Universal Medical Sciences.* 1891. Vol. I., H.–52.

[e] *Berlin. med. Wochensch.* XXV., Nos. 7-9. 1888.

[f] *Recherches sur le bacille typhique et l'étiologie de la fièvre typhoïde.* Würzburg. 1888.

[g] *Centralblatt für die med. Wissensch.* 1889. Juni 1.

however, Eberth admits the correctness of the observations; and still more recently, Dr. Joseph Giglio,[a] of Palermo, in an original paper, entitled "Ueber den Uebergang der mikroskopischen Organismen des Typhus von der Mutter zum Fötus," as a result of his experiments, comes to the conclusion that the bacillus cultivated by him is the genuine *Bacillus typhosus*. To confound it with saprophytes is out of the question.

Gaffky's results agree entirely with the descriptions given by Eberth, Koch, and Meyer in regard to the shape and appearance of the bacilli. On the average the rods are about thrice as long as they are broad; their length corresponds to about the third part of the diameter of a red blood corpuscle. The rods are 2 μ. broad, and form filaments up to 50 μ. long. In isolated spots one may see somewhat longer threads, which on more thorough examination are seen to be made up of several segments. The extremities of the bacilli are distinctly rounded off. In several of the cases examined by Gaffky, the bacilli found in the internal organs contained unmistakable spores, which appeared as round portions, remaining unstained and occupying the whole breadth of the bacilli.

The *Bacillus typhosus* occurs in the stools of enteric fever, but it is impossible to recognise it with the microscope alone, owing to the vast number of microbes constantly to be found in the dejections of this disease. Nor does it stain well for diagnostic purposes. Hence it is necessary to obtain pure cultivations for its complete recognition.

The difficulty of staining this organism, and its occurrence in solitary isolated clumps, enabled it for a long time to elude the vigilance of observers. In connection with this, Dr. German Sims Woodhead mentions a plan which may prove useful to inquirers. "These bacilli," he writes,[b] "are said to be stained with difficulty, but I have found that if the sections in which they are present are first allowed to remain for about ten minutes in a one-fifth per cent. solution of corrosive sublimate, and then stained by Gram's method, the bacilli are most deeply stained, although Fränkel and other

[a] *Centralblatt für Gynäcologie.* 1890. No. 46, page 819.
[b] *Bacteria and their Products.* London: Walter Scott. 1891.

observers state that the colour is invariably discharged if Gram's method be used." This, however, is only part of the difficulty, for although the bacilli are found in clumps in the adenoid follicles, in the spleen, and commonly in the mesenteric glands, it is only, as Flügge says, "after the examination of a large number of sections that one or several of these follicles can be found."

The *Bacillus typhosus* develops readily in a medium of nutrient gelatin—the cultivations appearing after twenty-four hours. Rods and threads appear under the microscope, and they seem to be endowed with an evident and peculiar motion. The parasite develops to an extraordinary extent on prepared potato at a temperature of 37° C. (98·6° Fahr.). Spores form after three or four days. On microscopical examination the inoculated mobile bacilli are found in surprising abundance. The whole surface seems to consist almost entirely of bacilli, which stain only with moderate intensity with aniline dyes. A globulin substance, behaving like myosin, seems to be the nutrient material in the potato. Typhoid bacilli also grow luxuriantly on sterilised blood serum of sheep, in solidified blood serum around punctures made with the platinum needle, in meat infusion, as well as in some vegetable infusions (Gaffky).

A discussion has lately taken place as to the mode of origin of enteric fever in India and other tropical and subtropical climates, and is referred to by Dr. Cayley in the Third Edition of Murchison's *Treatise on the Continued Fevers*. In this discussion there seemed to be a general consensus of opinion that the disease may arise *de novo*, and two theories were put forward as to the mode of origin—the entogenous and the ectogenous. The former theory implies that the disease may be generated in the system, without any infection from outside the body, from the effects of climate, changed modes of life, or even the decomposition of fæces in the intestinal canal. This apparent mode of origin I myself long since recognised, chiefly in relation to retention and decomposition of the fæces in the bowels, but I prefer the term "autogenous" or perhaps "autochthonous," to "entogenous," as an adjectival prefix to enteric fever arising in this way.

However, since the discovery of Eberth's *Bacillus typhosus*, and the establishment of its causal relation to enteric fever, **the doctrine of the *de novo* or spontaneous origin of the disease**, whether *without* (pythogenic) or *within* (autogenic) the body, **has become untenable.**

My opinions, therefore, have of necessity been modified, and the doctrine I now hold is this—Enteric fever arises only when the spores of the specific microbe already named enter the body, and especially the intestinal canal, of a susceptible individual.

But the state of health which accompanies habitual constipation, with fæcal decomposition in the intestines the result of that constipation, enormously increases the susceptibility of an individual, and, in fact, acts so powerfully as a *predisposing cause* as almost to appear to be the *exciting cause* of an attack of the disease.

I believe that under such circumstances a very minute dose of the specific poison will suffice to kindle an attack of enteric fever, which might consequently seem to be of independent or spontaneous origin—to be, in a word, autogenous or autochthonous.[a]

Enteric fever probably arises in the following way:—The specific bacilli, or rods, form spores inside the organs of one sick of the disease, especially in the mucous membrane of the small intestine. The micro-organisms are then discharged with the motions in their most resistant condition—*i.e.*, as **resting spores**—and thus pass into faulty or leaky drains or cesspools, or into the ground. In these situations they may remain quiescent, and therefore harmless, for a long time for want of suitable nourishment or of a suitable temperature. At last these resting spores arrive by chance in a body capable of being infected, and there they develop into bacilli, and begin anew their cycle of existence as pathogenic microbes. Gaffky further shows that it is highly probable that the development of the spores does not occur

[a] These several terms are derived from the Greek, namely :—
"Entogenous," from *ἐντός, within ; γεννάω, I beget* or *produce.*
"Ectogenous," from *ἐκτός. without ; γεννάω.*
"Autogenous," from *αὐτός, self ; γεννάω.*
"Autochthonous," from *αὐτόχθων, sprung from the soil itself* (Lat. *terrigena*), *indigenous.* From *αὐτός, self ; χθών, the earth, ground.*

only in the human organism, but that—as in case of the bacilli of splenic fever (*Bacillus anthracis*)—they may sprout and form bacilli in favourable circumstances even *outside the animal economy*, increasing enormously in numbers, and in the warmer part of the year forming spores afresh.

Paths of Infection.—In Gaffky's opinion—and he is no doubt correct in his view—the most diverse paths stand open to the infecting germs for admission into the human organism, and it would be one-sided to fix one's attention solely on one or another of these. Besides the air which we breathe, and the water which we drink, our food may be the carrier of the typhoid virus.

I. The poison may be **transported by currents of air.** Murchison cites remarkable instances of such an occurrence—outbreaks of enteric fever having been caused by exhalations from drains, or sewers, or water-closets. Such outbreaks took place at the Peckham police station in 1859, at Chatham in 1872, in the School and Abbey Cloisters at Westminster in 1848, and at a school at Clapham in August, 1879. Under these and similar circumstances pneumonic fever, called by Dr. T. W. Grimshaw and me "Pythogenic Pneumonia,"[a] is also apt to arise.

II. The poison may be, and probably most usually is, **conveyed by drinking water.** Of this Dr. Hilton Fagge specifies not fewer than 11 very striking and conclusive examples. The most singular of these was an outbreak at Lausen in the Valley of Ergolz in the Jura in August, 1872, when 130 out of a population of about 800 people were attacked, all of whom used the water of a public fountain, fed from two sources. One of these was a spring into which water percolated from certain meadows in another valley, separated from the Ergolz Valley by a mountain, the Stockhalder. In that other valley two cases of enteric fever occurred in July, 1872. The discharges from the patients were thrown into an adjoining stream. In the middle of the month just named the water of this stream was used to irrigate the meadows, and three weeks later the epidemic at Lausen began.

[a] *Dubl. Journ. of Med. Science.* 1875. Vol. LIX., page 399.

III. **Milk may be contaminated** with the poison, and when used may give rise to the disease.

The first epidemic traced to such an origin occurred in Islington in 1870, and was investigated by Dr. Edward Ballard. Similar outbreaks took place at Armley, near Leeds, in the summer of 1872, and in the St. Marylebone District, London, in the summer of 1873. About Christmas, 1878, a remarkable epidemic was caused by infected milk in the Pembroke Township, Dublin. This was thoroughly investigated by Sir Charles Cameron, who published an excellent account of it in the *Dublin Journal of Medical Science* for July, 1879 (Vol. LXVIII., No. 91, Third Series). All the households affected were supplied with milk from a particular dairy, the owner of which had been ill with "fever" since the middle of December. Two children had also been ill with fever of some kind during the same month, and all the patients had remained in a small house attached to the dairy. The excreta were daily thrown out on a dung-heap close to the cow-sheds, and the milk-pails were so placed that they could hardly escape being infected by means of particles of matter from the dung-heap carried by the wind.

The bibliography of milk-epidemics of enteric fever is rapidly increasing.

In the "Transactions of the Second Intercolonial Medical Congress of Australasia," Dr. Allen records the occurrence of an outbreak at Melbourne in the spring of 1879. The son of a milkman died of enteric fever. Of 93 households supplied with milk by the milkman in question, 23 were visited by enteric fever. Forty persons were attacked, of whom 3 died. In the middle of June, 1889, enteric fever suddenly appeared in a community of 3,000 inhabitants in Sweden. One hundred and four cases with 11 deaths occurred in less than four months. It was discovered that those attacked received their milk from the same dairy. The milk was gathered from various sources and redistributed. Two persons ill of fever, presumably typhoid, were probably the starting point of the outbreak.

In the *Edinburgh Medical Journal* for 1890-91 (Vol. XXXVI.,

pages 801–814), Dr. H. Littlejohn reports an outbreak of typhoid fever due to milk infection.

To the *American Lancet*, Detroit, 1891 (New Series, XV., pages 121–128), E. P. Christian contributes a paper, entitled "Cows' Milk and Typhoid Fever."

In 1890, A. Vincent[a] published a "Note sur une épidémie de fièvre typhoïde propagée par le lait."

IV. There is some reason to believe that **meat** may, under exceptional circumstances, **convey the poison** of enteric fever. Dr. Cayley[b] states that several outbreaks from this cause have been reported from Switzerland, where cattle are, according to Huguenin,[c] not uncommonly affected by true enteric fever. The most remarkable outbreak of this kind was one which took place at Kloten, near Zurich, in 1878. Upwards of 700 persons were attacked in consequence of eating decomposed veal from a calf, which Huguenin believes to have been affected by enteric fever.

Legrain (*Ann. de la Policlin. de Paris*, October, 1891, and *Brit. Med. Journ.*, November 7, 1891) says that the problem of the origin and propagation of enteric fever is probably solved by the discovery of Eberth's bacillus and by the finding of this micro-organism in the water suspected of conveying the disease. Three theories have been put forward—(1) autotyphisation, and the identity of the *B. coli communis* with Eberth's bacillus lends some strength to this idea; (2) direct contagion; and (3) the presence of the germ in external media, especially in water, and its transmission in that way. This last is the most generally accepted theory. Brouardel in 1887 admitted that though the disease could be propagated by water, air, the patient's linen, or the hands of those looking after him, yet in 99 out of 100 cases it was through the medium of water. The author then gives the details of an epidemic occurring in a home for idiots and the weak-minded. After describing the insanitary condition of the river, M. Legrain draws attention to the increasing prevalence of gastro-intestinal

[a] Genève. Taponnier et Studer. 1890. Page 15.
[b] *Croonian Lectures*, 1880.
[c] *Correspondenz-Blatt für schweiz. Aertze.* No. 75. 1878.

affections in the home. Thus among an average of 170 treated yearly, there were 25 such cases in 1887, 57 in 1889, and 84 in 1890, the epidemic taking place from January to May of the last-named year. Two conclusions are drawn—(1) that the highest figure representing the gastro-intestinal disturbance and that representing enteric fever corresponded, and (2) that the epidemic was the climax of a preparatory period of gastro-intestinal disorders. The author agrees with Chantemesse that gastric disturbance with fever, apart from simple indigestion, may be abortive enteric fever.

Mode of Invasion.—A question arises as to the organ in which the typhoid germs that have once entered the body first settle, and afterwards extend from this their primary seat of invasion, so as to bring about the general disease. Eberth lays stress upon a case observed by Meyer,[a] in which death ensued on the second day of illness. In this case there were found at the *post-mortem* examination hyperæmia of the lungs, spleen, and kidneys; in the lower portion of the ileum marked swelling of the solitary follicles and Peyer's patches, but nowhere any trace whatever of necrosis or of loss of substance. None of the mesenteric glands were swollen. In this recent case, microscopical examination revealed an exceptionally large deposit of the bacilli of Eberth and Koch in the cells of the submucosa and in the intermediate muscular layers.

Eberth concludes from this case that the bacilli are first localised in the intestinal mucous membrane, that thence they pass into the mesenteric glands, thence into the blood-stream and accumulate again in the spleen, and, as Gaffky would add, in the other organs. Eberth further points out that anatomical investigations have afforded no evidence of the admission of typhoid germs through the lungs. On this point Gaffky does not agree, for he considers it as highly probable—or at least the possibility cannot be contested—that the lungs may occasionally represent the seat of invasion. Be this as it may, there is no doubt that a close correlation exists between enteric fever and that variety of acute pneumonia, or **pneumonic fever**, to which the term "**Pythogenic Pneumonia**" is now applied.

[a] *Der Typhus-Bacillus und die intestinelle Infection.* Volkmann's *Klinische Vorträge.* 1883. No. 220.

At the time of writing (November, 1891), there is under my care in the Meath Hospital a young woman with characteristic typhoid stools, and whose urine gives a striking reaction with Ehrlich's test; but whose illness began with right apex pneumonia, with rapid breathing, cough, glutinous expectoration (not, indeed, deeply coloured when the patient was first seen by me), dulness on percussion, and, finally, the most typical *crepitus redux*.

Towards the end of October, 1882, the following remarkable outbreak of disease came under my notice. On the 12th of that month a lad, aged thirteen, was admitted into Cork-street Fever Hospital from 6 Malpas-street, Dublin, suffering from acute pneumonia.

Malpas-street is very unhealthy—the houses are old and dirty, ill-drained and dilapidated. The street runs down to the bottom of a valley, through which a small tributary of the Poddle river flows sluggishly. The district is a prolific hotbed of disease. On Oct. 31 the boy's father (John C.), a boatman, aged thirty-six, came in with the same disease. On the 20th of the same month two girls, both aged fourteen, were admitted to the Meath Hospital in enteric fever—one from 11 Malpas-street and the other from No. 13. On Nov. 27 a girl, aged twenty, was admitted to Cork-street Hospital in enteric fever from 7 Malpas-street, next door to the house from which the two cases of pneumonia had come a few weeks previously. On Dec. 12, John C., was again admitted to the Meath Hospital from 6 Malpas-street with "renal dropsy." It was he who suffered from pythogenic (?) pneumonia in the previous October, as narrated above. Another coincidence occurred in March, 1883. On the 18th of that month Winifred N., aged nineteen, came into Cork-street Hospital from 6 Malpas-street in an attack of "febricula," and the following day Anthony L., aged twenty-seven, was admitted from the same house with left basic croupous pneumonia.

A very similar instance of the correlation existing between enteric fever and pneumonia came under my observation in the autumn of 1881. Four cases of illness occurred in a Training College in Dublin within a few weeks. Two of the four patients suffered from true enteric fever; a third, from an attack of acute

gastro-intestinal catarrh or—as some may think—from an abortive enteric fever; and a fourth from acute pneumonia, reminding one of Laennec's "epidemic pneumonia," which in recent times has received the names of "sewer-gas" or "pythogenic" pneumonia. The drinking-water was proved by Sir Charles Cameron[a] to be the source of the sickness in all the four cases.

"Whether it be," writes Gaffky, "that the spores of the typhoid bacilli are taken up in drinking water, or, in rare cases, along with articles of food; whether it be that they are inspired with the air breathed, remain attached to the mucous membrane of the mouth and throat, and are afterwards swallowed, in all probability they pass without damage through the stomach, sprout to form bacilli in the alkaline contents of the intestine, multiply there and penetrate into the intestinal mucous membrane at those spots which are most adapted for their reception—viz., the Peyer's patches and solitary follicles. Afterwards they arrive in the mesenteric glands, where they form the very numerous characteristic masses, and are then carried away in the blood-current into the other organs. Becoming fixed here and there in these, they multiply to form those groups, the almost constant presence of which in the spleen, liver, and kidneys, has been described in detail in the earlier part of this work. Obviously the infection will be more certain to ensue the greater the number of spores taken into the body."

Virchow[b] maintained that infection in enteric fever most frequently took place by means of drinking water. He wrote: "The striking limitation of the anatomical changes to a deeply situated portion of the intestine, the lower end of the ileum and cæcum, seems to indicate that a local action of the morbific matter takes place, as these are exactly the situations where the contents of the intestine are relatively most frequently retarded, where therefore the longest contact with the mucous membrane occurs. This consideration harmonises best with the inception of the typhoid matter in drinking water."

[a] *Dubl. Journ. of Med. Science.* February, 1882.

[b] "*Canalisation oder Abfuhr. Eine hygienische Studie.*" Virchow's *Archiv*. Band 45. Heft 2. Side 294.

CHAPTER XXXVII.

Clinical Description of Enteric Fever.

Stage of Incubation or Latent Period—Stage of Invasion—Stage of Glandular Enlargement — Ulceration and Sloughing — Amphibolic Stage—Stage of Lysis—Convalescence—Duration of the Fever.

As in the case of Typhus, we may most conveniently consider the clinical history of Enteric Fever as the disease passes through the different periods or phases of its development—the stages, in fact, of incubation, invasion, glandular enlargement, ulceration or sloughing of the intestinal glands and lysis, and convalescence (Murchison.)

I. Stage of Incubation.—From elaborate researches, Murchison was led to conclude that—1. The **period of Incubation** of enteric fever is most commonly **about two weeks**; 2. Instances of a longer duration are more common than in typhus or relapsing fever; 3. It is often less than two weeks, and **may not exceed one or two days.**

Liebermeister admits that the period of incubation is difficult to be determined, because "it is hard to fix the exact date of the infection, and often hard to fix that of the commencement of the disease." He considers that, according to our present (1875) experience, the average period of incubation is three weeks. In a paper, "Ueber die Incubationzeit des Abdominaltyphus," published in 1875, Professor Quincke,[a] of Berne, gave some cases—due to drinking contaminated water—in which the duration of incubation was very accurately ascertained. In these cases the shortest period was eight days, the longest certain period between sixteen and eighteen days. In the Marylebone milk epidemic of 1873, a child was taken ill five days after drinking the infected milk (Cayley). Hilton Fagge is responsible for the statement that

[a] *Correspondenz-Blatt für schweizer Aerzte.* 1875. No. 8.

it has been conjectured the incubation is shorter when the poison is inhaled with the breath, longer when it is swallowed in drinking water.

During this **latent period**, the patient may feel quite well, or, on the other hand, he may complain of being "out of sorts;" there may be languor, a tendency to diarrhœa, and vague feelings of discomfort and chilliness, with headache and loss of appetite.

II. **Stage of Invasion.**—This lasts from the first marked feeling of illness until decided febrile symptoms have developed. It is a badly defined period, for **the advent of enteric fever** is essentially **gradual** and **insidious** in most cases. It lasts, according to Murchison, for one or more days.

Of late years the classical insidious onset of enteric fever has in many instances given place to a more abrupt and vehement advance, characterised by decided rigors, violent headache, and rapid rise of temperature. This, at least, has been our experience in Dublin during and since the epidemic of 1889. In a word, the whole course of the disease has become more typhus-like than formerly.

The earliest symptoms are, headache and pains in the limbs, with irregular chills, giddiness, and languor. Sometimes nausea and vomiting are prominent symptoms, and still more frequently there is urgent diarrhœa, particularly if the patient has taken a saline aperient while under the impression that he is suffering from a mere bilious attack.

III. **Stage of Glandular Enlargement.**—This overlaps the invasion, and extends to the twelfth or fourteenth day. The pulse now increases in frequency, temperature rises, especially in the afternoon or evening, the fever being of a remittent type, the skin is hot, the tongue is furred and red at the edges, and epistaxis is apt to occur. The nights are restless and disturbed, and the patient feels weak, but not to anything like the same extent as in typhus. Indeed it is quite usual for the sufferer from enteric fever to pursue his ordinary avocations during the whole of the first week of his illness, or even longer.

So far there is nothing absolutely pathognomonic of enteric fever,

but Murchison points out that the concurrence of diarrhœa or gastric disturbance, with an evening temperature of 103° or 104° F. and prostration, in a young person, ought always to make the practitioner suspect that this is the disease which he has to combat.

In very rare instances enteric fever sets in with sudden maniacal delirium (Hilton Fagge). On the other hand, a person may *ail* for one, two, or three weeks, after which a definite attack of this fever may begin and run its usual course.

At the close of the first week and during the early days of the second, the symptoms are more or less headache, prostration, loss of appetite, diarrhœa with liquid motions of an ochrey-yellow colour, a frequent soft pulse liable to great variations in rate and strength, fever of a variable remittent type. There is a warm, dry skin, with occasional clammy sweating. The pupils are dilated, and the conjunctivæ are clear, the nose bleeds from time to time, there is a clear complexion with a hectic flush upon the cheek, so that the patient is like one in pulmonary consumption.

The **tongue** is at first covered with a creamy fur, through it the enlarged papillæ begin to show, and a triangular red space appears at the tip. The edges also are red. The epithelium is subsequently shed, and the surface of the whole organ in severe cases becomes red, glazed, dry, and transversely fissured and sore. Occasionally the tongue becomes cracked, dry, and brownish. In most cases, but not invariably, the area of **splenic dulness** is **increased**. Tympanites, or meteorism, is commonly observed, sometimes to an extreme degree. There is tenderness on pressure over the abdomen, with gurgling *(gargouillement)* in the iliac fossæ. Intestinal hæmorrhage may occur. Occasionally constipation prevails and may even be obstinate.

The Rose-Rash.—Between the seventh and twelfth days an eruption of isolated, elevated, rose-coloured spots first appears, chiefly over the abdomen and back. The soft papules, of which this almost pathognomonic rash consists, are deleble on pressure, vanishing to reappear when pressure is removed. They come out in successive crops for ten days or a fortnight, or longer, each crop lasting for two, three, or more days. There may be only three or

four rose-spots, but on the other hand they may be counted by hundreds.

This stage of the fever derives its name from the enlargement of the intestinal and mesenteric glands which is proceeding apace during the first fortnight of the disease.

IV. **Stage of Ulceration and Sloughing.**—This stage extends from about the twelfth or fourteenth day to some time between the twenty-first and twenty-eighth day (Murchison).

In mild cases cerebral symptoms may be wanting, but in the severer forms of enteric fever headache gives place in the third week to somnolence, increasing delirium, particularly at night, and progressive nervous and muscular prostration. **Inordinate tremor** is regarded by Sir William Jenner as indicative of the presence of deep ulceration of the intestine, such as is likely to lead to perforation or hæmorrhage, and Murchison held the same opinion. In these serious cases also sordes collect on the teeth, diarrhœa persists, the tongue is dry, glazed, red, and fissured, and bed-sores are apt to form over the sacrum and near the great trochanters. The patient daily loses strength and wastes rapidly, and at last the "typhoid" or "ataxic state," of which I have so often made mention, may be developed, the patient either dying of coma or slowly improving towards the close of the third or in the fourth week.

In many instances, the typhoid stage is never reached. Yet the patient is not free from danger, for, apart from pulmonary or other complications, he may succumb in the third or fourth week to one or other of the perilous results of the intestinal lesions—exhausting diarrhœa, hæmorrhage, or—most perilous of all—perforation of the intestine, and peritonitis—with or without perforation—local or general.

The duration of this stage is uncertain, and for a time there may be a period of uncertainty, or of changing fortunes (Murchison), to which Wunderlich[a] has given the name of the **amphibolic**[b] **stage.** This doubtful period usually follows the acme, or fastigium, of the fever, and in it the range of temperature is more or

[a] *Medical Thermometry.* New Syd. Soc. 1871. Page 315.

[b] Gk. ἀμφίβολος, *attacked on both* or *all sides;* hence, *doubtful, ambiguous.*

less irregular. After remissions, or even a partial collapse, **recrudescences** of the fever, lasting for several days, may occur. These "flarings" of the expiring fever must be distinguished from true relapses in convalescence.

V. **Stage of Lysis.**—As it usually begins, so enteric fever ends—**gradually.** Resolution takes place by **lysis, not by crisis.** This is the rule, but there are exceptions to it. For example, Dr. Hilton Fagge cites the case of a medical friend, whose temperature (which had been ranging between 101° and 103° with great regularity) was found about the twentieth evening to be only 101·1°; next morning it was 100°, in the evening 99·3°; on the twenty-second morning it was 98·3°, and for several days afterwards it remained slightly below normal. A very similar case has lately passed through the hands of Dr. H. T. Bewley, of the Adelaide Hospital, Dublin. Through his kindness I have been enabled to reproduce the temperature chart of his patient's case in Plate VIII. (Fig. 9). In most cases, however, the fever subsides day by day, and the morning temperatures may not exceed 99° or 98° or 97°, while the evening readings still reach 104° or 103° or 102°. The temperature range, therefore, is of an intermittent or remittent type.

These "**spikings**" of the temperature daily tend to become less and less marked. Simultaneously, the tongue becomes moist—its epithelial coat being replaced—the head symptoms abate, and fresh rose-spots fail to appear.

VI. **Convalescence,** according to Murchison, can be said to be fairly established only *when the temperature is normal on two successive evenings.* Independently of its liability to interruption by complications, relapses, or sequelæ, this stage is slow and halting. The pulse remains quick, and appetite, health, and strength are regained only by degrees. I have repeatedly met with instances in practice where six months, or even a year, elapsed before the wonted health and strength returned.

Duration of Enteric Fever.—In ordinary cases this is **from three to four weeks.** Of 200 cases which recovered, Murchison ascertained the mean duration to be 24·3 days; of 112 cases which proved fatal, the mean duration was 27·67 days. Of 215 cases

noted by C. E. Hoffmann,[a] the mean duration was 28·9 days. In children, especially, the fever may terminate several days before the end of the third week (Hilton Fagge). On the other hand, death may supervene at an early date, rarely however, before the fourteenth day. Both Trousseau[b] and Hoffmann,[c] indeed, report each a case fatal in less than four days, and Murchison[d] gives details of one fatal in 47 hours. Trousseau's case of short duration is particularly interesting, because he was able to fix with precision the date at which the attack began, and after death on the fifth day, the intestinal lesions were found present in a marked degree—"l'autopsie cadavérique permit de constater l'existence d'une des éruptions dothiénentériques les plus confluentes que nous ayons jamais vues, et, chose remarquable, nous n'étions qu'au cinquième jour de la maladie."

[a] *Untersuchungen über die path. anat. Veränderungen der Organe beim Abdominaltyphus.* Leipzig. 1869.

[b] *Clinique méd. de l'Hôtel Dieu de Paris.* 1865. Vol. I., page 244.

[c] *Loc. cit.*, p. 38.

[d] Murchison. 1884. 3rd Edition. Page 551.

CHAPTER XXXVIII.

Analysis of the Symptoms of Enteric Fever.

The Physiognomy of Enteric Fever.—The Surface : lenticular rose-spots, *taches bleuâtres*, purpura spots, vibices, sudamina, accidental or adventitious rashes.—Desquamation.—The Circulation.—The Respiratory System.—The Digestive System : nausea, vomiting, meteorism or tympanites, *gargouillement*, constipation, diarrhœa, intestinal hæmorrhage.—Spleen.—The Urinary System.—The Nervous System : Liebermeister's four grades of nervous disturbance.—Angel Money's observations on muscular irritability.—Organs of Special Sense : the eye, ear, nose, cutaneous sensibility.—Emaciation.

Following Murchison's systematic plan, let us now consider the different symptoms in more detail.

I. **The Physiognomy.**—The patient in enteric fever wears an expression of languor, *ennui*, and even sadness. The heavy stupid expression of typhus (**Facies typhosa**) is absent except in those severe cases where the disease assumes the typhoid or ataxic state. There is commonly a pink blush on one or both cheeks, like the flush of hectic fever in a consumptive patient.

II. **The Surface.**—The skin is at first hot and dry, becoming moist in the evenings; afterwards a tendency to perspiration becomes more decided. After the seventh day small, circular, pink or rose-coloured spots appear, in greatest number on the lower part of the abdomen and the back. The total number may not exceed ten, twenty, or thirty; but, on the other hand, there may be several hundred. These spots are elevated and resemble papules of smallpox in their first stage, but unlike them they are never hard or "shotty." They can be felt as rounded—not conical—elevations in the skin. It is necessary to look for them the moment the surface of the body is stripped, for exposure to the cold air at once causes the development of *cutis anserina*. As a result of this, the arterioles of the skin are compressed and themselves contract. In this way the blood supply to the rose spots is cut off, so that they become pale or entirely disappear for a time. They disappear on pressure to reappear once pressure is removed. Their diameter is from half a line to two lines. In profile, they are like little lenses set in the skin—hence Louis' term adopted by Trousseau—**taches**

rosées lenticulaires, that is, *lens-like*, or *lenticular*, *rose-spots*. The rose-spots come out in **successive crops**, each set lasting three or four days; and this goes on for seven, fourteen, or even twenty-one days. There is **no relation between the number** of the spots **and the severity of the attack.** They disappear after death, and never become petechial during life. From the statistics of the London Fever Hospital the rash of enteric fever would seem to be present in 77 per cent. of the cases. Liebermeister thinks that the rash is seldom absent at some stage of the fever, but there can be no doubt that it is often wanting, more particularly in children.

Murchison draws the following contrast between the rose-spots of enteric fever and the maculæ of typhus:—

Enteric Fever.	Typhus.
1. Pink or rose-coloured throughout.	1. May be dirty pink or red at first, but soon become reddish-brown.
2. Undergo no change, until they fade or disappear. Never converted into petechiæ.	2. Become gradually darker, and are often converted into petechiæ.
3. Circular—half a line to two lines in diameter.	3. Of irregular form.
4. Isolated and few in number.	4. Numerous, and adhere in patches.
5. No subcutaneous mottling.	5. Mottling in addition to spots.
6. Elevated above the skin.	6. Not elevated, except at first appearance.
7. Disappear on pressure as long as they last.	7. Do not disappear on pressure, except at first.
8. Rarely appear before the 7th day.	8. Appear on 4th or 5th day.
9. Appear in successive crops.	9. Never in successive crops.
10. Each spot lasts only three or four days.	10. Many of the spots may last to the end of the fever.
11. Never present on the dead body.	11. Often persist after death.
12. A large number does not indicate danger.	12. Direct ratio between the number and darkness of the spots and the severity of the case.

Besides these pathognomonic rose-spots, spots of a delicate blue-tint of indefinite size and shape are sometimes observed on the fair skin over the abdomen and back of the typhoid patient. These are the **maculæ cæruleæ** of the writers, the **taches bleuâtres** of the French. There is nothing pathognomonic in these markings—first, because they are found in other diseases, such as pneumonia,

simple fever, and non-maculated typhus; secondly, because they seem to arise from an independent cause—the irritation of pediculi (see page 236).

Other epiphenomena of the disease connected with the skin are:—**purpura spots, vibices,** and **sudamina** or **sweat-vesicles.** The last-named are thus described by Trousseau, in his *Clinique Médicale de l'Hôtel Dieu*:—"La miliaire pellucide, improprement appelée *sudamina*, qui apparait ordinairement du onzième au vingtième jour, quelquefois plus tard, est constituée par de petites bulles, arrondies ou oblongues, ressemblant alors à des larmes, remplies d'un liquide transparent."

Occasionally the skin is affected by the co-existence with enteric fever of other specific diseases showing characteristic symptoms, such as scarlatina, measles, smallpox, and above all typhus.

In addition to all the foregoing, I drew attention, in a paper[a] read before the Royal Academy of Medicine in Ireland on November 16, 1888, to certain other **accidental** or **adventitious appearances of the skin,** which are of a somewhat rare occurrence, but, from a diagnostic point of view, of considerable importance. These are—(1) **Simple Hyperæmia**; (2) **Miliary Eruptions**; (3) **Erythematous Rashes**; (4) **Urticaria.**

In the four cases detailed in that paper, there was no evidence to connect the skin rashes with the administration of any drugs.

It is necessary to make this statement, remembering, with Dr. Radcliffe Crocker,[b] that "a precisely similar rash occurs after certain drugs, especially copaiba, quinine, belladonna, salicylic acid, and others. In these cases the rash is probably due to irritation of the alimentary canal acting reflexly on the vaso-motor centre."

The conclusions I arrived at were as follow:—

1. Not infrequently, in the course of enteric fever, an adventitious eruption occurs, either miliary, urticarious, or erythematous.

2. When this happens, a wrong diagnosis of typhus, or measles, or scarlatina may be made, if account is not taken of the absence of the other objective and subjective symptoms of these diseases.

[a] *Accidental Rashes in Typhoid Fever.* Trans. Royal Acad. of Med. in Ireland. 1889. Vol. VII., page 10.

[b] *Diseases of the Skin.* London: H. K. Lewis. 1888. Page 58.

3. The erythematous rash is the most puzzling of all; but the prodromata of scarlet fever are absent, nor is the typical course of that disease observed.

4. This erythema scarlatiniforme is most likely to show itself at the end of the first, or in the third week of fever.

5. In the former case, it probably depends on a reactive inhibition of the vaso-motor system of nerves; in the latter, on septicæmia, or secondary blood-poisoning; or both these causes may be present together.

6. The cases in which this rash appears are often severe; but its development is important rather from a diagnostic than from a prognostic point of view.

7. Hence, no special line of treatment is required beyond that already employed for the safe conduct of the patient through the fever.

The erythematous rash here alluded to is, without doubt, the *Erythema punctatum* of Dr. M'Call Anderson,[a] the *Erythema scarlatiniforme* of Hardy, the *Roséole scarlatiniforme* of Basin, the *Symptomatic Erythema* of Dr. James Nevins Hyde,[b] the *Erythema scarlatiniforme* of Dr. H. Radcliffe Crocker,[c] the *Diffuse Erythema* of Liebermeister,[d] the *Scarlet Rash* of Charles Murchison.[e]

On April 13, 1883, Dr. Thomas Whipham read a paper before the Clinical Society of London, entitled "Two Cases of Enteric Fever accompanied by an Erythematous Eruption resembling that of Scarlatina."[f]

The most important point about Dr. Whipham's two cases is that they both proved fatal, and that an opportunity was afforded in each instance of verifying the diagnosis of enteric fever by a *post-mortem* examination.

According to an abstract from the pen of Dr. Robert Saundby

[a] *A Treatise on Diseases of the Skin.* London: Charles Griffin & Co. 1887. Page 89.

[b] *A Practical Treatise on Diseases of the Skin.* Second Edition. London: J. & A. Churchill. 1888. Page 154.

[c] *Loc. cit.* Page 58.

[d] Von Ziemssen's *Cyclopædia of the Pract. of Med.* 1875. Vol. I., p. 183.

[e] *The Continued Fevers of Great Britain.* Third Edition. 1884. Page 516.

[f] *Transactions of the Clinical Society of London.* Vol. XVI., page 150.

in the *London Medical Record* of December 15, 1878, MM. Raymond and Nélaton reported in *Le Progrès Médical* of October 19 of the same year, the histories of two cases of typhoid fever in the wards of M. Moutard-Martin, at the Hôpital Beaujon, which were accompanied by a very singular eruption, both in its aspect, its course, and the time of its appearance. MM. Raymond and Nélaton remark that analogous rashes have been recorded, in particular by Griesinger, who says:—"In rare cases we observe, at the same time as the roseola [*i.e.*, the rose-spot eruption], a rash *like* urticaria." To their two cases MM. Raymond and Nélaton add a third noticed by M. Moutard-Martin, in which a rash resembling that caused sometimes by copaiba, and which he called first "febrile urticaria," but afterwards "papular erythema," supervened on the sixth day of typhoid fever. The rash gradually lost its bright colour, and slowly disappeared. The hands and feet desquamated in large patches; the rest of the body showed only branny desquamation. As to the cases observed by MM. Raymond and Nélaton, they believe that they were **not scarlatina**, for the following reasons:—1. The form of the eruption, and its predominance in parts unusual in that disease. 2. The absence of prodromata, and of the concomitant scarlatinal symptoms, angina, state of tongue, &c. 3. The course followed by the desquamation. On the hands there were no large flakes. One of the patients showed the rash on the fifteenth day of his fever; the other showed it on the eleventh or twelfth day. Both cases were very severe, and were of an ataxo-adynamic type.

In the Proceedings of the "Société Médicale des Hôpitaux de Paris" for 1873, M. Siredey reports a case in which "towards the fifteenth day of a typhoid fever a scarlatiniform erythema appeared, which, commencing in the right cheek, spread rapidly over the face and neck, and by the following day occupied the whole surface of the body. Almost immediately, in the groins, the axilla, and on the back, the epidermis was raised by a serous exudation." M. Guyot, consulted by M. Siredey, agreed with him in rejecting the notion of its being scarlatina; nevertheless, desquamation occurred in large flakes.

It is not easy to arrive at a satisfactory conclusion as to the **ætiology** of this **accidental erythema** in enteric fever. Bearing in mind that it commonly occurs early in the disease—when a determination of blood to the skin may be presumed to have followed the initial rigors of the fever—we may, with some show of reason, attribute the development of the exanthem to **an inhibition of the vaso-motor centre**—the correlative of the stimulation of that centre by which the precedent rigors are explained—causing relaxation and overfilling of the arterioles of the skin, the impure blood further producing a passing dermatitis in the form of an erythema.

When the rash happens in the second or third week, we should remember that we are approaching **the sweating stage** of typhoid fever, and that there is again a determination of impure blood to the skin. Or we .may regard this later appearance of the rash as of **septicæmic origin**, agreeing in time with the period of "spiking temperature," which is itself so suggestive of a septicæmic tendency. As Dr. Radcliffe Crocker says: "It [erythema scarlatiniforme] is seen occasionally in septicæmic conditions, as after surgical operations, but not so frequently since antiseptic precautions have been generally adopted." And this may throw some light on M. Trélat's observations on "Scarlatina following Surgical Operations," which appeared in *Le Progrès Médical*, September 14, 1878,[a] as well as on the supposed origin of scarlatina from the decomposition of slaughter-house refuse, which was suggested many years ago by Dr. Alfred Carpenter,[b] of Croydon.

In the *British Medical Journal* for December 7, 1878, there is a very interesting note on "Septicæmic Eruptions." In 1868, M. Verneuil proved the existence of several different cutaneous eruptions as taking place during purulent infection. About the same time, Dr. Braidwood was giving special attention to the study of one of those peculiar forms of pyæmic eruptions which bear a very close resemblance to the erythema observed in scarlatina. M. Guéniot tried to prove, in accordance with the well-known fact

[a] See *Lond. Med. Record*, Oct. 15, 1878. Page 417. [See page 157].

[b] *The Lancet*, January 26, 1871, page 110; and *Manual of Public Health for Ireland*, 1875, page 169.

that women who have been recently confined are often subject to cutaneous eruptions, that scarlatiniform erythemata had been observed, the so-called puerperal *scarlatinoïde*. In addition to the above-mentioned two classes of eruptions, M. Claudien Aulnas adds a third, which has also been observed in the course of purulent internal diseases. Here we have sufficient evidence of the septicæmic or pyæmic origin of erythema scarlatiniforme, whether in enteric fever or in other zymotic diseases or blood-poisonings.

Of all these adventitious appearances connected with the skin in enteric fever, the erythematous rashes are the most important, because they may so closely simulate scarlatina. The miliary eruption, if coupled with the rheumatoid pains which are not infrequently present in typhoid, might cause a wrong diagnosis of rheumatic fever to be made; while nettle-rash (*Germ.* Nesselsucht), if associated with catarrh, might be taken for measles. But, serious as we should consider these errors of diagnosis to be, a much more disastrous mistake would be the confounding of erythema with scarlatina, or, conversely, the overlooking of scarlatina in the belief that the rash was simply erythema.

Desquamation occurs in fine branny scales. The skin feels rough and, perhaps, dry, while the process is taking place. The hair often falls out, and atrophic markings may appear on the nails.

III. **The Circulation.**—The pulse varies much. At first it does not exceed 90 or 100 beats in a minute; sometimes its rate is below the normal standard (that is, 72 in an adult male, 84 in an adult female). The prognosis is bad if the pulse-rate in an adult persistently exceeds 120. Another unfavourable sign is failure in volume when the patient's arm is elevated. The pulse is often **dicrotic**[a] in enteric fever (Germ. *doppelschlägiger Puls*; Fr. *Pouls dicrote, p. rebondissant*)—that is, the pulse yields a sphygmographic tracing, or conveys to the fingers a sensation of a double beat for each systole of the left ventricle. Sometimes—as in a case under my care at the time of writing—dicrotism is visible to the eye in the radial, or even the ulnar, artery. Such a dicrotic pulse indicates relaxation of the arterial walls and low blood-tension in the arteries. Repro-

[a] Gk. δίκροτος, *double-beating* From δίς, *twice*, κροτέω, *to strike.*

duction of sphygmograms of dicrotism will be found in Murchison's work (Third Edition, page 140, third tracing, Fig. 6), but especially in Liebermeister's monograph on "Typhoid Fever" in von Ziemssen's "Cyclopædia of the Practice of Medicine."[a] No sudden fall in the pulse-rate occurs towards convalescence (as in typhus), nor is the heart weakened to the same extent, or so frequently, in enteric fever as in typhus. In this connection the younger age of the average typhoid patient is to be remembered.

IV. **The Respiratory System.**—Little need be said on this point. The rate of breathing as a rule varies with that of the pulse, unless the latter is very slow. There may be "nervous respiration," and the breath is offensive in the "typhoid" or "ataxic stage." The occurrence of true pneumonia as a not infrequent complication will be alluded to further on.

V. **The Digestive System.**—The condition of the *tongue* has been already sufficiently described (see page 365). **Nausea** and **Vomiting** are common symptoms—hence the name "Bilious Fever" which is sometimes used. Vomiting at the outset of the attack is not of evil import, but later on—in the third or fourth week—it is one of the **earliest symptoms of peritonitis.** At the same time, it is right to mention that I have repeatedly known obstinate vomiting at all stages of enteric fever to depend on overfeeding.

Meteorism, or **tympanites**, is very often present and in excess, principally over the colon. It is usually associated with pain, tenderness on pressure, and diarrhœa.

Gargouillement, or a sensation of *gurgling*, in the ileo-cæcal region, is often elicited on gentle pressure. It is supposed to mean regurgitation through the ileo-cæcal valve, but to my mind it is a sign of little or no importance, and an unskilful search for it may seriously injure the patient.

The bowels are sometimes even obstinately confined in enteric fever, although more or less diarrhœa is the rule. **Constipation is not a favourable** indication. In 1854, Dr. Wilks[b] found in the case of a girl, who died at the end of the third week, and whose

[a] *English Translation.* Vol. I, page 84. 1885. Figs 4 and 5.
[b] *Guy's Hospital Reports.* Third Series. Vol. I., page 319. 1855.

bowels had been confined, that the small intestines were filled with hard scybala, and that there was an ulcer underneath each. Louis, Jenner, and Hudson, all report fatal cases, which had been characterised by persistent constipation.

According to Murchison, **diarrhœa** was present in 93 out of 100 cases observed by him. The severity and danger of enteric fever are closely related to the intensity of the diarrhœa. But to arrive at a reliable opinion, it is necessary to inquire of the nurses in charge what they understand by "diarrhœa." Sometimes one profuse evacuation in the 24 hours may well be termed diarrhœa; whereas, on the contrary, a patient may be disturbed every half hour by a "needing," when only a little mucus and fragments of fæces may be passed—not sufficient to justify the application of the term "diarrhœa." The discharges have a heavy putrid and offensive smell. These are often of the colour of yellow ochre and of the consistence and appearance of pea soup. If allowed to stand, they separate into two strata—a flaky sediment containing thin fæces, the *débris* of sloughing ulcers (after the fourteenth day), undigested food, and many prismatic crystals of triple phosphate, and a supernatant yellowish or pale brown liquid, alkaline in reaction, having a specific gravity of 1015, and containing about 4 per cent. of solid matter—chiefly albumen and soluble salts, particularly chloride of sodium. Schönlein,[a] in 1835, first observed triple phosphate in the dejecta of enteric fever; but they are not peculiar to it and their presence simply means that decomposition is rapidly taking place. Other kinds of enteric stools described by Murchison are—the pultaceous, frothy, mudlike, "birdlime," bloody.

Intestinal hæmorrhage is an important symptom. Copious bleeding (over six ounces) occurred in 58 of 1,564 cases under Murchison's care—that is, in 3·77 per cent. Liebermeister's estimate of the frequency of hæmorrhage, based on the observation of 1,743 cases of enteric fever at Basle, is 7·3 per cent. Griesinger observed 32 cases of hæmorrhage in 600 enteric fever patients, or 5·3 per cent. Louis found bleeding in 5·9 per cent. of his cases. The

[a] "Ueber Crystalle im Darmcanal bei Typhus abdominalis." Müller's *Archiv.*, 1836.

accident may occur as early as the 5th or 6th day, when it is probably caused by capillary hyperæmia or by "dissolution of the blood." More commonly, hæmorrhage is not seen until after the 14th day, when it is most likely due to erosion of an artery at the ulcerated and sloughing base of one of Peyer's patches. Opinions differ as to the bearing on prognosis of intestinal hæmorrhage. Graves and Henry Kennedy considered that in certain cases it was of marked benefit. Graves[a] spoke of it as "a critical discharge of blood from the bowels" not to be unnecessarily interfered with. Yet he saw four patients in whom the occurrence of hæmorrhage from the bowels induced death—"in all the fever had a marked *gastric* character." Trousseau,[b] who originally held the view that these hæmorrhages are serious complications, attracted by the teaching of Graves—"un homme d'une aussi grande valeur, d'une aussi grande renommé"—changed his opinion and wrote:—"Pour mon compte, après m'être longtemps rangé à cet avis, je professe aujourd' hui une doctrine tout à fait opposée, à savoir, que les hémorrhagies intestinales, dans la fièvre typhoïde, loin d'avoir la gravité qu'on leur accorde, constituent le plus souvent un phénomêne de favorable augure."

On the other side, great names also can be quoted, especially those of Murchison and Liebermeister. The former authority no doubt admits that a moderate bleeding *before the twelfth day* may do good by relieving intestinal congestion; but when the bleeding occurs later in the disease and is profuse, it is a formidable symptom. The bleeding makes it probable that the ulceration has extended to the vessels beneath the transverse muscular fibres of the intestine, and such ulceration is not unlikely to go on to perforation should the patient survive the hæmorrhage. Of Liebermeister's own cases, 38·6 per cent. died, whereas the mortality among those without hæmorrhage was only 11 per cent. This author admits that his statistics are not quite as conclusive as they at first sight appear, for in some fatal cases the bleeding is in no way respon-

[a] *Clinical Lectures.* New Syd. Soc. Edition. 1884. Vol. I., pages 155 and 156.

[b] *Clinique Méd. de l'Hôtel Dieu.* Paris. 1865. Tome I., p. 225.

sible for the result. In many others, however, it apparently contributes to the production of cardiac paralysis. While, therefore, intestinal hæmorrhage must be regarded, on the whole, as affecting the prognosis unfavourably, yet each individual case must be judged on its own merits. According to Liebermeister, further, a **copious hæmorrhage early** in the disease is highly **unfavourable**, the more so as it **contraindicates** the use of one of the most valuable therapeutic agents in such cases—to wit, **cold baths**. An abundant hæmorrhage at a later period—the end of the third or beginning of the fourth week—though not without danger, may **do good** by **lowering temperature** permanently.

In his article on "Abdominal Typhus" in the *Real Encyclopädie der gesammten Heilkunde*, Dr. W. Zuelzer says that in the intestine, the bleeding sometimes fills several loops of intestine so full that the **tympanitic percussion note disappears**. From this we are often enabled **to diagnosticate latent intestinal hæmorrhage**. To the correctness of this statement I can testify from personal observation.

In passing, Liebermeister remarks that the frequency of intestinal hæmorrhage has materially **diminished**—from 8·4 per cent. to 6·2 per cent.—under the cold water treatment.

Although I am not prepared with accurate statistics on the point, I have seen many cases of profuse intestinal hæmorrhage in enteric fever, and the impression left upon my mind is that an unexpected and surprising improvement in the patient's state sometimes follows *one* copious hæmorrhage, whereas repeated hæmorrhages of less intensity nearly always terminate in death.

The symptoms of intestinal hæmorrhage are—sudden or rapidly increasing prostration, pallor, quick failing pulse, and a marked, even if temporary, fall of temperature.

The **spleen** is very constantly enlarged in enteric fever, particularly in patients under 30 years of age, and towards the close of the second week of the fever. In children, Barthez and Rilliet[a] met with splenic enlargement in 28 out of 105 cases. Dr. J. C. Wilson, the writer of the very exhaustive article on "Enteric or Typhoid

[a] *Traité des maladies des Enfans.* Paris. 1853. Deuxième Édition.

Fever" in Keating's "Cyclopædia of the Diseases of Children,"[a] says that the **enlargement of the spleen** is so frequent and so considerable in this disease that it has acquired a certain degree of **diagnostic importance.**

The **spleen enlarges,** as a rule, **downwards** and **forwards.** So long as the organ still lies inside the margin of the ribs, the enlargement is to be gauged only by **percussion.** When the spleen passes below the ribs and invades the epigastric, umbilical, and left lumbar regions, **palpation** gives accurate information, in addition to percussion, as to the size, shape, and consistence of the enlarged viscus. Weil's diagram of splenic enlargement shows very well the form and direction which this pathological condition takes. Guttmann[b] points out that, as enlargement of the spleen is invariably attended by increase in the thickness (the antero-posterior diameter) of the organ, the **intensity of the dulness** is always **much greater** than over the normal spleen—the percussion sound of a large splenic tumour is as absolutely dull as that heard over the liver, while the sense of resistance experienced in the percussing finger **is augmented** to a very unusual degree. It is to be remembered, however, that extreme tympanites may displace the spleen upwards or outwards.

V. The **Urinary System.**—During the first week or ten days the urine is diminished in quantity, is dark coloured, acid, of high density (Sp. grav. 1025 to 1030), and contains an excess of urea to the amount of one-fifth (Edmund A. Parkes), and of uric acid also. According to Brattler, quoted by Parkes, there is a close correspondence between the amount of urea and the temperature—the greater the amount of urea the higher the temperature. This view is disputed (see page 278).

After the second week, the quantity of urine increases greatly until convalescent, when 80 or 90 ounces may be passed daily. In this second period the urine is pale, feebly acid or alkaline, and of low density—perhaps only 1005 or 1003 in convalescence (Murchison).

[a] Philadelphia: J. B. Lippincott Company. Vol I., page 470. 1889.

[b] *Handbook of Physical Diagnosis.* New Syd. Soc., London. 1879. Vol. LXXXIV., page 354.

All through the fever chlorides are greatly diminished in quantity, for these reasons—there is less salt taken with food, much escapes by the skin in perspiration, there is a good deal in the motions from the bowels, and lastly, much is retained in the system until convalescence, when it is discharged in abundance.

Albumen is less frequently present in the urine than in typhus, and when albuminuria occurs, it does so *later* in the disease.

VI. The **Nervous System.**—Neuralgic and rheumatoid pains are common in the early stages of enteric fever. When these pains are articular, the case may at first simulate acute rheumatism. Vertigo and headache are common symptoms—the latter is most severe during the first week. It is not usually very intense, but it may be so. In the Autumn of 1890 I attended a gentleman who suffered agony from headache in the first week of his illness, which ultimately proved fatal from the supervention of the ataxic state. The headache is generally referred to the forehead.

Liebermeister[a] distinguishes **four grades of nervous disturbance**, which usually occur in succession in severe cases of enteric fever.

1. In the earlier part of the first week the nervous symptoms are—malaise, restlessness, headache, unfitness for mental occupation, unquiet sleep with dreams.

2. In the latter part of the first and the beginning of the second week, the patient is somewhat apathetic, his sensations are blunted, and his memory fails. When half awake and not thinking of himself, he becomes delirious. At other times his mind is clear.

3. In the course of the second week, through the third, and into the fourth, there are—muttering delirium, a drowsy condition, or even sound sleep, from which the patient can be aroused for a time, and even incited to exercise his volition. [*Coma vigil* of Jenner—*Κῶμα ἄγρυπνον* of Hippocrates and Galen.] Sometimes the delirium is more violent, with great restlessness, and paroxysms of intense excitement, approaching mania.

4. In the worst cases, usually in the third week—rarely earlier, not infrequently later—there is constant loss of consciousness, from which the patients can no longer be aroused. They lie without

[a] Von Ziemssen's *Cyclopædia of the Pract. of Med.* Vol. I., page 87. 1875.

any evidence of mental activity, do not re-act when spoken to or shaken. [This state is the *Lethargus* (s.c. *morbus*) of the ancients—ληθαργία, drowsiness, lethargy—Galen]. Hiccough is often present in this condition of profound nervous prostration.

The disturbances of this, the highest, degree correspond to an almost complete abolition of the functions of the cerebrum, and when this paralysis extends to the medulla oblongata it leads to death.

In a paper on "Reflex Actions, Knee-jerks, and Muscular Irritability in Typhoid Fever, Phthisis, and other Continuous Fevers," Dr. Angel Money[a] points out that in all cases of enteric fever the knee-jerk is exaggerated, so much so that its equivalent (contraction of the *quadriceps extensor femoris*) may often be produced by drawing down the patella with the forefinger, and then percussing the straining forefinger—a method which Dr. Money first learned from Dr. Gowers. He also finds that muscular irritability is greatly increased in this fever, and this increase shows itself in several ways. When a muscle is tapped by immediate percussion with the finger or stethoscope a contraction of the whole muscle is brought about, and at the same time a series of fibrillary contractions occur in the belly of the muscle. These conditions are best observed in a voluminous muscle like the calf. The facial muscles are not so irritable, though occasionally percussion over or about the malar process may cause a very obvious contraction of the *orbicularis palpebrarum*. The tongue shows fibrillary tremors rather earlier than the trunk and limb muscles. When the knee-jerks are greatly exaggerated, as they sometimes are in enteric fever, fibrillary contractions of the muscles occur, and spontaneous contractions of the whole, or nearly the whole, of the muscles are not infrequently present. These are, of course, well known under the name of "subsultus tendinum." When this marked degree of irritability exists, "ankle clonus" is usually present and is easily elicited.

VII. The **Organs of Special Sense.**

1. The **Eye** is clear and bright, and the **pupils** are usually dilated, indicating anæmia of the eye. In great stupor and coma

[a] *Lancet.* 1885. Vol. II., page 842. November 7.

the pupils are, however, often contracted, and the eyes are hyperæmic or congested.

2. The **Ear.—Buzzing** noises are often complained of in the early stages, and **deafness** of one or both ears is a common symptom later on—towards the close of the second week and onwards. Louis observes: "La plus extrême surdité n'ajoute rien à la gravité du pronostic." Murchison does not quite agree in this opinion. Bilateral deafness may be caused by catarrh of the Eustachian tubes. The remarks already made on the deafness of typhus apply to this symptom in enteric fever also.

3. The **Nose.—Epistaxis** or **nose-bleeding** is a very common symptom, especially in children. Rilliet and Barthez met with it in one-fifth of their 107 cases. It may take place at any time in the fever, and may recur again and again so as to produce extreme anæmia or not seldom to destroy life.

4. **Cutaneous sensibility.—Hyperæsthesia** of the surface was noticed by Murchison in about 5 per cent. of his cases. It is most common in women and children, and is not generally of grave importance. It is best marked over the abdomen and lower limbs, a fact which may lead to an error in diagnosis—the tenderness on pressure due to peritonitis being mistaken for mere hyperæsthesia, or *vice versâ*.

VIII. **Emaciation** is a very marked and characteristic symptom in enteric fever. Its causes are: the prolonged illness, the special implication of the digestive system in the morbid process, and the wasting nature of the discharges. Recently (October, 1891), a notable example of marasmus after a protracted enteric fever was under my care in the wards of the Meath Hospital in the person of a little girl aged twelve years, who is almost literally only "skin and bone." The patient has since recovered.

CHAPTER XXXIX.

Relapse in Enteric Fever.

Relapse is a rare occurrence—Definition of True Relapse—Relapses not to be confounded with Recrudescences—Clinical Record of a case of Relapse—Statistics of Occurrence of Relapse—Relapse not so dangerous as the first attack—Ætiology of Relapse—Probable influence of: (1.) Constipation; (2.) Enlargement of the Spleen.

Among the many causes of renewed pyrexia, or feverishness, in the later stages of enteric fever, or in convalescence from this disease, **true relapse** necessarily occupies a foremost place. And this arises, not so much from any increased danger to the patient's life—which is theoretical rather than founded on fact—as from the comparative infrequency of the occurrence of true relapse in this form of continued fever.

By "**true relapse**" I understand a **second attack**, in which the characteristic phenomena of enteric fever present themselves in sufficient number **to establish the diagnosis of the disease**—for example, enlargement of the spleen, abdominal tenderness, ochrey diarrhœa, and rose spots; or epistaxis, feverishness with evening exacerbations, abdominal tenderness and tympanites; or any other grouping of the symptoms of this fever met with in practice; the fact being admitted that a perfectly typical case of primary enteric fever, showing *all* the characters of the disease, does not often come under observation, even in the wards of a large epidemic hospital. "By a relapse of enteric fever," writes Murchison,[a] "is understood a second evolution of the specific febrile process, after convalescence from the first attack is fairly established. Relapses must not be confounded with the *recrudescences*, which are common during the stage of ulceration."

[a] *The Continued Fevers of Great Britain.* Third Edition. Edited by W. Cayley, M.D., F.R.C.P. London: Longmans, Green & Co. 1884. Page 552.

I will detail the salient points in a case which falls within the limits of the foregoing definition, and which I communicated to the Royal Academy of Medicine in Ireland in 1885[a]:—

On Saturday, January 24, 1885, Mrs. Mary B., aged twenty, a domestic servant, was admitted into Cork-street Fever Hospital, under my care, at the request of the late Dr. S. M. MacSwiney, F.K.Q.C.P., who had been called to see her when she was already several days ill, and who recognised her ailment as enteric fever. She had been married not long previously, but menstruation was regular up to the time of her illness, and there was no reason to believe that she was pregnant.

When I visited the hospital on the morning of the 25th I found that Mrs. B. had complained of weakness and chilliness nine days before. Diarrhœa soon set in, for which she was treated by a respectable general practitioner, who was at the disadvantage of seeing the patient up and dressed, and so failed to recognise the true nature of the case. At last she took to bed, and was seen by Dr. MacSwiney, who advised her removal to hospital.

At my first visit, her pulse was 120, respirations 32, and temperature (10 a.m.) 103·2°. The area of splenic dulness was enlarged. There was a good deal of tympanites, and some tenderness on pressure existed in the iliac fossæ. She was passing from four to six yellowish fluid motions in the twenty-four hours. There was insomnia, and her mental state was unsatisfactory—nervous and excited. Crepitating râles were audible over the back of both lungs. Several typical rose-spots were detected on the trunk, and also some taches bleuâtres. A glycerine and laudanum poultice was applied to the abdomen. The back of the chest was drycupped. She was ordered ice, milk and arrowroot, and 6 ozs. of port wine. Chalk mixture, with chlorodyne and compound tincture of chloroform, was prescribed, as well as a full dose of Dover's powder at bedtime. Next day the chest was poulticed with linseed meal sprinkled with powdered camphor, and on the 27th 3-grain doses of quinine were given thrice a day. On the 28th turpentine

[a] *Trans. of the Acad. Med. in Ireland.* Vol. IV., page 1. 1886. See also *Dub. Journ. Med. Science.* Vol. LXXX., page 486. December, 1885.

fomentations were applied to both chest and abdomen, and on the 29th a further increase of diarrhœa required the administration of an enema containing 12 minims of tincture of opium in two ounces of mucilage of starch.

On January 30, the 15th day of the fever, the patient appeared to be going on well—P. 106, R. 26, T. 101·8° in the forenoon, rising only to 102·6° in the evening. At night some intestinal hæmorrhage occurred. Next morning a poultice of crushed ice was applied to the abdomen, and three grains of ergotin were ordered to be injected hypodermically should hæmorrhage recur. This did not happen, and the patient began to make excellent way; her pulse and respirations fell and diarrhœa ceased, giving place to obstinate constipation—a point of some moment in the light of the subsequent history of the case. Reference to the clinical chart will show that from the 16th to the 23rd days of the fever inclusive the evening exacerbations of pyrexia were very marked, the temperature being from 3° to 4° higher than in the forenoons.

On the morning of February 8 (24th day) the body was covered with a plentiful crop of sudamina, but the temperature was only 98°. The following evening it did not exceed 100·1°, and 48 hours afterwards it became permanently normal.

Mrs. B. remained in hospital until February 28, when she was sent to the Convalescent Home, Lynden, Blackrock, Co. Dublin, this being the 18th day of complete apyrexia and the 44th day from the commencement of her illness. During the last three weeks of her stay in hospital the bowels were obstinate, and had to be kept in order by the almost periodic use of simple enemata or the occasional administration of castor-oil in small and repeated doses.

During the first few days of her stay at the Convalescent Home, Mrs. B. made satisfactory progress. Her stomach then became irritable; she lost her appetite, began to cough, and felt hot and cold by turns. She came into Dublin, and in the course of a few days called to see me. I found her tongue furred and her pulse quick. There were few, if any, physical signs of chest affection, but she looked pale and thin, like one who would readily run into

acute consumption. Acting under my advice, she went home to bed, and I visited her on Thursday, March 19, the twelfth day from the appearance of symptoms of her second illness. Her pulse at 5 p.m. was 108, and her temperature was 103°.

Next day she was again admitted to Cork-street Hospital, under my care. The morning temperature was only 99·1°, but in the evening the thermometer marked 102·8° in the axilla. There was some increased splenic dulness on percussion, and a few rose-spots were found on the chest and abdomen. On this and the three following days there was a tendency to diarrhœa—two or three yellow soft motions occurring each day. Afterwards the bowels acted regularly once a day. The course of this second fever is sufficiently well shown on the temperature chart (Plate VIII., Fig. 4). It will be seen that there was a well-marked remittent tendency in the temperature range—the morning readings were moderate or low, while the evening ones were between 102° and 103°. On the 20th day, although the pulse remained steady at 88, the temperature rose from 98·0° in the forenoon to 102·6° in the evening. A somewhat similar rise next day was succeeded by a rapid defervescence, which was completed in three days, and so more completely resembled a crisis than is usual in enteric fever. At this time, also, the pulse fell below normal, beating from 66 to 76 times a minute.

It is only necessary to add that Mrs. B. made a quick and satisfactory recovery; all chest symptoms subsided, her appetite returned, and she speedily regained her wonted health and strength. On the 31st of March (the 24th day of her second illness) she was transferred to the care of my friend, Dr. J. M. Redmond, F.R.C.P.I., who then succeeded me as Physician to Cork-street (Fever) Hospital, and on the 15th of April she left hospital, finally convalescent. During the last ten days of her stay in hospital there was again some little trouble from constipation.

The foregoing case suggests some interesting points for discussion. In the first place, there seems little reason to doubt that it affords an example of true relapse in enteric fever, as distinguished from a secondary symptomatic pyrexia occurring in convalescence from that disease, and due to one or other of the recognised causes

of such a sequela—septicæmia, enteritis, pneumonia, acute tuberculosis, and so on. In the case before us, there was a long period of **apyrexia**—twenty-four days—between the two fevers, the second of which, no less than the first, presented a grouping of symptoms sufficient to warrant the diagnosis of enteric fever. "It is on the presence of the eruption, and on the absence of any local inflammation to account for the pyrexia, that the diagnosis of a true relapse must be based."—(Murchison).

This being so, the case demands attention because of the **rarity of relapse** even in this form of continued fever. Murchison[a] states that, during seven years (1862–68), relapses were observed in 80 of 2,591 cases in the London Fever Hospital—that is, in 3 per cent. Griesinger noted them in 6 per cent. of 463 cases at Zurich; Human in 8 per cent. of 548 cases at Leipzig; and Maclagan in 13 (10 per cent.) of 128 cases at Dundee.

Relapse occurred in 21 cases out of 129 observed by Dr. F. C. Shattuck, of Harvard University (16·28 per cent). In one case there was a second relapse. The duration of the second attack varied from 11 to 29 days, the average being 19 days. There was only one death. Treatment was expectant.

Jaccoud considers that relapses are much more frequent in enteric fever than is usually believed. He has seen more than 60 cases—between 9 and 10 per cent. of his personal statistics in the wards of La Pitié and in private practice.

Eichhorst collected statistics of six hundred and sixty-six cases of enteric fever treated at Zurich during three years. He found that a second attack occurred in 4·2 per cent. (28 persons). Men are more liable to relapse than women. The severity and fatality of the relapse equalled that of the first attack.

As compared with typhus, in which true relapse is one of the rarest events in clinical observation, these are all relatively high percentages. Out of 18,268 cases of typhus reported at the London Fever Hospital during 23 years, there is *only one instance* of a true relapse, although in several instances a genuine has been preceded by an abortive attack.[b]

[a] Murchison. *Loc. cit.*, page 552. [b] Murchison. *Loc. cit.*, page 189.

Again, it is worth noting that in the case I have just detailed the **relapse ran a milder** and **slightly shorter course than the preliminary attack.** The first fever terminated on the twenty-seventh day. Then came an apyrexial period of 24 days—from the 11th of February to the 7th of March—and this was lastly followed by a fever which ran a twenty-four days' course. All this is in accordance with clinical experience. Thus, Murchison says that the duration of the second attack is usually, but not necessarily, shorter and milder than that of the first.[a] Of 24 cases collected from various sources by Michel, the mean duration of the first attack was 27 days; of the intermission, 11 days (shortest 2 and longest 31 days); and of the relapse—shortest, 16 days; longest, 30. In 53 cases which came under Murchison's own observation, and which he has tabulated, the average duration was—of the first attack, 27·0 days; of the intermission, 11·76 days; and of the relapse, 16·4 days. It is noteworthy that of these 53 patients only 5 died in the relapse, the mortality being at the rate of 9·4 per cent. compared with 17·26 per cent. in primary attacks in 5,911 cases admitted into the London Fever Hospital. From this it would appear that a relapse is only about half as dangerous to life as a first attack.

The last point to be considered is the **probable ætiology** of relapse in enteric fever. In 1846 Hamernjk put forward the view that relapse was due to **reabsorption of the "typhous material"** thrown off by the patient's own bowel.[b] Dr. T. J. Maclagan adopts this view, believing that the surviving healthy glands become inoculated by the sloughs thrown off from those first affected. In accordance with this theory, Maclagan further holds that relapses are met with only when there has been constipation in convalescence. This condition was certainly present in Mrs. B.'s case, and we are justified in assuming that the non-elimination of the fever poison, owing to constipation, was at all events one factor, and that an important one, in the causation of the second attack.

[a] Murchison. *Loc. cit.*, page 553.

[b] Murchison. *Loc. cit.*, page 555; and Hamernjk, Prag. Vierteljahrssch. 1846. X. I. (*Zur Pathologie und Diagnose des Typhus*).

It may be objected that to adopt this view in its entirety is to pledge ourselves to the exploded theory of the local origin of the phenomena of enteric fever—is to revert to Broussais' doctrine, according to which the pyrexia of the disease is the result of a local inflammation of the intestinal glands. But this is not stated. What we hold is that, as a result of constipation, the fever poison finds no exit from the system *viâ* the bowels, it is then absorbed by the glands which have hitherto escaped the characteristic secondary inflammation of the disease, and passing into the blood once more sets up an essential, not a symptomatic, fever.

One other theory of relapse may be drawn from the analogy of true relapsing, or spirillum, fever. In the third volume of the *Irish Hospital Gazette* (April 1, 1875, page 105), Dr. Gerald F. Yeo quotes from the *Centralblatt für klinische Medicin* an observation of Dr. M. Laptachinski, that with each attack of pyrexia in spirillum fever a **discharge of the spleen contents into the blood** takes place. From observation of the epidemic of relapsing fever of 1847–49 in Ireland, Dr. Alfred Hudson, then of Navan, Co. Meath, was led to believe in the frequent co-existence of the poison of endemic typhoid with that of epidemic relapsing fever—the one disease replacing the other in the same patient.

He also noticed the influence upon the blood of the reabsorption into the circulation of a quantity of depraved blood, laid up, as it were, in the congested spleen, and commingling with the circulating mass after crisis, in the purifying effect of which it had not shared. He adds:—"I shall be mistaken if future observations do not prove this to be an important element, not only in the production of relapse, *but also in determining the enteric lesions*, which occur during the second and third attacks, in this form of fever."[b] Remembering how constantly the spleen is enlarged and congested in enteric fever, we may perhaps apply to this disease also the ingenious pathological hypothesis which was first advanced, as we have seen, by Dr. Hudson, in explanation of the relapses in spirillum fever.

Towards the close of the year 1886, I met with another instance

[b] *Lectures on the Study of Fever.* Second Edition. 1868. Pages 147 and 287.

of true relapse in enteric fever, in the person of a member of the medical profession—a gentleman aged thirty-two years. The temperature in this case is given in Fig. 5 of Plate VIII. The primary attack lasted exactly 28 days, and was followed by a week of apyrexia, during which the bowels were confined. On the thirty-fifth day rigors occurred in the afternoon, and temperature rose to 104° within 30 hours. This new fever lasted 14 days, and was accompanied by a fresh but scanty crop of rose-spots from the seventh day onwards.

CHAPTER XL.

Enteric Fever (*continued*).

Temperature—Complications and Sequelæ.

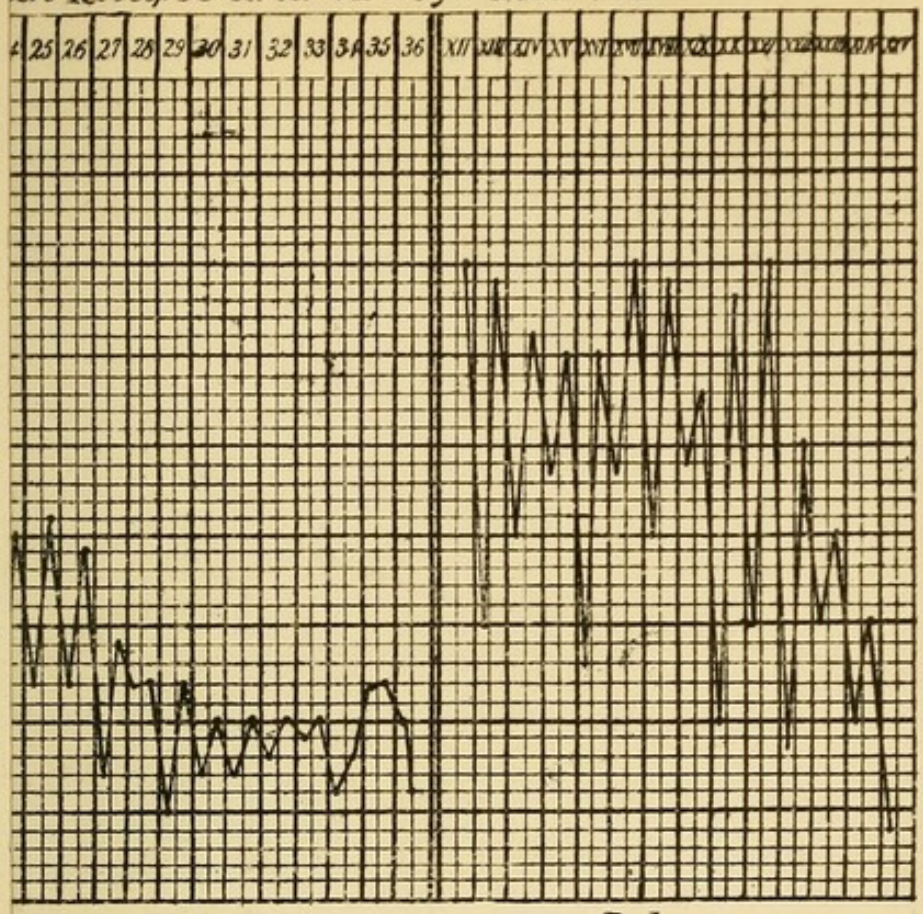
Temperature important in diagnosis and prognosis.—Initial or Prodromal Stage : remittent type.—Fastigium : continuous type.—Amphibolic Stage.—Defervescence : remittent type with "spiking."—Test of complete recovery.—Remittent type of pyrexia in young children —Moderate pyrexia in old patients.—Apyrexial or Afebrile Enteric Fever. Complications and Sequelæ: of respiratory tract, the circulation, the nervous system, organs of special sense, digestive tract, the urinary organs, female organs of generation, tissues, integuments, and bones; marasmus, sudden death.—Coexistence of other specific diseases.

Temperature.—The most comprehensive account of the behaviour of the temperature in enteric fever is that given by Wunderlich, from whose monograph on "Temperature in Diseases" I have so often quoted in this book. He remarks that the presence of this disease can be decided by the temperature only when both morning and evening temperatures have been taken for several days. In the beginning of the fever about three days' observations are required for the purpose of **diagnosis**; but in the fastigium and in convalescence from four to six days' record is requisite.

Thermometrical observations have also an important bearing on **prognosis** in this disease. In the second week the severity of a case is commonly indicated by the temperature alone. The thermometer further enables us to recognise irregularities in the course of the fever, the advent of complications, a recrudescence or a relapse, a tendency to death, the effects of treatment, and the transition to convalescence.

Enteric fever is characterised by a fever-movement, which lasts for at least three weeks, unless in exceptional cases, and in those which prove fatal at an early stage. The **maximal temperature**, as a rule, is **not less than** 103·3° F., and is more commonly between

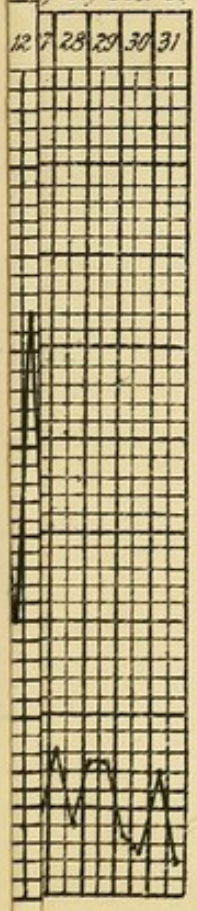
ith Relapse after 24 days interval.



r. Relapse.

Fig. 9 – Enteric Fever, terminating by Crisis.

2½ years.



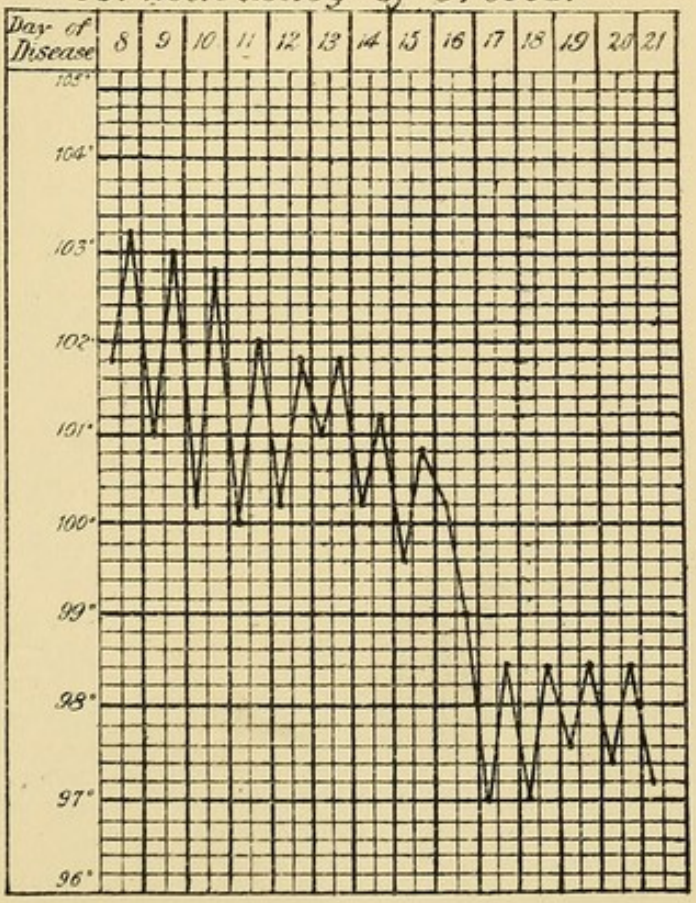


Plate VIII.

CHARTS OF TEMPERATURE RANGES IN ENTERIC FEVER.

Fig 1.—Normal Enteric Fever.

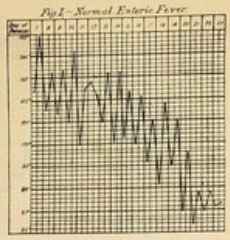


Fig 2.—Normal Enteric Fever.

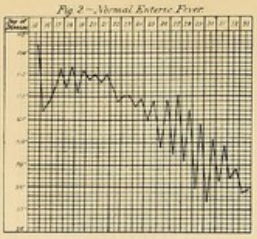


Fig 3.—Enteric Fever, with Recrudescence.

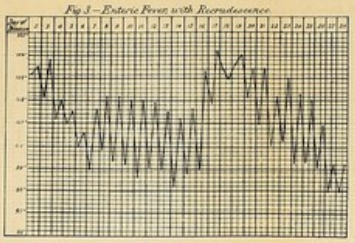
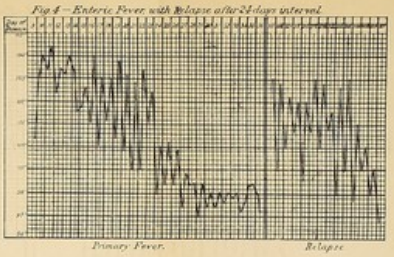
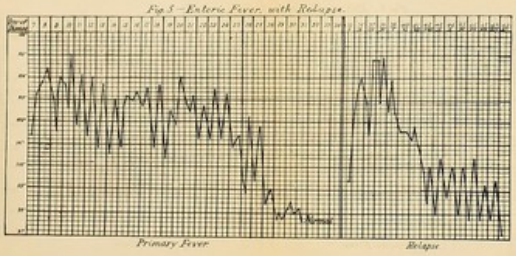


Fig 4.—Enteric Fever, with Relapse after 24 days interval.



Primary Fever. *Relapse.*

Fig 5.—Enteric Fever, with Relapse.



Primary Fever. *Relapse.*

Fig 6.—Protracted Enteric Fever.

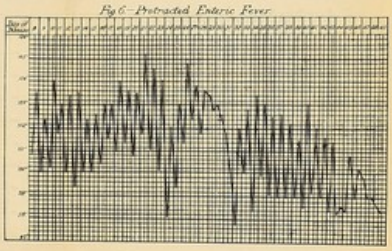


Fig 7.—Ataxic Enteric Fever.

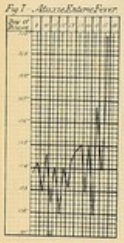


Fig 8.—Enteric Fever in a child aged 4½ years.

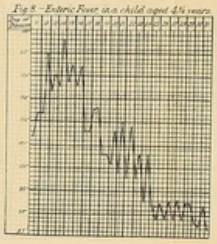
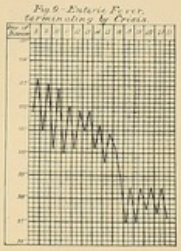


Fig 9.—Enteric Fever, terminating by Crisis.



104° and 105·8°. It may rise to hyperpyretic degrees, but not easily above 110°. In one of Dr. Reuben J. Harvey's cases—that of a young woman, aged twenty, who died in Cork-street Hospital on March 17, 1880, apparently on the eighteenth day of ataxic typhoid fever—the temperature in the axilla a few minutes before death registered 109·8°. The chart is given in Plate VIII., Fig. 7. Except in fatal cases, however, it is rare to meet with temperatures above 106·7°.

At the beginning the temperature rises in **a zig-zag fashion**, in such a way that during the four days or so occupied by the **pyrogenetic** or **initial stage** the thermometer rises about 2° or 2·5° Fahr., from morning to evening, falling again from evening to morning only 1°, or at most 1·5° Fahr., with the result that about the fourth evening a reading of 104° is reached or slightly exceeded.

From this time onward for seven or eight days, the course of the temperature is tolerably uniform—slight morning remissions being followed by moderate evening exacerbations in this stage, the fastigium of the fever.

It will be observed that, so far, we have had to deal with a **remittent type** of fever during the pyrogenetic stage, and a **continuous** or **sub-continuous type** during the fastigium.

In the middle of the second week, between the 9th and 12th days, slight and severe cases show a marked difference.

In *slight* cases the fastigium then shows a tendency to terminate—its short daily curves are gradually converted into the steep daily curves of the period of convalescence. The temperature ranges begin to "**spike**," and the fever-type is once more remittent. In such cases the temperature approaches to normal in a zig-zag fashion in the course of from 6 to 10 days: defervescence is gradual—by lysis. Recollect, however, that even in these mild cases, at any moment the even tenor of the temperature range may be interrupted by the supervention of some complication.

We may expect a severe course of the fever when the morning temperature is persistently above 103°, and the evening temperature touches 105°, when any irregularity of temperature occurs in the

second week, when moderation of the temperature does not occur, at least about the twelfth day. Increasing fever towards the close of the second week is always an ominous sign of coming trouble. The same remark applies to a rising temperature in the third week, or to a temperature which is higher in the third than it was in the second week. Complications commonly occur about this time. They generally raise the temperature, and abolish or mask the morning remissions. **Profuse hæmorrhage** or **perforation** of the intestine may cause **sudden** and **considerable fall of temperature.**

In these severe cases, a period of changing fortunes—a period of uncertainty—often intervenes. This is called the **amphibolic stage**, and has already been explained (see page 366). It sometimes lasts only three or four days; generally a week, or ten days, sometimes even longer.

In fatal cases, in the death agony, or at the moment of death, the temperature may be subnormal, but more usually it is high or excessive—107·6° to 110°.

Sometimes the amphibolic stage terminates in a protracted fever (*lentescirende Process*—Wunderlich), of quite indefinite duration and depending upon continued ulceration in the bowels, or bronchorrhœa, or marasmus.

Although defervescence generally takes place gradually in enteric fever—that is, by lysis, yet it may occur suddenly—that is, by crisis. Dr. H. T. Bewley, F.R.C.P.I., has kindly permitted me to publish a clinical chart of such a case. (See Plate VIII., Fig. 9.)

Complete recovery is to be admitted only when the temperature shows complete freedom from fever in the evenings—when, in fact, it has been normal for at least two successive evenings. For a short time the thermometer may fall below normal, but trivial causes—such as change of diet, excitement, and so on—may cause it to rise afresh from time to time in convalescence.

Young children show a very remittent type of temperature in enteric fever. In middle-aged and elderly subjects, the intensity of the fever as marked by the thermometer is less pronounced than it is in children and adolescents.

Dr. Cayley supplements Murchison's account of the behaviour of

the temperature in the disease by a paragraph on apyrexial or afebrile enteric fever. He writes:—

"Cases or even epidemics of Enteric Fever have been observed in which the temperature throughout has not risen above the normal point, and has often been subnormal, though the disease has been of a severe type, with well-marked intestinal lesions. This abnormal range of temperature seems to have always been due to the patients having previously been exposed to great hardships and insufficient food."—(Murchison's "Treatise on the Continued Fevers." 1884. Page 518.)

An epidemic of this kind is described by Dr. Struve as having occurred among the German troops besieging Paris in 1870, and another similar epidemic was recorded by Dr. O. Fräntzel in the *Zeitschrift für klin. Medicin* for 1880.

Complications and Sequelæ.

I. **The Respiratory Tract.**—1. **Bronchitis** is **rarer** in enteric fever than it is in typhus. Murchison noted its presence in 21 out of 100 cases. It may occur early or late in the fever, but most commonly both bronchitis and hypostatic consolidation of the lungs supervene in the fourth week, when these complications either kill or indefinitely prolong convalescence.

2. **Pneumonia** is **more common** than in typhus. Murchison noted it in 13 out of 100 cases, and Austin Flint (according to Bartlett[a]) in 12 out of 73 cases. It commonly occurs in the third or fourth week, but may usher in the disease. In this latter case its presence is probably an indication that the enteric fever poison has entered the system through the lungs. It is most commonly a **lobular pneumonia**, but occasionally it occurs under the form of **croupous pneumonia**. When there is an unusual tendency to pneumonia and pleurisy in enteric fever, the disease receives the name of the "**thoracic form.**"

3. **Pleuritis** is more usual than in typhus. It may terminate in empyema, or in an interlobar pleural abscess (Murchison). Pleurisy was observed in the hospital at Basle 64 times in 1,743 cases of enteric fever. Twenty-one of the 64 cases, or nearly one-third,

[a] *The Fevers of the United States.* Fourth Edition. Philadelphia. 1856.

terminated fatally. In most of the fatal cases (14) the pleurisy was dependent upon some affection of the lungs—such as hæmorrhagic infarction, gangrene, pneumonia (Liebermeister).

4. **Pulmonary Tubercle** is a more common sequel of enteric fever than of typhus. This is due, according to Murchison, to the longer duration of the fever and to the greater emaciation which accompanies it. But surely the chief causes are the **unhealthy state** of the **glandular system**, which often predisposes to enteric fever, and is certainly intensified by it; and the **infiltrated condition** of the **lung tissue** in this fever. This is apparently the view taken by Liebermeister,[a] who says—"The cases under consideration (of general miliary tuberculosis) occur in persons who have the specific poison of tubercular phthisis lying latent within them, and the fever, with its sequelæ, only serves as an exciting cause for the development of these processes." Murchison[b] states that tubercle should be feared when **hectic fever** and **bronchitis persist** after the fourth week, and Stokes taught that a **quick pulse-rate** in convalescence indicated—(1) tuberculosis in the lungs and other parts; (2) "secondary reactive inflammation in the mucous glands of the intestines;" (3) *phlegmasia dolens*. At the same time Stokes held that the bronchial affection of fever often simulated phthisis, especially in those cases where the patient lost strength, and grew pallid and emaciated.[c]

5. **Laryngitis** is a **serious** complication, more common on the Continent than in Great Britain and Ireland. Ulceration and partial destruction of the epiglottis appeared to Louis to be such characteristic secondary anatomical lesions in enteric fever that he said—"Si on venait à les observer chez un sujet qui aurait succombé à une maladie aiguë, elles annonceraient d'une manière presque certaine, et sans aller plus loin, que l'affection est une fièvre typhoïde."[d] These laryngeal ulcerations are at present regarded as secondary changes, depending on a circumscribed

[a] Von Ziemssen's *Cyclop. of the Pract. of Med.* Vol. I., page 173.
[b] *Lectures on Fever.* 1874. Pages 188, 189.
[c] *Loc. cit.* Page 135.
[d] *Recherches sur la fièvre typhoïde.* Paris. 1841. Page 321.

"diphtheritic" infiltration of the mucous membrane. They are small, but may extend in breadth and depth. Sometimes they become confluent. They are commonly found on the posterior wall of the larynx, and may engage the posterior insertion of the vocal cords. They may also involve the epiglottis, especially on its lateral edges, when they cause much disturbance. The voice becomes hoarse and rough, there may be severe cough and difficulty of swallowing (*dysphagia*), or no marked symptoms at all may be present. As a rule, these ulcerations get well without leaving any untoward after-effects; but, on the other hand, they may lead to death by producing **perichondritis laryngea**—the **perichondritis typhosa** of Rokitansky—or œdema of the glottis.

In the latter case life is occasionally saved by tracheotomy. In the former case, should the patient survive, necrosis and exfoliation of the cartilages of the larynx, abscesses in the neck, and permanent laryngeal disease may result. Of all these accidents Trousseau gives a full account in his lectures on *Dothiénentérie.*[a]

6. **General Emphysema** and **Pneumothorax** may result from a sloughing ulcer of the larynx, or from ulceration of a small bronchial abscess, or a gangrenous cavity in the lung. Chomel,[b] Wilks,[c] W. T. Gairdner,[d] Beck,[e] and W. Cayley,[f] are our authorities on this subject.

II. **The Circulation.**

1. **Degeneration of the muscular tissue of the heart** exists in all severe cases of enteric fever, though not to the same extent as in typhus. Dr. Hayem's researches on the changes which occur in the heart in enteric fever (*Gaz. Hebdom. de Méd.*, 1874, Nos. 50, 51), would go to show that these consist not only in a granular and fatty infiltration of the muscular fibres, but also in inflammatory changes. In addition to the signs of weakened-heart given by Dr. Stokes, a careful physical examination in cases of developing

[a] *Clinique Méd. de l'Hôtel Dieu.* Paris. 1865. Tome I., pages 274, *et seq.*
[b] *Leçons de Clinique Méd.* Paris. 1834. Tome I.
[c] *Trans. Path. Soc. Lond.* Vol. IX., page 34. 1837.
[d] *Glasg. Med. Journ.* January, 1865.
[e] *Verhandl. der phys.-med. Gesellsch. in Würzburg.* 1868. Page 2.
[f] *Trans. Clin. Soc. Lond.* Vol. XVI.

heart-failure will show—(1) an enlarged area of præcordial dulness on percussion, particularly in a transverse direction across the chest and towards the right side (over-filling of the right chambers); (2) evidences of an undue mobility of the heart—when the patient changes his position from side to side, the heart "sags" over, now to one side, now to the other; and (3) failure of the radial pulse when the arm is held aloft. Coldness of the lobe of the ear is another sign of failing heart not to be despised. This heart degeneration leads to many complications in different parts of the body—for example, hæmorrhagic infarctions in the lungs, spleen, or kidneys, with resulting necrotic or gangrenous processes, bedsores from malnutrition, and so on.

2. **Pericarditis** and **Endocarditis** are rare. The former lesion is generally latent, but its physical signs reveal its presence.

3. **Hæmorrhages** from the nose (*epistaxis*) and intestines (*melæna*) are not uncommon; and **acute hæmophilia** is occasionally met with, constituting what has been called "hæmorrhagic putrid fever."

4. **Venous Thrombosis** is more common in enteric fever than in typhus. **Arterial Thrombosis**, leading to spontaneous gangrene, is very rare, even less so than in typhus.

5. **Pyæmia.**—Murchison has occasionally met with examples of this condition during convalescence.

III. **The Nervous System.**—Meningitis, mental fatuity, and mania, convulsions, muscular tremors and chorea (Nothnagel), neuralgia and hyperæsthesia (Nothnagel), are occasionally met with. Paralysis is also an occasional sequel.[a]

For a full account of the Nervous Lesions in Enteric Fever reference may be made to a monograph by H. Nothnagel, entitled: "*Die nervösen Nachkranheiten des Abdominaltyphus.*" [a]

Liebermeister draws attention to a condition of special interest, which sometimes develops at the height of the disease, and which he calls "irritation of the brain, with depression of temperature." He has seen eight or ten cases in which head symptoms, suggestive of meningitis, or well-marked melancholic or maniacal symptoms, with insensibility of the pupils to light, set in about the middle of

[a] *Deutsches Archiv. für klin. Medicin.* Band IX., page 480. 1872.

the second week. These symptoms are due, in Liebermeister's opinion, to irritation of the brain by the high temperature; and the fall of temperature—from 104° to between 97° and 102° as extreme limits—which accompanies these symptoms he attributes to the heat-regulating centre being itself involved in this irritation.[a]

IV. **Organs of Special Sense.**—**Otorrhœa** not infrequently occurs, particularly in children. When the internal ear is involved, meningitis may result, as already explained. **Deafness** may follow enteric fever. **Amaurosis** and **amblyopia** are mentioned as sequels by Nothnagel. The **corneæ may slough** from atrophy.

V. **Organs of Digestion.**—1. Catarrhal (Liebermeister) or diphtheritic (Murchison) **pharyngitis** may cause **dysphagia**, which may also result from dryness of the throat, muscular paralysis in severe cases, or as a purely nervous affection.

2. **Vomiting** may occur—(α) at the beginning of the fever; (β) in the middle and later periods from overfeeding or in connection with peritonitis; (γ) in convalescence, when it is often a nervous symptom, according to Trousseau,[b] and is best treated by giving solid food:—"Les accidents que nous signalons ici sont des accidents nerveux, des troubles de sécrétions; le meilleur moyen de les combattre, est d'insister au contraire sur une alimentation solide."

3. **Diarrhœa**, from an **atonic** state of the intestinal ulcers, may exhaust the patient after the fever has ceased.

4. **Dysentery** has often been known to co-exist with enteric fever (Murchison, Forget, R. D. Lyons, and Gairdner).

5. **Jaundice.**—Murchison met with three cases, in all of which death ensued, the liver being small in two which were examined, and its secreting cells loaded with oil. It is usually a **fatal** complication, but is happily **rare.**

6. **Peritonitis**, next to perforation, is the complication of enteric fever which is most to be dreaded. Its symptoms are:—Sudden collapse, excruciating pain, great tenderness on pressure, vomiting and diarrhœa. The pulse is quick and thready, the extremities are cold, and the face wears an anxious, pinched appearance.

[a] *Loc. cit.* Page 178.

[b] *Clinique Méd. de l'Hôtel Dieu.* Paris. 1865. Tome I., p. 264.

Sometimes peritonitis is latent, but the last two symptoms will generally reveal its presence even then.

The **causes** of this dangerous complication are, after Murchison—

(1). Perforation of the intestine.

(2). Extension of inflammation from the mucous to the peritoneal coat of the intestine without perforation.

(3). Softening and rupture of infarctions in the spleen.

(4). Bursting into the peritoneum of a softened mesenteric gland.

(5). Bursting into the peritoneum of an abscess in the wall of the urinary bladder, or in the ovary, or in the sheath of the rectus muscle.

(6). Ulceration of the gall-bladder, leading to perforation.

Where peritonitis results from extension of inflammation from the mucous to the peritoneal coat of the intestine without perforations, its distribution may be local, not general.

7. **Perforation of the bowel**, with escape of its contents into the peritoneum is the deadliest accident which can occur in enteric fever, It occurred in 3·04 per cent. of 1,580 cases under Murchison's care, in 2·3 per cent. of 600 cases observed by Griesinger, and in 2·74 per cent. of 73 cases observed by Austin Flint in America. From these figures we may say that one in about every 33 enteric fever patients dies from perforation—the accident occurring in the third, fourth, or fifth week, and much more commonly in men than in women. Indigestible food, distension of the ulcerated bowel with gas or fæces, vomiting, and movements on the part of the patient, are the usual determining causes of the catastrophe, which is speedily followed by death in most instances. Evidence, however, is forthcoming to show that recovery does in rare cases follow perforation of the bowel in enteric fever.

Hoelscher[a] studied the records of 2,000 deaths from enteric fever in the Munich Hospital, and found that of this number 114 deaths were caused by perforation. Not a single death from perforative peritonitis was recorded as occurring in the first two weeks of the disease.

[a] *Munchener med. Wochensch.* January, 1891. Vol. XXXVIII., page 43.

Notes of 43 cases of perforation of the intestine in 389 cases of Continued Fever—that is, in 11 per cent., are given in the *Post-mortem* Records of the Continued Fevers, contained in the third medical volume of the "Medical and Surgical History of the War of the Rebellion," prepared under the direction of the Surgeon-General of the United States Army, by Charles Smart, Major and Surgeon, U. S. A.[a] Of these cases of perforation 12 occurred in 50 cases of undoubted enteric fever—that is, in 24 per cent.; 6, in 61 cases of so-called typho-malarial fever, or in 9·8 per cent.; and 24, in 313 *mixed* cases. In addition to these 42 cases, there was the case of a private, aged 20, who succumbed to an intercurrent attack of pneumonia.

"The occurrence of perforation," writes Murchison, "is denoted by the sudden supervention of collapse, with or without rigors, but with acute pain and tenderness of the abdomen, which at the same time is tense and tympanitic. Vomiting is common, and often precedes the other symptoms for several days, and is then often accompanied by an increase of diarrhœa, with or without intestinal hæmorrhage. The decubitus is dorsal, with the legs drawn up; the temperature rises; the pulse is rapid, thready, or imperceptible; the breathing is thoracic; the countenance pale, pinched, and expressive of suffering; and there is great thirst, and often suppression of urine. Soon the prostration becomes extreme, the extremities cold, and the face covered with large drops of perspiration, and the patient gradually sinks, the mind remaining clear to the last."[b]

When recovery takes place after perforation—as it does in rare instances—it is brought about: (1) by the cessation of peristalsis; (2) by pressure of the surrounding viscera; (3) by the formation of adhesions; (4) by extrusion of mucous membrane so as to close the rent in the peritoneum; and (5) by intrusion of a portion of omentum, which becomes adherent and so stops the rent.

VI. The **Urinary Organs.**—**Nephritis** and **hæmaturia** are serious

[a] Washington: Government Printing Office. 1888. Pages 334, *et seq.* Cf. page 452, *loc cit.*

[b] *Loc. cit.* Pages 570, 571.

complications of enteric fever, and **vesical catarrh** from neglected retention of urine sometimes gives trouble in convalescence.

VII. The **Female Organs of Generation.—Profuse menstruation** may complicate enteric fever. Contrary to the opinion of Rokitansky and Niemeyer, pregnancy does not confer immunity from this fever. The same remark applies to lactation. Dr. J. Marshall Day, the Resident Medical Officer of Cork-street Fever Hospital, has furnished me with particulars of five cases, all of which have come under notice within the past few months, illustrative of this statement. A young woman, aged 22, was admitted on the second day of enteric fever, nursing an infant five weeks old; another patient, aged 25, was admitted on the eighth day, nursing an infant of three months; a third was a married woman, seven months pregnant, admitted on the twenty-first day, who did not abort; a fourth was a young woman, aged 26, six months pregnant, five days ill on admission, who did not abort; and the fifth was a married woman, ten days ill, three months pregnant. This patient aborted, but made a good recovery from fever. **Pregnancy** is a less serious complication than was formerly supposed, nor does abortion or miscarriage necessarily take place.

VIII. The **Tissues, Integuments,** and **Bones** may be the seat of the following complications:—

1. **Erysipelas,** mostly of the face, appears in an advanced stage, is sometimes associated with otorrhœa, and is often fatal.

2. **Anasarca.**—Locally from venous thrombosis; general, from weak heart in convalescence. Liebermeister gives several reasons why œdema is not present with weak heart *during* the fever.

3. **Bedsores** (gangrene from pressure) are more common in enteric fever than in typhus—extreme emaciation, long duration, and diarrhœa being the cause of this.

4. **Cancrum oris,** or **Noma,** is a rare complication in children. Murchison met with only one instance. Two cases are mentioned by Charles West.

5. **Ulceration from blisters** (Louis), or **too hot poultices.** Of this I have occasionally seen deplorable examples—one quite recently.

6. **Necrosis** is a more frequent sequel of this fever than of typhus.

7. **Periostitis** is an occasional sequela, to which Sir James Paget has drawn attention.[a]

8. **Buboes.—Parotitis** is an infrequent, but serious complication. Murchison met with 6 cases of parotid bubo, of which 5 terminated fatally. Trousseau says[b]: "Ce que les anciens auraient appelé une crise ou une métastase, je l'appelle une très funeste complication." According to Hoffmann, 16 cases of suppurative parotitis were found at Basle among about 1,600 enteric fever patients, 7 of the 16 proving fatal. Liebermeister considers that the frequency of suppurative parotitis has greatly diminished since the introduction of a systematic antipyretic treatment.

To the foregoing complications and sequelæ, Murchison adds **Marasmus**. After a severe attack, the patient remains weak and anæmic, and progressively emaciates. His appetite may, or may not, be good. His digestion is bad, and he suffers from flatulence, borborygmi, or rumbling noises in the abdomen, and occasional diarrhœa.

In fatal cases Murchison found the intestinal mucous membrane abnormally smooth, and the mesenteric glands were shrivelled. In the second volume of the *British Med. Journal* for 1871, page 547, an interesting paper on this subject by Dr. Clifford Allbutt, of Leeds, will be found. It is on "Marasmus as an Occasional Consequence of Enteric Fever."

To Murchison's list of the complications of enteric fever, Dr. Cayley adds "**Sudden Death**," sometimes during the height of the attack, but much more frequently at the commencement of convalescence, at the end of the third or in the fourth week. Various explanations of this untoward event have been suggested, such as cardiac softening, thrombosis or embolism of the pulmonary artery, "reflex spasm" (Dieulafoy), ischæmia of the brain, pneumatosis of the blood, mental emotion or muscular exertion. Dr. Cayley gives two illustrative cases. In the second of these the patient, a fat, flabby woman of thirty-five, died suddenly on the 23rd day of a

[a] *Barth. Hosp. Reports.* 1877.
[b] Trousseau. *Loc. cit.* P. 246.

mild enteric fever. In the morning she seemed to be going on well, and expressed a wish for food; at 4 50 p.m. she suddenly cried out that she was in great pain, and fell back dead. An incomplete record of the *post-mortem* appearances informs us that there was a firm laminated clot in the right ventricle, extending into the pulmonary artery; but the intense pain, expressed by an articulate cry, points to perforation as the proximate cause of death. And this presumption is strengthened on the supposition that her "wish for food"—*i.e.*, solid food, had been gratified.

Co-existence of other Specific Diseases with Enteric Fever.—Scarlatina, measles, variola, vaccina, pertussis or whooping-cough, diphtheria, erysipelas, and especially typhus, may all co-exist with typhus—notwithstanding John Hunter's dogma to the contrary.[a] Murchison had notes of 8 cases in which the eruptions of enteric fever and of scarlatina co-existed. Dr. A. P. Stewart saw a case at the Middlesex Hospital of the co-existence of small-pox, vaccina, and enteric fever. I believe that, in former days, when the essential difference between typhus and typhoid had not yet come to be recognised, it may even have been the rule for these two fevers to run their course concurrently—the enteric fever cases contracting typhus in the wards in which they were placed for treatment.

As a typical and classical example of the co-existence of typhus and enteric fever, I would instance a case presented in admirable detail by Dr. Christopher J. Nixon,[b] Physician to the Mater Misericordiæ Hospital, Dublin, to the Medical Section of the Royal Academy of Medicine in Ireland, in December, 1888. The patient died, and the intestinal lesions of enteric fever were proved to be present, while the typhus rash had been observed from the fifth day of the disease, death occurring on the twelfth day. In the discussion which followed the reading of Dr. Nixon's paper several analogous cases were mentioned—one of these had occurred in my own practice shortly before.[c]

[a] See page 28.

[b] *Trans. Royal Academy of Med. in Ireland.* Vol. VII., page 22. 1889. And *Dubl. Journ. of Med. Science.* Vol. LXXXVIII., page 97. August, 1889.

[c] *Dubl. Journ. of Med. Science.* Vol. LXXXVII., page 248. March, 1889.

CHAPTER XLI.

Varieties of Enteric Fever.

"Typho-malarial Fever"—Varieties of Enteric Fever: Abortive form (*fièvre muqueuse*), latent form (*Typhus ambulatorius*), "gastric," or "bilious fever," spleno-typhoid, acute or inflammatory form, infantile remittent fever ("worm fever," or "gastric fever"), senile enteric fever, afebrile or apyrexial form.

Many varieties of Enteric Fever have been described by systematic writers—for example: The *adynamic* or *low nervous* form, the *ataxic* form, the *abdominal* form, the *thoracic* form, the *hæmorrhagic* form, and the *ague-like* form. This last is seen chiefly in persons who have been exposed to malarial poisoning, and in whom the poisons of the two diseases may be supposed to co-exist. This form received the name of **Typho-malarial Fever** from Dr. Woodward, and the Royal College of Physicians of London, following the opinion expressed at the International Medical Congress at Philadelphia in 1876, places the name as a sub-division of enteric fever, and describes the disease as a combination of malarial and enteric fevers, as, in other words, a compound fever resulting from the simultaneous action of two distinct poisons. In the *Lancet* for 1887, Dr. J. Edward Squire protested against these views, and used the term "typho-malarial" to designate a malarial fever which assumed an adynamic type, such as is presented in enteric fever—the form he had observed among the troops around Suakim during the campaign of 1886.

The correct view, no doubt, is that "typho-malarial fever" is really enteric fever showing itself in an individual already the victim of malarial poisoning, perhaps months or years before. It is surely a matter of every-day clinical experience that malaria asserts its presence whenever its victim falls sick of an acute disease.

The varieties of enteric fever which call for especial notice are the following:—

1. The **abortive form**, in which the fever does not run its regular course, the intestinal lesions undergoing resolution instead of

advancing to ulceration. This is the **fièvre muqueuse** of French writers. The attack begins like one of ordinary enteric fever, but about the 10th or 12th day an improvement is observed, and the fever dies out in the early part of the third week.

2. A more dangerous, because more insidious, variety is that to which the name **latent enteric fever** is applied. It was described by Dr. Hewett, of London, in 1826, and also by Louis, Chomel, and other writers. A patient is feverish and complains of lassitude and weakness, or is thought merely to be suffering from a "feverish cold," or from a "bilious attack" or a "gastric fever." His illness is sometimes spoken of as "simple continued fever." Suddenly, under such circumstances, he becomes delirious or maniacal, or profuse intestinal hæmorrhage sets in, or symptoms of perforation appear and the patient dies. Cases of this kind are called in Germany "**Typhus ambulatorius,**" because the victim may pass through his illness on his feet, until the final catastrophe comes.

3. The so-called "**Gastric**" or "**Bilious Fever**" is really enteric fever in a rudimentary or aberrant form. Of this kind was one of the series of four cases of illness due to the use of impure drinking water in a Training College in Dublin, which came under my notice some years ago, and to which I have already referred (see page 361).

4. Contrasted with the last-named variety, is the so-called "**Spleno-typhoid.**" At the Tenth International Medical Congress at Berlin in 1890, Eiselt,[a] of Prague, made a communication on what he described as "*Die lienale Form des Ileotyphus*"—"the splenic form of Enteric Fever," in which the spleen bears the brunt of the infection while intestinal complications are wanting. The spleen is voluminous, but spirilla are not found in the blood, and that the cases are really examples of enteric fever is demonstrated by the origin of the attack and by intestinal ulceration found in fatal cases.

In the *Proceedings of the Pathological Society of Dublin* for 1882 (Vol. IX., New Series, page 26), will be found an account of a case

[a] *Verhandlungen des X. Internat. Med. Congresses.* Berlin. Band II. Abtheilung V. Innere Medicin. 210.

of Spleno-typhoid which occurred in my practice in the autumn of 1880. Further allusion will be made to this case at page 422.

5. In certain instances the fever is very **acute**, and death may occur in the first, or early in the second week, before ulceration has commenced in the bowel. Trousseau[a] describes this variety as **la forme inflammatoire**, and Murchison gives several examples of it in his work on Fevers. Some years ago I witnessed a case of this kind in the Epidemic Wards of the Meath Hospital.

6. **Infantile Remittent Fever**, often called "Worm Fever" or "Gastric Fever," is now known to be identical with the enteric fever of adults. According to Murchison's experience, idiopathic remittent fever in children is almost invariably enteric. An excellent account of the fever will be found in Dr. Charles West's work on the "Diseases of Infancy and Childhood."[b] It is to this author that we are principally indebted for establishing the identity of infantile remittent and enteric fever.

7. **Senile Enteric Fever** may be veiled or latent. The abdominal symptoms are often but slightly marked, and temperature does not range high. The fever, however, is protracted and collapse is not uncommon. Hilton Fagge suggests that the atrophy of the lymphatic organs in old age, including those of the ileum, may be an anatomical condition which is unfavourable to the reception and multiplication of the enteric microbe. The same line of thought would explain the special features of the disease as it presents itself in elderly patients.

8. The **afebrile** or **apyrexial form** is of rare occurrence. Although the general symptoms, and especially the nervous symptoms, as delirium and stupor, may be well marked and rose-spots are present, yet the temperature remains normal throughout, or may even be subnormal. The intestinal lesions are usually slight, and the disease often terminates at the end of a fortnight. An outbreak of this type occurred in the German army besieging Paris in the winter of 1870–71, and has already been mentioned at page 395.

[a] *Clinique Méd. de l'Hôtel Dieu.* Paris. 1865. Tome I., p. 241.

[b] London : Longmans, Green & Co. 1874. Sixth Edition. Pages 769, *et seq.*

CHAPTER XLII.

Enteric Fever (*continued*).

Diagnosis—Prognosis and Mortality—Pathology.

No single symptom pathognomonic of Enteric Fever.—Diagnosis depends on ætiology, course of the disease, and particularly the temperature.—Ehrlich's Test. Diseases apt to be confounded with Enteric Fever: Typhus, relapsing fever, remittent fever, scarlatina, smallpox, pyæmia and puerperal fever, gastro-intestinal form of influenza, tuberculosis, trichiniasis, ulcerative endocarditis, acute rheumatism, &c. Prognosis and Mortality: Influence of age, sex, season, station in life, recent residence in an infected locality, intensity of the poison, family constitution, personal constitution and habits, previous diseases.—Modes of Death: Coma, syncope, asthenia or anæmia, hyperpyrexia (rarely).—Fatal Complications.—Pathological Anatomy: Specific Lesions —Non-Specific Lesions: affecting muscles, heart, liver, kidneys, nerve-cells, salivary glands, pancreas, larynx.

"There is not a single symptom belonging to typhoid fever which can be characterised as pathognomonic." So writes Liebermeister. Yet the **diagnosis** is tolerably certain in most cases, and absolutely so in well-marked cases.

Due consideration having been paid to the circumstances under which the illness arose, to the age and condition of the patient as regards pregnancy and so on, the diagnosis turns upon the course of the disease, particularly as regards the behaviour of the temperature, upon the enlargement of the spleen and the abdominal symptoms, and upon the presence of the rose-spots.

Probably it is to the temperature that we are to look with most confidence for a diagnosis in the early days of the fever.

Wunderlich considers that one is justified in **positively diagnosticating** enteric fever in attacks of moderate severity during the fastigium, when previously healthy persons of youthful or middle age, after being ill about five days or a week, exhibit evening temperatures of 103·5° to 105°, alternating with morning temperatures which are 1·4° to 2·7° Fahr. lower. He qualifies this statement by adding:—"Unless some other disorder of any

sort can be discovered to explain the height of the fever, or unless they have been the subjects of gross neglect immediately before coming under observation."

Wunderlich further thinks that we may with great probability assume that enteric fever **is not present**, when even on the first day or on the second morning the temperature rises to 104° F.; when, between the 4th and 6th days, the evening temperature in a child, or adult under middle age, never reaches 103° F., and indeed if it has failed to do so two or three times; lastly, when as early as the second half of the first week considerable or progressive diminutions of the evening temperature are met with.

Ehrlich's Test.—In 1882, Ehrlich[a] announced the fact that it is characteristic of the urine in enteric fever, measles, and acute tuberculosis to yield a deep-red colour with diazo-benzene-sulphonic acid—one of the anilin derivatives—whereas normal urine gives only a yellowish colour (like sherry or brown vinegar) with this reagent. Ehrlich obtains the reaction, not with the acid in question itself, but with sulphanilic acid. In applying the test two solutions are used—*A*, a saturated solution of sulphanilic acid in dilute hydrochloric acid (1 in 20); *B*, a 0·5 per cent. solution of sodic nitrite in distilled water. *A* is prepared by making up 50 cubic centimetres of hydrochloric acid to 1000 cc. with water and adding sulphanilic acid to saturation. To 200 cc. of this mixture 5 cc. of a ½ per cent. solution of sodium nitrite are added. The action of hydrochloric acid upon the nitrite of sodium forms nitrous acid, which, in the presence of sulphanilic acid, is converted into diazo-benzene-sulphonic acid. This is added to the urine in equal parts. Ehrlich has recently[b] recommended that 5 to 6 times the volume of absolute alcohol should be added to the fluid to be tested, and the reagent, prepared as above, should be added drop by drop to the filtrate. The urine thus treated should be rendered alkaline with strong ammonia. When the test is applied to the urine of a patient with enteric fever, the colour rapidly turns red, the tint varying from the yellowish-red of bichromate of potassium solution

[a] Zeitsch. für klin. Med. V. 285. 1882.
[b] Charité-Annalen. XI. 139. 1886.

through ruby-red to the colour of port wine. On shaking the test tube a froth is produced, which usually assumes a delicate and very characteristic pink colour.

Dr. Frederick Howard Taylor,[a] of the London Hospital, has recently (1889) submitted this diagnostic sign of enteric fever to a careful trial at that hospital. He finds that the reaction is not always given until the latter part of the first week, but *in every case* it is forthcoming during a great part of the febrile period. It is this fact which constitutes the great value of the test. Unfortunately, however, *in exceptional cases*, the reaction is given in other diseases. Herein lies the weakness of the test.

Dr. Taylor arrives at the following conclusions:—

1. The absence of the reaction is practically proof positive that the case in question is not one of enteric fever (provided that the disease has lasted six days or more, and that the temperature has not yet fallen to normal).

2. Its presence suggests, but does not prove, that the case is one of typhoid—the probability being greater the deeper the tint produced.

3. As the other diseases in which it occurs least seldom are not those which most closely resemble typhoid, but the reverse—for example, measles and phthisis pulmonalis—the significance of these exceptions is very much lessened.

On November 5th and 6th, 1891, I had the advantage, through the kindness of Professor Emerson Reynolds, and with the aid of Mr. Emil Werner, of testing the diagnostic efficacy of this very striking test in a series of specimens of urine. Two of the series were non-typhoid urines, two others were from enteric fever patients, and the fifth was the urine of a gentleman, aged 29, on the 13th day of well-marked enteric fever, he having suffered from equally well-marked enteric fever 14½ years previously, when a lad of 15 years. In all cases a change of colour in the urine was observed on application of the test. In the non-typhoid urines only a deeper yellow was produced; in the undoubted primary typhoid urines a beautiful rose coloration developed. In the case of recurrence, changes interme-

[a] *Lancet*, May 4, 1889.

Plate IX.

CHARTS OF TEMPERATURE RANGES IN RECURRENT ENTERIC FEVER.

Fig. 1.—First Attack, April, 1877. Patient aged 15 years.

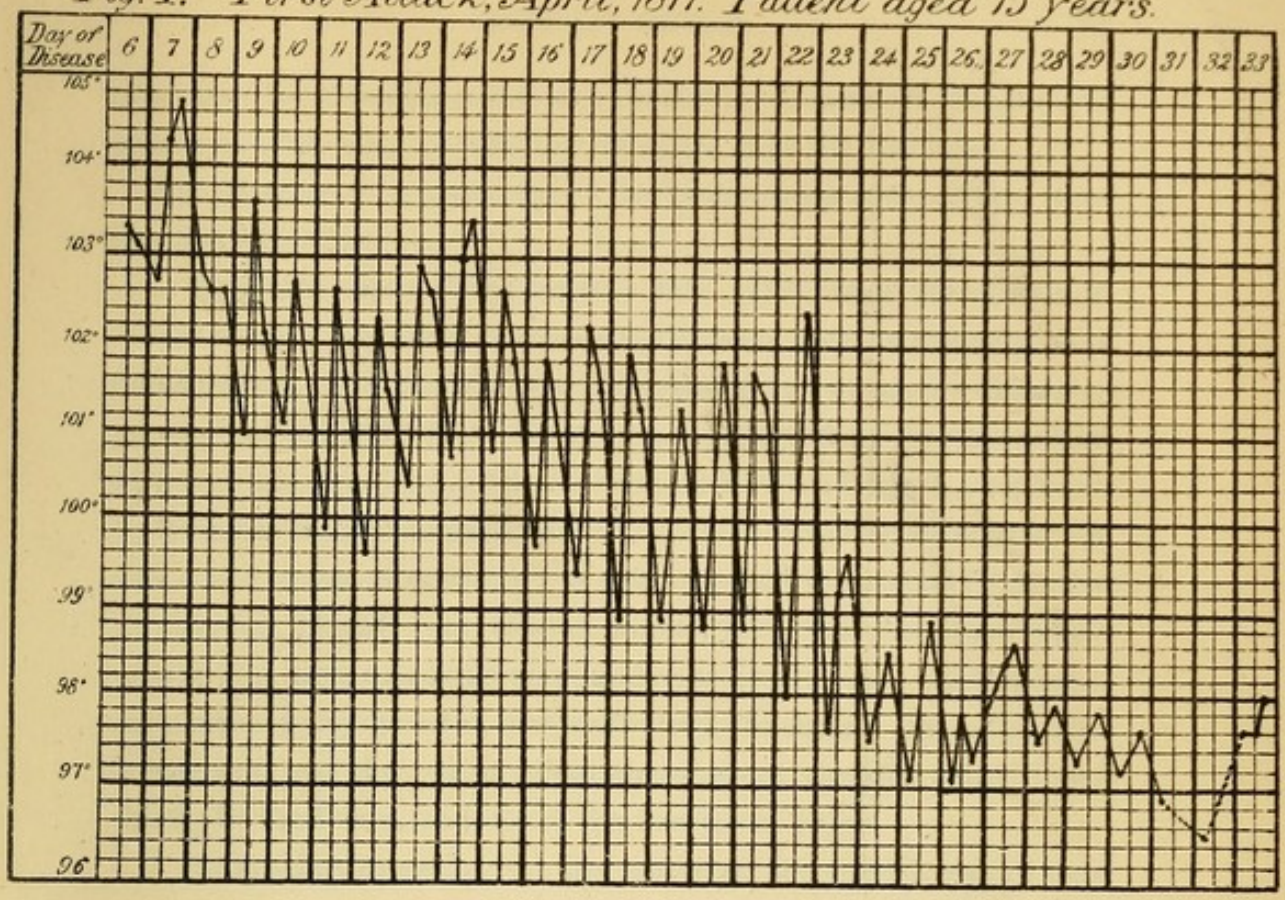
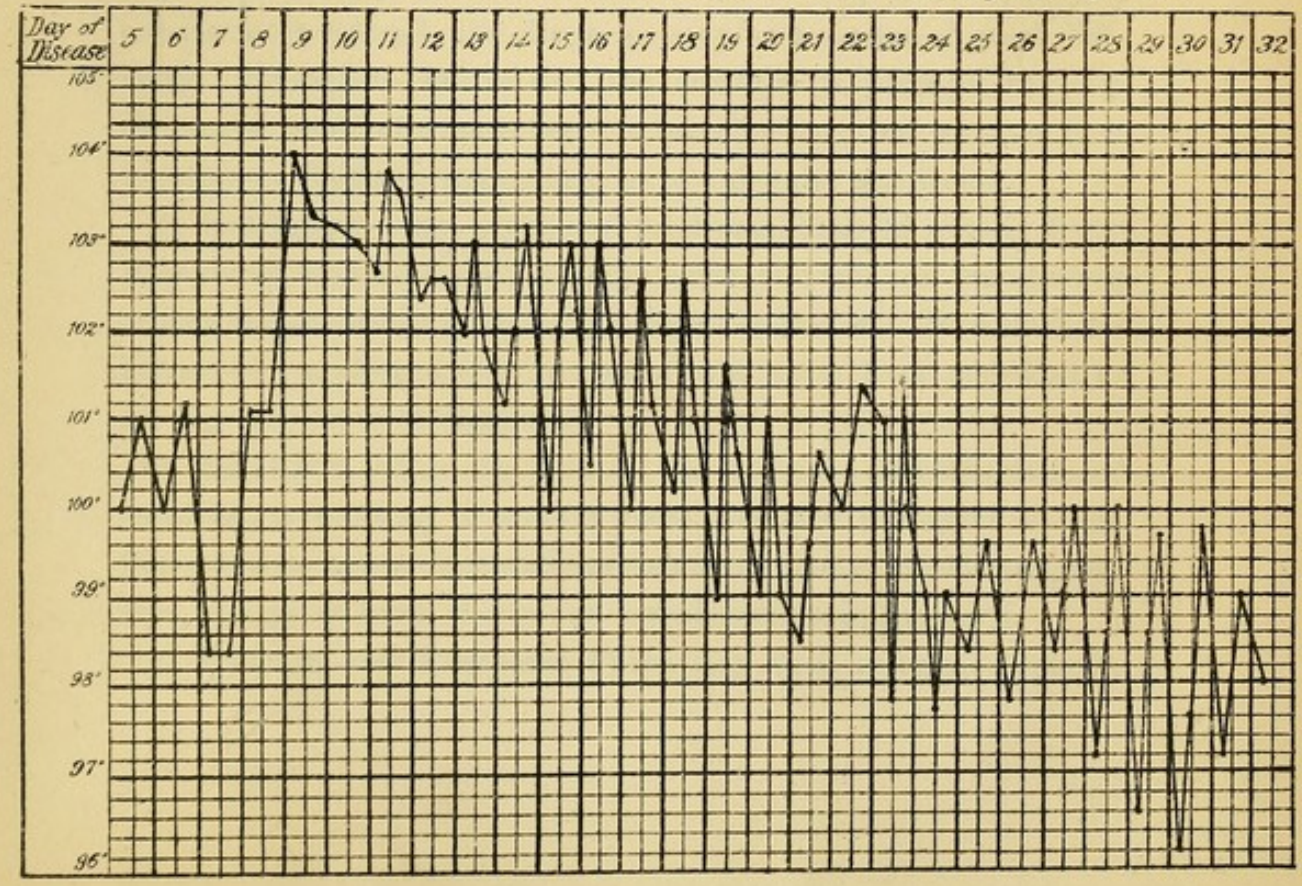


Fig. 2.—Second Attack, October, November, 1891, Patient aged 29 years.



diate between these extremes were noticed—it was as if the previous attack exerted some controlling influence over the reaction.

The diseases which are apt to be confounded with enteric fever are the following:—

1. **Typhus.**—Especially because this fever may sometimes be attended with diarrhœa, just as enteric fever may be attended with constipation. But enteric fever, in contrast to typhus, is a disease of youth, begins insidiously, is usually accompanied by diarrhœa, lasts at leasts ten days longer (24 as compared with 14 days), terminates by lysis, has an unchangeable eruption, which never becomes petechial and which fades after death; is characterised in general by a clear complexion, bright eyes, dilated pupils, persistent headache which sets in late, and comparatively moderate prostration; tenderness of the abdomen, tympanites, *gargouillement*; in enteric fever, epistaxis is common, and the pathological changes are specific, being found chiefly in the ileum, and about the ileo-cæcal valve.

2. **Relapsing Fever.**—But the ætiology, the sudden rise of temperature in this disease, the persistent pyrexia, the rapid defervescence and profuse sweating, the absence of rose-spots, diarrhœa, &c., and the characteristic relapse—are all points of contrast too marked to be mistaken or ignored.

3. **Remittent Fever.**—In countries where both this disease and enteric fever prevail, the diagnosis may be a matter of great difficulty. Murchison thinks that the eruption is, perhaps, the only distinctive mark of enteric fever to be relied on; but we have now in addition Ehrlich's test to help us to a definite conclusion.

4. **Scarlatina.**—An accidental prodromal rash in enteric fever may simulate this disease; but the mode of onset and the absence of sore throat should guide us to a right conclusion. In a child the lobster-like redness of the surface after a warm bath may lead to a temporary error in diagnosis.

5. **Smallpox.**—But the pain in the back, so characteristic of smallpox, is wanting in enteric fever, and the rose-spots of this latter disease appear late and are soft and velvety, deleble on pressure, and never hard, shotty and acuminated as in smallpox.

6. **Pyæmia and Puerperal Fever** may both resemble typhoid.

But rose-spots are absent, while an icteroid tinge, rigors, and profuse sweatings are commonly present. The ranges of temperature in pyæmia also are extremely great.

7. The gastro-intestinal form of **Influenza** may simulate enteric fever; but the course of the two diseases is quite different, as will appear from the annexed clinical record, which I included in my account of the influenza epidemic of 1889–90, as observed in Dublin[a]:—

"On Wednesday, January 8, 1890, Mr. W. B. S. enjoyed a day's shooting in the Co. Wicklow. The following day he returned to town in his usual good health; but in the afternoon felt chilly, complained of headache and nausea, and felt utterly miserable. He went to bed early, but passed a wretched night—restless and sleepless. Next morning I found him complaining of pains in the eyeballs, back of the head, and small of the back. Pulse 84; temperature, 99·9°; tongue thickly coated; complete loss of appetite and nausea. He felt entirely prostrate, and, at my evening visit, expressed his belief that some fish which he had eaten for dinner had thoroughly disagreed with him. Two miserable days of sickness followed, the temperature rising on the morning of the 5th day to 103·1°. A short cough had set in, and the eyes were suffused and tender. There was constipation, and he complained of weight and fulness in the pit of the stomach. Dr. James Little saw him with me and thought it likely that the fever would run on for some time. A quiet day gave promise of a restful night, and this promise was abundantly fulfilled. He had an excellent night, partly due to 20 grains of antipyrin, with 20 minims of tincture of gelsemium in a draught in divided doses at bedtime. Next morning, pulse 76; temperature 98·7°, rising to 100·2° in the evening, but without any return of restlessness. Subnormal temperatures followed for a few days—96·4° being one observation. The tongue cleaned very slowly, and several days of extreme languor and weakness preceded final convalescence. This gentleman's wife had, a few days previously, suffered from influenza, from which she was recovering when he fell ill. She nursed him and got a

[a] *Dubl. Journ. of Med. Science.* Vol. LXXXIX., page 300. April, 1890.

relapse, accompanied with cough, bronchial catarrh and absolute loss of appetite."

8. "The various manifestations of **Tuberculosis**," writes Murchison, "constitute the maladies most difficult to distinguish from enteric fever." He instances tubercular meningitis, tubercular peritonitis, acute tuberculosis of the lungs, and latent tubercle. He mentions a distinctive mark of tubercular meningitis which he learned by letter from Dr. Zuelzer, of Berlin. On ophthalmoscopic examination Cohnheim detected minute tubercles in the chorioid in a large number of cases of acute tuberculosis. In enteric fever, further, as pointed out by Sir William Jenner, headache ceases before delirium begins—both symptoms run concurrently in tubercular meningitis. The abdomen, too, is often retracted (scaphoid) in tubercular meningitis, and the *tache cérébrale* is easily produced.

In tubercular peritonitis, again, the temperature generally becomes sub-normal after a time.

As to pulmonary tuberculosis, we now possess an important aid to diagnosis in the microscopical examination of the sputum, in which tubercle bacilli may generally be found in abundance. The most convenient test to employ is the carbol-fuchsin (Ziehl-Neelsen) fluid, modified by Günther. Ten cc. of a concentrated alcoholic solution of fuchsin are added to 90 cc. of a 5 per cent. solution of carbolic acid. The specimen of sputum is immersed in a watchglassful of this fluid, which is then warmed. When thus fuchsin-coloured, the specimen is placed in a watchglassful of a solution consisting of 100 grammes of a 25 per cent. sulphuric acid containing 2 grammes of methylene blue. The preparation is then washed in water and mounted in Canada balsam. The bacilli appear red, all other fungi and cells blue.

9. **Trichiniasis** gives rise to pyrexia, with vomiting and diarrhœa, followed by ataxic, or typhoid, symptoms; and thus it may simulate enteric fever. The diagnosis turns on the presence of severe muscular pains, œdema of the eyelids, and sometimes of the whole body; the absence of rose-spots, of enlargement of the spleen, and of epistaxis (Murchison).

10. **Ulcerative (septic) endocarditis** has often been mistaken for

enteric fever. The discovery of a cardiac murmur should in most cases suffice to prevent this error, and the occurrence of hæmaturia or hemiplegia, while not decisive, would aid the diagnosis (Hilton Fagge).

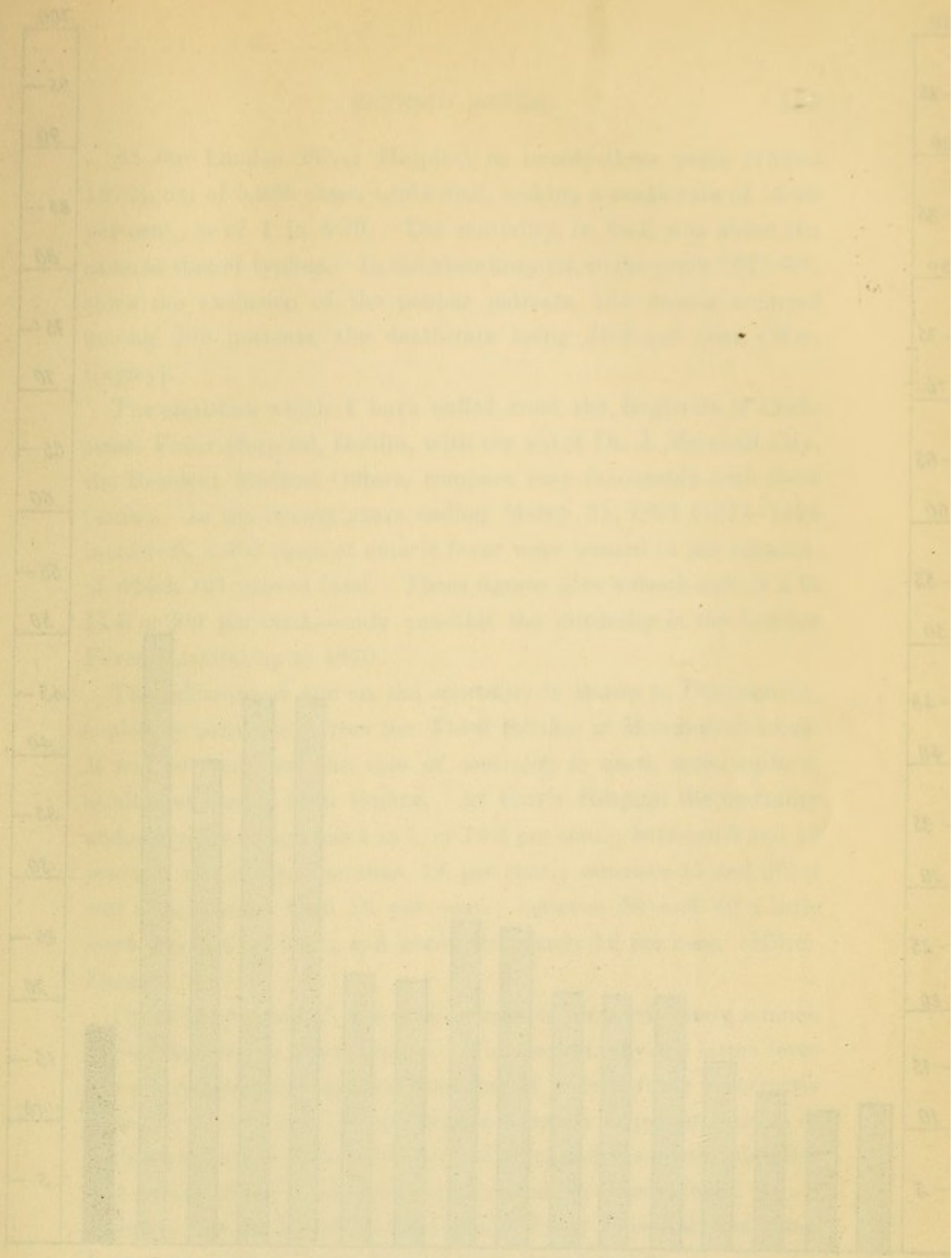
11. **Acute Rheumatism.**—The occasional association of enteric fever with symptoms of acute rheumatism is alluded to by Dr. Cayley,[a] who quotes a case, reported by Dr. Finlay to the Clinical Society of London, in which the primary attack presented the symptoms of acute rheumatism; a relapse occurred after 14 days with all the characters of enteric fever, and on the 10th day of the new attack perforation caused death. On *post-mortem* examination, besides a recent slough, some ulcers in the process of healing were found in the ileum, showing that the first attack had been accompanied by ulceration of Peyer's patches. As to the essential nature of this illness, for our part we are content to adopt Murchison's words. He says:—"In several instances I have known the pains in the limbs (in enteric fever) assume a neuralgic character, and prevent sleep; while in others they are articular, and the case at first *simulates* rheumatism."

Other morbid conditions which have occasionally led to doubt or confusion are:—Mania, pneumonia, gastro-enteritis, a "bilious attack" (all mentioned by Murchison); acute diffused diphtheritic colitis, typhlitis, perinephritic abscess, abscess of the liver, secondary to ulcerative colitis or to suppuration in the broad ligament of the uterus, diphtheritic inflammation of the labia (all mentioned by Hilton Fagge and Pye-Smith); and extravasation of urine, leading to stupor, muttering delirium, and a dry brown tongue (Sir William Gull).

Prognosis and Mortality.—"The forecast in enterica," says Dr. Hilton Fagge,[b] "depends partly upon the condition of the patient before its invasion, partly upon the symptoms which gradually develop themselves as each case goes on, and partly on early and judicious treatment."

[a] Murchison on *Continued Fevers*. Third Edition. 1884. Page 535.

[b] *Text-book of the Principles and Practice of Medicine*. Third Edition. 1891. Vol. I., page 168.



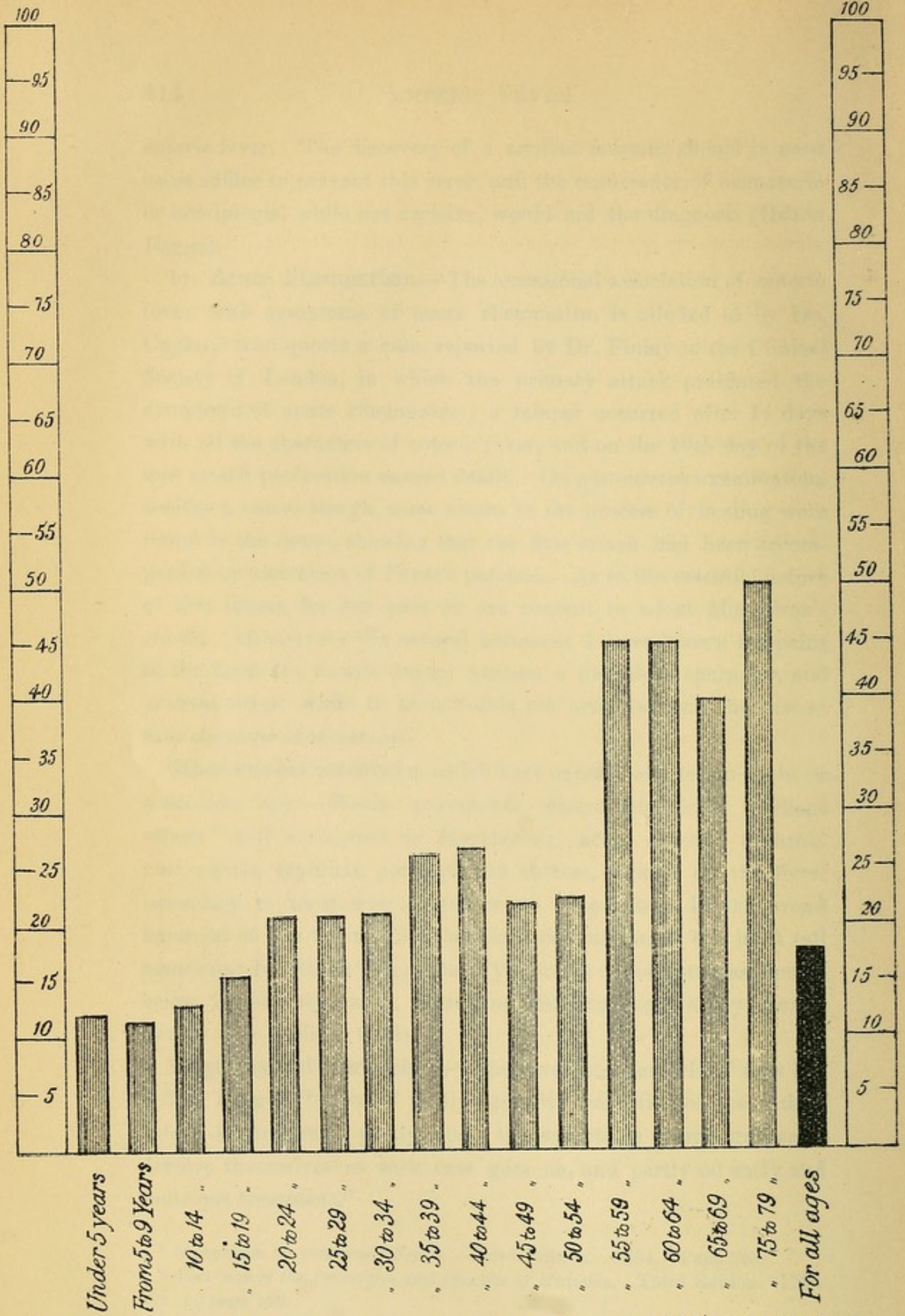


Diagram D shows the variations, according to Age, in the rate of Mortality of 5911 cases of Enteric Fever, admitted into the London Fever Hospital, (Compare with Diagr: B. Only 47 of patients were above 55 years)

At the London Fever Hospital, in twenty-three years (1848–1870), out of 5,988 cases, 1,034 died, making a death-rate of 17·26 per cent., or of 1 in 5·79. The mortality, in fact, was about the same as that of typhus. In the same hospital, in the years 1871–82, since the exclusion of the pauper patients, 144 deaths occurred among 905 patients, the death-rate being 15·9 per cent. (Wm. Cayley).

The statistics which I have culled from the Registers of Cork-street Fever Hospital, Dublin, with the aid of Dr. J. Marshall Day, the Resident Medical Officer, compare very favourably with these results. In the twenty years ending March 31, 1891 (1871–1890 inclusive), 1,405 cases of enteric fever were treated in the hospital, of which 121 proved fatal. These figures give a death-rate of **1 in 11·6 or 8·6 per cent.**—only one-half the mortality in the London Fever Hospital up to 1870.

The influence of **age** on the mortality is shown in Diagram D., copied by permission from the Third Edition of Murchison's work. It will be seen that the rate of mortality is much more uniform at all ages than it is in typhus. At Guy's Hospital the mortality under 5 years of age was 1 in 7, or 14·3 per cent.; between 5 and 15 years, it was rather less than 12 per cent.; between 15 and 30, it was a little more than 16 per cent.; between 30 and 40 a little more than 26 per cent., and above 40 nearly 50 per cent. (Hilton Fagge).

Up to forty years of age enteric fever is proportionately a much more fatal disease than typhus. The reason why the latter fever has a gross mortality greater than that of enteric fever is correctly stated by Murchison. It is because a much larger proportion of typhus patients exceed forty years of age, and the death-rate after that period of life is much in excess of that in enteric fever.

Sex.—The death-rate in the London Fever Hospital was about 1 per cent. higher among females than among males.

Season and **Station in Life** do not materially affect the death-rate, but **recent residence in an infected locality** raises it considerably, and so do the **intensity of the poison** and **family constitution.** In this last particular, enteric fever is akin to scarlatina (page 181).

Lastly, the prognosis is **bad** in the obese, the very muscular, the intemperate; as well as in those who are the subjects of gout or of kidney disease.

Death is brought about by **coma**, or by **syncope**, most commonly by the end of the second, or early in the third, week; by **asthenia** or **anæmia**. in the third or fourth week, or even later; and only rarely by **hyperpyrexia**.

At a meeting of the Pathological Society of Dublin, on January 15, 1876, Dr. James Little[a] showed the intestinal lesions in a case of acute enteric fever in a young woman who sickened on January 2, with sore throat and vomiting, and died on the 8th—that is, on the **seventh day** of her illness.

The **complications** which most frequently terminate fatally are: perforation, peritonitis, intestinal hæmorrhage, pulmonary congestion, pneumonia, bronchitis, and diarrhœa.

Pathological Anatomy.—Enteric fever differs from both typhus and relapsing fever in the almost invariable presence of **specific lesions**, particularly in the glandular structures of the ileum and of the mesentery. But this fact is in no way subversive of the doctrine that enteric fever, like typhus or relapsing fever, is an **essential disease**. The words of Fordyce, quoted by Graves,[b] are exactly applicable:—"Fever is a disease which affects the whole system; it affects the head, trunk, and extremities; it affects the circulation, absorption, and the nervous system; it affects the body, and it affects the mind; it is therefore a disease of the whole system, in the fullest sense of the term. It does not, however, affect the various parts of the system uniformly and equally, but, on the contrary, sometimes one part is more affected than another."

"In several specific fevers," says Hilton Fagge,[c] "we find, in addition to the universal 'intoxication' with the poison, that it fixes itself peculiarly in certain *foci*, as we may call them, where it

[a] *Dubl. Journ. of Med. Science.* Vol. LXII., page 60. 1876.

[b] *Clinical Medicine.* New Syd. Soc. 1884. Vol. I., page 121.

[c] *Text-Book of the Principles and Practice of Medicine.* Third Edition. Vol. I., page 154. London: J. & A. Churchill. 1891.

produces definite local lesions. Thus measles particularly affects the respiratory mucous membranes, and scarlet fever the throat. But nowhere is this localisation of the disease so remarkable as in enteric fever. In fact, so striking and clinically important are the local lesions that it was possible for Broussais and his school to regard them as the primary disease, and the fever as merely a symptomatic result. Probably the 'typhoid deposits,' as they used to be called, in the intestine, are to be regarded as infective granulomata, produced, like the products of tubercle and leprosy, by the local action of specific bacilli."

The **morbid anatomy** of enteric fever is thus **epitomised** by Murchison :—

1. The agminated or solitary glands of the ileum, the mesenteric glands, and probably the spleen, are invariably diseased.

2. Many other secondary lesions are found, which are not constant or essential. The chief of these are—peritonitis, granular or other degenerations of the liver, kidneys, heart and voluntary muscles, ulcerations of various mucous surfaces, pneumonia, bronchitis and hypostatic congestion of the lungs, and an increase of intra-cranial fluid. There are no signs of inflammation in the brain, or of its membranes, to account for the cerebral symptoms.

3. There is no specific typhous exudation, and no evidence that the secondary lesions are due to the deposit of a material like that found in the intestinal and mesenteric glands.

4. The enlargement of the intestinal and mesenteric glands is not due to any attempt at elimination, but to inflammation, which is probably excited by absorption of a poison in the bowel.

In 1864, Zenker[a] pointed out two kinds of degeneration in the fibres of **voluntary muscles** in enteric fever. Some of these fibres become granular; others undergo conversion into a glassy-looking (hyaline)[b] substance, in which no striæ can be recognised, and which splits up transversely into discs. The change is especially

[a] *Ueber die Veranderungen der wilkuhrlichen Muskeln im Typhus abdominalis.* Leipzig. 1864.

[b] Gk. *ὑάλινος, of crystal,* or *glass,* from *ὕαλος, any kind of clear, transparent stone,* hence *glass* (Lat. *vitreum*).

marked in the adductors of the thighs and in the recti abdominis. During life, muscles thus affected become soft and pliable. Hoffmann has shown that similar forms of degeneration occur in the tongue, accounting perhaps for its tremulousness. Zenker himself remarked that the changes now described are not peculiar to enteric fever, but may take place in other febrile diseases, if sufficiently severe and protracted.

The **heart** is soft and pale in fatal cases, with dilatation of the right and sometimes of the left ventricle. Its muscular fibres are more or less granular and may have lost their transverse striation. Zenker[a] once observed hyaline degeneration.

The **liver** is softened, on section presenting a pale or "clayey" look. Examined microscopically, its cells are found to be granular and disintegrating. Minute gray nodules are, according to Wagner and other German pathologists, often found in the substance both of the liver and of **the kidneys**; the epithelium of the last-named organs are the seat of cloudy swelling and granular degeneration.

Hoffmann described the nerve-cells in **the great basal ganglia of the brain** as being deeply pigmented. He also found an enormous overgrowth of cells in the acini of **the salivary glands** and of **the pancreas**, which structures, according to this authority, feel unusually hard and dense.

The **larynx** is sometimes ulcerated. Hoffmann observed such a state of things in 28 out of 250 cases. This lesion has been already described (see page 396).

It will be necessary to treat of the more specific lesions of enteric fever in a separate chapter.

[a] *Loc. cit.* See also Chapter XVIII., pages 178 & 179.

CHAPTER XLIII.

Intestinal and Splenic Lesions of Enteric Fever.

Specific Lesions of the small intestine, mesenteric glands, spleen.—The stomach, duodenum, and jejunum usually healthy or seat of non-specific lesions.—Neighbourhood of ileo-cæcal valve, the chief seat of disease in both ileum and cæcum.—"*Enterica sine enteritide.*"—"Infective Granuloma" (Hilton Fagge).—The Intestinal Lesion: its four stages—(1.) enlargement and infiltration (2.) softening and ulceration; (3.) "typhoid ulcer;" (4.) cicatrisation.—*Plaques dures et molles* (Louis).—*Plaques réticulées et gaufrées* (Chomel).—Characters of the "Typhoid ulcer."—"Atonic ulcers."—"*Shaven-beard*" appearance (*l'état pointillé*).—Perforation of the peritoneum.—Lesions of the Mesenteric Glands.—Lesions of the Spleen: *Putrilage*, abscess.

As the brunt of the onslaught of the specific poison falls in scarlatina on the throat, in measles on the respiratory mucous membranes, in smallpox on the skin, and—shall I add—in pneumonic fever on the lungs, so in enteric fever the battle-field is first and chiefly the small intestine, the neighbouring mesenteric glands, and the spleen. The stomach, notwithstanding the frequent occurrence of anorexia, nausea, and vomiting, in many cases escapes all pathological change; in others the morbid appearances which it may present—for example, increased vascularity, softening, mammillation,[a] and superficial ulceration (Murchison)—are inconstant and non-essential. They may be observed with equal frequency after death from other diseases. Louis long ago pointed out that "la fièvre typhoïde" had no more claim to the name of *gastro-entérite*, than pneumonia had to that of *gastro-peripneumonie*.

The duodenum and the jejunum are usually healthy in enteric fever, and so also may be the greater part of the ileum.

But, starting from the ileo-cæcal valve, morbid changes are found to extend upwards into the cæcum and ascending colon in about one-third of the fatal cases; but more particularly and much

[a] Lat. *Mamilla, a nipple.* Fr. *mamelonnation*, the condition of being mammillated, that is, *of having* (or *presenting*) *nipple-like prominences.*

more frequently—backwards into the ileum, in some instances only for a few inches, in others for two or three feet, or further.

These morbid changes affect both the solitary glands and the agminated glands (Peyer's patches) of the ileum, and also the solitary glands of the colon. But the distribution of the lesions is most variable. In some cases the solitary glands entirely escape; in others, the glands of the ileum are alone attacked; in others, those of the cæcum suffer; in a few the lesions can be traced to the sigmoid flexure or even to the rectum (Hilton Fagge).

In very exceptional cases, Peyer's patches remain unaffected, and the solitary glands bear the whole brunt of the disease. Murchison saw two examples of this variety, which Cruveilhier designated *la form pustuleuse*. More commonly, Peyer's patches are the selected seat of the morbid changes, but even then the diseased processes seem to start from the ileo-cæcal valve, and to extend backwards into the ileum only for a comparatively short distance.

This apparent caprice of distribution of pathological changes suggested to Dr. Hilton Fagge the question whether there may not be some cases of enteric fever in which no glands suffer at all. In a specimen exhibited by him to the Pathological Society of London in 1875, the only lesions in the intestines were the following:—"One ill-defined purplish red patch, of about the size of a shilling, situated a foot above the valve; and a little higher up another patch, presenting similar characters, except that in its centre there was a darker spot the size of a pea, with a breach of surface, visible only when it was examined under water."[a] Hilton Fagge considered that it is by no means unlikely that in mild cases of enteric fever, such as never could prove fatal except by some accident, the intestinal lesions are often very slight, and may possibly in rare cases be altogether absent. This view is, of course, diametrically opposed to the dogmatism of Murchison,[b] who speaks of "the specific lesions, which *are invariably present*."

[a] *A Case of Enteric Fever with extreme ulceration of the Larynx, and but little affection of the Ileum.* Trans. of the Path. Soc. of London. 1876. Vol. XXVII., page 40.

[b] *Treatise on the Continued Fevers.* Third Edition. 1884. Page 617.

But is such dogmatism philosophical, or in accordance with analogy? Sure, if we may have *Variola sine variolis*, *Scarlatina sine anginâ*, *Morbilli sine catarrho*, we may expect occasionally to meet with *Enterica sine enteritide*. Dr. Stokes,[a] no doubt, went too far when he said: "It would be more philosophical to say that no form of fever has any special anatomical change; that where such does take place it is of a secondary character; and that, when it arises in the digestive system, it is more frequently observed in one form of fever than in another." Yet there is a kernel of truth in such a statement, and so long as the essential or specific nature of each febrile disease is admitted, so long must we look upon the local lesions as secondary, and therefore accidental rather than essential.

At a meeting of the *Société des Hôpitaux de Paris*, March, 1890, Dr. Vaillard described the following case:—A soldier after an attack of influenza became very ill, suffering from stiffness in the neck, headache, coma, constipation; temperature, 104° F. Death on the 10th day. At the autopsy were discovered hyperæmia of the meninges and lungs, and enlargement of the spleen, while the intestines were perfectly healthy. Cultivation experiments showed the presence of an organism, which appeared to be the bacillus of typhoid, in the spleen, lungs, and medulla. A streptococcus was also present. Vaillard believes that this was a case of enteric fever without the usual symptoms.

In the discussion which ensued, Dr. Chantemesse stated that he had found the typhoid bacillus in a dead-born fœtus, whose mother aborted during an attack of typhoid. Its intestine was perfectly healthy. It had died of typhoid septicæmia. In another case which he had observed a man died of typhoid. Many typhoid bacilli were found in his organs, but the intestine was perfectly healthy, with the exception of one ulcer of the size of a lentil.

These observers therefore conclude that there may be in rare cases **a typhoid septicæmia without any local lesions.**

On January 23, 1891, Dr. Sidney Phillips[b] submitted to the

[a] *Lectures on Fever.* 1874. Page 238.

[b] *Trans. of the Clin. Soc. of London.* Vol. XXIV., page 104. 1891.

Clinical Society of London notes of two cases of typhoid fever, fatal at a late period of the disease without ulceration of the intestine.

On November 27, 1880, I laid before the Pathological Society of Dublin specimens from a case of enteric fever in which there was no disease of the glands of the ileum, while the spleen was extremely large, soft, and friable, and in a state of *putrilage*. The ileum was carefully examined. In places the mucous membrane was congested to the extent of two or three inches, but there was not the slightest trace of past or present disease of the agminated or solitary glands. Peyer's patches were, indeed, apparently less distinct than usual. They were not hyperæmic, and did not present the "shaven-beard" appearance. This was, no doubt, an example of what is now known as "spleno-typhoid." (See page 406.)

At the same time, in an overwhelming majority of cases, characteristic lesions are certainly present in the intestines of enteric fever patients. These I will now try to describe as briefly as may be.

Certain facts led Dr. Hilton Fagge to the belief that **during the period of incubation** the invading *Bacilli typhosi* produce a local "**infective granuloma**," and that this remains latent until they (or their spores) leave the intestinal follicles by the lymphatic channels, and overspread the entire organism in swarms.

Be this as it may, the primary local lesions in the intestines run a course somewhat parallel to that of the symptoms, and with Bretonneau (of Tours), Trousseau, and Liebermeister, we may divide the history of the development and retrocession of these lesions into **four several periods of seven days** each, or **weeks**; or, with Murchison, we may describe the lesions as passing through **four stages**, although the disease is often arrested at the end of the first. These stages closely correspond with periods of seven days each, so that there is no contradiction between the several authorities I have just named. The stages are:—1. The stage of **enlargement** of the intestinal glands (Fr. *Engouement*). 2. The stage of **softening** and **ulceration** (Fr. *L'étage furonculeuse*). 3. The stage of the genuine "**typhoid ulcer**" (Fr. *Le bourbillon*). 4. The stage of **cicatrisation.**

I. The **first stage** probably **begins with the disease**, perhaps even in the closing days of the period of incubation. The solitary and agminate glands in that portion of the ileum nearest to the ileo-cæcal valve swell, and become the seat of what Liebermeister calls a **medullary infiltration.** The neighbouring mucous membrane is usually, but not necessarily, hyperæmic. These changes go on **through the first week**, so that by the eighth day or so Peyer's patches are, in cases which have proved speedily fatal, found indurated and elevated from half a line to two lines above the surface of the bowel. The hardness of the diseased patches or plates varies. If marked, the patches form the **plaques dures** of Louis. If the enlargement is only slight, and the consistence of the patches is soft, they are the **plaques molles** of Louis. These are the **plaques réticulées** (the network plates), and the **plaques gaufrées** (the honeycombed plates) of Chomel. Dr. T. J. Maclagan endeavoured to show that the **plaques molles** are always excited by a secondary inoculation with poison thrown off by a **plaque dure**—in other words, that the *plaques dures* are primary, and the *plaques molles* are secondary lesions. This view has not been confirmed.

As is well known, a Peyer's patch is a roundish or elliptical area, situated at the free border of the intestine, 1·5 centimetres in length, 7·2 centimetres in breadth, with its long axis corresponding to that of the intestine (at least, in the ileum), and consisting of twenty or more lymph follicles grouped together and separated from each other only by thin prolongations of the submucous tissue (E. Verson [a]). In enteric fever, the proper structure of a Peyer's patch first becomes enlarged by a proliferation of its cellular elements, the surrounding connective tissue next becomes involved, until, at last, the whole patch is converted into a continuous mass of altered gland-tissues. This, according to Murchison, is what happens in the case of the *plaques dures.* In the *plaques molles* the morbid process stops short of this—the glandules become enlarged, but not to such an extent as to run into one another.

[a] *Manual of Human and Comparative Histology.* Edited by S. Stricker. Vol. I., page 565. New Syd. Soc. 1870.

II. The **second stage** is nearly **completed** by the end of the second week. The infiltration of Peyer's patches gradually progresses, and some of the swollen patches ulcerate and become partially or wholly necrotic—the process leading to the formation of sloughs stained a bright ochre or yellow by the bile (Liebermeister). Wm. Budd[a] considered that "this yellow matter is the peculiar 'typhoid matter,' whose presence is typical of the disease, and whose formation and elimination constitute the essence of the intestinal process." This view is not now entertained by any authorities on enteric fever, so far as I know.

Ulceration seems sometimes to begin as an abrasion of the surface of the diseased follicles, and gradually to extend through their substance; much oftener, however, the whole or most of the infiltrated tissue dies in a mass, forming a soft, shreddy, flocculent slough of a bright ochre colour as above (Hilton Fagge). The former process affects the *plaques molles*, the latter the *plaques dures* (Murchison).

III. The **stage** of the "**typhoid ulcer**" is, in Murchison's words, that which intervenes between the commencement of ulceration and the commencement of cicatrisation. It is impossible to fix its limits, as they vary in different patients, and in different ulcers of the same bowel. For the sake of convenience, however, we may say that ulceration usually takes place after the 9th or 10th day, and in the third week sloughs fall off, the cleaning off of the ulcers being in general well-nigh completed by the 21st day or so.

"**Typhoid ulcers**" are distinguished by the following characters:—

1. **Situation.**—In the lower third of the ileum, chiefly near the ileo-cæcal valve.

2. **Size.**—They vary in diameter from one line to an inch and a half.

3. **Form.**—If their seat is a Peyer's patch, they are elliptical; if a solitary gland, circular; or they may be irregular from the coalescence of several ulcers.

[a] *Typhoid Fever: its Nature, Mode of Spreading, and Prevention.* By William Budd, M.D., F.R.S. London: Longmans, Green & Co. 1873. Page 47.

4. **Anatomical Relations.**—The elliptical ulcers are always opposite the attachment of the mesentery, and their long diameter corresponds to the longitudinal axis of the gut.

5. **Outline.**—Their margin is formed by a well-defined fringe of mucous membrane, of a purple or slaty-gray colour, one line or more in width, detached from the submucous tissue. There is no thickening or hardening of the edge, as in the tubercular ulcer.

6. **Base.**—This is formed by the submucous tissue, or by the muscular coat of the intestine, or by the peritoneum.

As the sloughs fall off, losses of substance of variable extent and depth take place, involving, it may be, the muscular coat, and even the peritoneum. In the latter case, of course, perforation occurs.

IV. At some time during the fourth week, the **reparative process** called **cicatrisation** begins, and may continue for a long time—in the case of an individual ulcer it occupies about a fortnight. Cicatrisation begins in the ulcers nearest the cæcum, and proceeds upwards. The floor of the ulcer becomes covered with a thin, gray, shining layer of granulation tissue, which is dove-tailed, so to speak, between the muscular coat and the detached fringe of mucous membrane. The ulcer then heals by a gradual growth of mucous membrane from the edges towards the centre—an epithelial covering being slowly formed over the ulcer, adherent at first, but afterwards becoming movable on the submucous coat. The gland-structure which has sloughed away is not regenerated. The cicatrix is slightly depressed, firmer, less vascular, and smoother than the surrounding mucous membrane. When held up to the light, the bowel appears thinner at the part. There is no surrounding puckering, and the calibre of the gut is not narrowed (Murchison). According to Chomel,[a] all traces of the ulcers disappear after a short time, but Rokitansky[b] has recognised them *thirty years* after an attack of enteric fever.

Occasionally the process of cicatrisation is much prolonged, owing to the ulcers becoming **chronic**, or, as some pathologists say,

[a] *Leçons de Clinique Médicale.* Tome I. Paris. 1834.
[b] *Path. Anat.* Syd. Soc. London. 1852. Vol. II., page 73.

atonic. These chronic or atonic ulcers after the fourth week may cause severe diarrhœa, or may lead to fatal perforation.

The pathological changes just described may proceed simultaneously in different parts of the small intestine—being most advanced near the ileo-cæcal valve, where cicatrisation may have already begun while fresh infiltrations are only taking place in portions of the bowel at a distance from that centre of disease. This state of things is beautifully shown in the coloured Plate which forms the Frontispiece to this work. The original drawings, from which the lithograph was skilfully and artistically produced by Mr. John Falconer, of 53 Upper Sackville-street, Dublin, were executed in water-colours at my request by my friend and pupil, the late Mr. Edmund P. Henn, at the Meath Hospital and County Dublin Infirmary. They faithfully represent the appearances in a fatal case of enteric fever under my care in the year 1879.

Fig. 1 shows two Peyer's patches and four solitary glands in the first stage of the typhoid disease in the ileum at a distance of some two feet from the ileo-cæcal valve. Fig. 2 represents the commencement of the second stage—that of ulceration—at a lower level in the ileum. It will be seen that at two points the tumefied Peyer's patch is beginning to ulcerate, and already a bright yellow coloration is commencing to show itself. Fig. 3 represents 4 inches of the ileum nearest to the valve, and also the neighbouring mesenteric glands—the latter greatly swollen. In this case the slough is fully formed, and is about to be cast off from a patch between two and three inches from the valve; while the slough from a large ulcer close to the valve itself has already been detached in part and cast off.

One is not to suppose that, in every case of enteric fever, the local lesions in the intestines run through all the four stages which have been just described. On the contrary, the instances are not few in which retrograde changes may occur as early as the close of the first week. In the milder cases, the swelling (or, *engouement*) of Peyer's patches may gradually subside by degeneration and absorption until both patches and separate follicles return to their normal state. If the follicles of a Peyer's patch retrograde faster than the

interstitial tissue, this latter may for a time form a projecting network (*plaques à surface réticulée*). Frequently, the swollen follicles soften and break down, so that the patches again present a reticulated appearance. Especially in this case, but sometimes with simple swelling, numerous little ecchymoses occur, so as to produce a punctate pigmentation of the patches. This gives a dotted appearance to the patches—the **shaven-beard** appearance of English, the **état pointillé** of French writers. Hilton Fagge denies that this condition is peculiar to enteric fever—in this opinion he is borne out by Murchison, who has seen this change in cases of typhus, cholera, phthisis, and so on.

Perforation of the peritoneum may take place, according to Murchison, in one of three ways.

1. It may be due to disintegration, or to an extension of the ulcerative process. The opening is then always minute and circular, just large enough to admit a pin or a stocking wire. This is the most common form of perforation.

2. A portion of the peritoneum may slough, and perforation may result from the partial or complete detachment of the slough. The openings are, in this case, many and of considerable size.

3. The perforation may result from rupture of the denuded peritoneum at the base of one of the destroyed Peyer's patches.

Among twenty-nine cases of perforation, of which Murchison had accurate notes, the first form was present in fifteen, the second in ten, and the third in four cases.

Dr. Robert S. Archer,[a] formerly Physician to the Netherfield Fever Hospital, Liverpool, in narrating the clinical histories of six cases of perforation of the intestine in enteric fever, very properly draws a distinction **between perforation** in the proper sense of the term and **rupture** of the peritoneal coat at the base of an ulcer. In the case of perforation, the process would seem to be a gradually progressive necrotic one, involving in order all the coats of the gut from within outwards, the peritoneal coat at length succumbing to the destructive change. The perforation is in this case usually more or less circular, and its edges are somewhat

[a] *Dubl. Journ. of Med. Science.* Vol. LXXXIV., page 90. 1887.

thickened by lymphy deposit. Its size may vary from a pin-hole opening to one half an inch or more in diameter. Lymph is thrown out around the opening, and adhesions are in process of formation.

In rupture of the peritoneal coat, on the other hand, the ulcerative process seems to stop suddenly at, or has not extended to, the serous membrane, and the latter gives way from the pressure of fæces or gas. In this case the opening takes the form of a more or less regular angular slit, with little or no deposit of lymph, and the edges may be brought together with considerable accuracy.

The **Mesenteric Lymph-glands** are very commonly enlarged in enteric fever—no doubt from absorption by the lacteals of contagion from the affected parts of the intestines (Hilton Fagge). Hence it is that the swelling of the glands is most developed in these parts of the mesentery which correspond to the diseased portions of the intestine. The swollen glands may not be larger than a hazel-nut, but some may be as large as a horse-chestnut, or a pigeon's egg, or even a hen's egg. At first the swollen glands are hyperæmic, rosy red or bluish-red, tense; afterwards, they become paler, assuming a pinkish or grayish tint. Suppuration sometimes occurs, while caseation or the deposition of calcareous salts forms part of the ordinary retrograde process. Examined histologically, the enlarged glands show a cellular hyperplasia—or numerical hypertrophy—with hypertrophy of the interstitial connective tissue (Liebermeister.)

Fig. 3 in the Frontispiece exhibits very well the great enlargement of the mesenteric glands near the ileo-cæcal valve, which is so often present.

In some cases other lymphatic glands are engaged also. For example, the retroperitoneal glands, the bronchial glands, and others—but these are secondary and accidental phenomena, usually due to non-specific irritations.

The **Spleen** is commonly **enlarged** and **softened**, being the seat of changes which are strictly analogous to those observed in the intestinal follicles and the mesenteric glands. The enlargement begins early in the fever, and can be detected after the middle of the first week. At this period, the organ is tense, firm, and hyperæmic. The spleen increases in size up to the third week and sub-

sides again in the fourth week. The enlargement is most marked in young subjects, and may be wanting in elderly persons. Louis found the spleen softened in thirty-four out of forty-six cases, and in seven it was reduced to a mass of "**putrilage**"[a] (Murchison). According to Rokitansky, the enlarged, softened spleen is liable to spontaneous rupture. **Bacilli** have frequently been found in the spleen in enteric fever. Their discharge into the general circulation when the organ suddenly subsides may account for some of the cases of **relapse** which have been recorded.

Rarely, **abscess of the spleen** occurs. Murchison's vast experience gives only two cases of this lesion, and he quotes five others from different authors. In the *Dubl. Journ. of Medical Science* for February, 1886 (Vol. LXXXI., page 109), will be found an interesting case in a boy, aged fourteen, which was reported by Dr. Robert S. Archer, then Physician to the Netherfield Hospital, Liverpool.

Such are the **primary local lesions** of the disease before us. The **secondary local lesions** are principally of the nature of a **parenchymatous degeneration** with **cloudy swelling** of various organs—such as the liver, heart, and voluntary muscles (*Myositis typhosa*)—which is not in any way characteristic of enteric fever, but which is forthcoming in all febrile affections in which pyrexia is sufficiently intense and persistent.

[a] Pultaceous or pappy matter, which forms in the course of certain necrotic or gangrenous affections. Lat. *putrilago—rottenness, corruption, putrefaction.*

CHAPTER XLIV.

The Prophylaxis of Enteric Fever.

Measures to be adopted for Checking the Development of the Fever Poison: Efficient drainage system.—Improved water-closets.—Drinking water to be taken direct from the main.—Use of chemical disinfectants.—Measures for Preventing the Propagation of the Fever Poison: Disinfection of excreta.—Treatment of bedding and body linen.—Ventilation of sick room.—Trace origin of first case of fever in each outbreak.

"A man," says Liebermeister,[a] "who avoids breathing the exhalations of privies and sewers, who does not handle linen foul with typhoid dejections, who does not drink unboiled water from infected springs, is as safe in a place where a typhoid epidemic is raging as in one where not a case of the disease exists."

This aphorism of Liebermeister places in our hand the key to the **preventive treatment**, or **prophylaxis** of enteric fever.

The subject may be considered under two headings—

1. The measures to be adopted for **checking the development of the fever poison.**

2. The measures to be adopted for **preventing the propagation of the fever poison.**

I. A careful study of Chapter IV., and particularly that portion of it which deals with the subject of disinfection without and within the body (page 31–38), will place the reader in possession of full information as to the best means at our disposal for achieving the first end in view—the checking or prevention of the growth and development of the specific poison of enteric fever.

It is a popular error to suppose that this poison is an offensive gas which can easily be smelled. On the contrary, it is probably quite inodorous, although often accompanied by bad smells, the result of putrefaction of the retained contents in defective drains.

[a] Von Ziemssen's *Cyclopædia of the Practice of Medicine.* Art. "Typhoid Fever." Vol. I., page 71.

1. The first essential is that there should be an **efficient drainage system**—consisting of (*a.*) flushing apparatus, (*β.*) properly laid, well-ventilated, and securely trapped house-drains, discharging into (*γ.*) well-constructed public sewers.

This is not the place to enter into details on the subject of house-drains and public sewers (I use these terms in their strictly legal, or technical and accepted sense). But a few remarks will, perhaps, prove useful. They are based upon an excellent description in Mr. Winter Blyth's "Manual of Public Health."

A house-drain should be constructed of glazed socket pipes, made of pipe-clay or a mixture of fire and pipe-clay—salt or glass-glazed.

These drain-pipes should each be provided with a collar or socket at one end, so as to receive the spigot end of the next pipe, the joints being carefully filled in with Portland cement, unless a patent joint, such as Stanford's, is used. In this a sort of ball joint is made, and a little grease renders all tight. All joints should be water-tight. For ordinary ten-roomed houses, 4-inch pipes are ample. Larger houses require pipes of 5 or 6 inches diameter, and country mansions may require for the main drain a 9-inch pipe.

The pipes should be laid on a concrete bottom, with a fall of at least *one in forty-eight*—that is, $\frac{1}{4}$ inch to a foot. When one drain joins another, the junction should be at an acute, not a right angle; or in the form of a curve—a larger pipe must receive a smaller, not *vice versâ*. Of late years, special lengths of pipe, gradually lessening in diameter from 9 to 6 inches, or from 6 to 5 or 4 inches, have been used for making such junctions. Drain pipes should not, if possible, be laid *under* a house. When a drain changes its direction, means of inspection should be provided.

Every drain should have an "inspection chamber" at its lower end, before it enters the "intercepting-trap," which cuts it off from the public sewer, as well as a "fresh-air" inlet in this same situation, and a "ventilating shaft," carried above the dwelling-house at the highest point of the drain. In this last-named situation also a "flushing-tank" should be provided, capable of discharging into the drain through a 4-inch pipe not less than 40 gallons of water at least once a day. It is a great mistake to shut up a city

residence for two or three months in the summer season, and to return to it in autumn without first causing the drains to be repeatedly flushed and thoroughly cleansed and disinfected. I am sure mischief to health is often caused by inattention to such details.

In addition to the house-drains, **water-closets** require attention. They should be placed at the back of the house, and if in structural connection with it a short lobby or corridor should be interposed to secure cross-ventilation. There are three chief types of water-closet—the pan-closet, the valve-closet, and the hopper-closet. Of these, I mention the first two only to condemn them. The hopper-closet differs from both in having no mechanical parts. It is simply a funnel terminating in a siphon—a good flush of water sweeping the contents away. An improved form of the hopper-closet is known as the "wash-out closet." It is often made of glazed stoneware cast in a single piece, and is cleansed after use simply by a good flush of water. The most modern closets have neither "safe" nor wood casing. The "safe" is a lead tray on the floor to prevent any overflow soaking through.

"Soil pipes," connecting the closet with the drain, should be constructed of lead, 4 inches in diameter, and run down *outside* the house, if feasible.

Each soil-pipe should be freely ventilated by being carried full bore, without curves or bends, to a few inches above the roof-ridge.

Under no circumstances should **drinking water** be drawn from a water-closet cistern or tank—indeed, each closet should be fitted with an automatic patent flushing tank of small size, used for the purpose of flushing alone; while drinking water should, in every instance, be taken **direct from the water-main.**

2. Whenever drains or cesspools are disturbed for purposes of cleansing, **chemical disinfectants** should be freely used, and the household should certainly vacate the house for the time being. The disinfectants in most request are:—carbolic acid (phenol), in solution, one part in 40 parts of water ($2\frac{1}{2}$ per cent.); copperas (or ferrous sulphate), 2 ozs. to one pint of water; Burnett's fluid (solution of chloride of zinc); chlorinated lime, commonly called

"chloride of lime;" Condy's fluid (a powerful oxidiser and deodoriser); chloralum; and *dry* charcoal. The three last named are disinfectants, but not antiseptics (see Chapter IV., page 31).

II. With the view of checking or **preventing the propagation of the poison** of enteric fever, the following means should in all cases be adopted:—

1. The **Excreta** should be **disinfected** as soon as they are discharged from the body. Both the freshly passed evacuations from the bowels and the urine should be mixed with a sufficient quantity of a solution of carbolic acid (1 in 40) or of ferrous sulphate, before they pass into the house drains or public sewers, or can permeate into the sources of drinking water. Carbolic acid is especially useful for this purpose because of its power of arresting fermentation.

For the **thorough disinfection** of fluid or semi-fluid stools, Prof. Uffelmann recommends the following methods: **Sulphuric**, or **hydrochloric acid**, diluted with double the quantity of water, should be mixed with equal parts of the fæcal matter, and allowed to stand, in the case of the former acid, for two hours; in the case of the latter, for twelve hours. In using **carbolic acid**, a 5 per cent. solution should be added to the stools in equal parts and left for twenty-four hours. **Corrosive sublimate** should be employed in a solution of 2 per 1,000, with half a part per 1,000 of hydrochloric acid. An equal quantity of this solution is added to the motions and set aside for at least half an hour, but preferably for twenty-four hours. **Quick-lime** ought to be added to the stools in the proportion of $2\frac{1}{2}$ parts per cent., and allowed to remain twenty-four hours. In the case of lime-water, a similar time should be allowed and the disinfectant should be employed in the proportion of $2\frac{1}{2}$ parts to one of fæces.

2. The **bedding** and **body-linen** from the patient should be **soaked** in carbolised water (4 ounces of carbolic acid to a gallon of water). It is a good plan, also, to **boil** or **bake** these possible fomites before they are sent to the laundry to be washed.

3. The **sick-room** should be **freely ventilated**—the purity of the air in the chamber may be tested by exposing solution of permanganate of potassium in saucers. The air is impure in direct

proportion to the rapidity with which the beautiful purple colour of this solution changes into a dirty rusty brown tint.

4. When **the first case** of enteric fever occurs in a given house, every effort should be made to **trace it back to its original cause.**

I am confident that the day is not far distant which will place in our hands an unfailing **weapon of defence** against the successful invasion of the body by the *Bacilli typhosi* of Eberth.

A careful perusal of the discussion on "**Immunity,**" which took place in the Section of Bacteriology at the Seventh International Congress of Hygiene and Demography, held in London, August 10th to 17th, 1891, will convince the reader that we stand on the threshold of a great discovery—the isolation of the substances which produce an acquired immunity in the case of each of the infective micro-parasitic diseases; and these, of course, include enteric fever.

In that discussion, Dr. Hans Buchner expressed the opinion that these protective substances were most probably albuminous bodies of very unstable constitution, and of a very complicated structure, which was specifically different in different cases. He proposed for these the name of "**alexins**" (Greek, *ἀλέξω, I ward off, defend*).

Mr. E. H. Hankin, B.A., Fellow of St. John's College, Cambridge, said that the discovery of the bacteria-killing power of blood serum had led to the view that immunity was caused by a bactericidal action exerted by the blood and lymph of an immune animal. This bactericidal power was due to the presence of certain **bacteria-killing substances,** to which the name "**defensive proteids**" had been given. These are the "**alexins**" of Buchner. They are divided into "**sozins**"[a] and "**phylaxins.**"[b] Sozins are defensive proteids present naturally in the normal animal. Phylaxins are present in animals which have artificially been made immune against a disease.

The view of immunity suggested by the discovery of these defensive proteids or alexins by no means excludes Metschnikoff's phagocyte theory, because they can be obtained from cells which were, or could become, phagocytes. (See Chapter III., page 22.)

[a] Greek: *σώζω, to save alive, to preserve.*

[b] Greek: *φύλαξ, a protector,* from *φυλάττω, to guard.*

CHAPTER XLV.

Curative Treatment of Enteric Fever.

No Specific for Enteric Fever.—Principles of Treatment apply equally to this fever and to typhus. Curative Treatment or Management: Hygiene, Diet, Stimulants, Antiseptic Drugs:—Iodide of potassium, calomel, arsenic, antimony, β-naphthol, salicylate of bismuth, salicylate of magnesium, carbolic acid, the sulphites, turpentine, free chlorine, quinine, oil of eucalyptus, camphor, creasote, thymol, naphthalin, salol.

"There is **no specific** for enteric fever," writes Murchison, "any more than for typhus." But he adds: "Although we **cannot cure** the disease, we **must treat** it." While deprecating the *nimia diligentia medici*, by depletion on the one hand, or by over-stimulation on the other, he says that it must not be thought that the best treatment is one of mere expectancy. He quotes, however, with approval Baglivi's[a] remark on "mesenteric fever" made two centuries ago—"Sed quod præ cæteris animadverto, in nullo morborum genere, tantâ opus est patientiâ, expectatione, cunctationeque, ad bene et feliciter medendum, tanquam ad bene curandum febres mesentericas."

The fact is that while the great principles of treatment of enteric fever are precisely the same as those which guide us in our management of typhus, there is no other disease which in the same degree taxes to the uttermost the resources of the physician. Of every "turn" in enteric fever, of every day or hour which marks its course, it may truly be said: "Latet anguis in herbâ." The attitude of the physician must be one of "armed expectancy," to borrow the expressive phrase given by Dujardin-Beaumetz[b] in 1889 to that form of symptomatic medication, which has also been described under the name of "the medication of indications."

The **Curative Treatment**, or, rather, the **Management** of Enteric Fever falls under certain convenient headings, such as—Hygiene, Diet, Stimulants, Antiseptic Drugs, Antipyretic Treatment, Treatment of Complications and Sequelæ, Management in Convalescence.

[a] *Opera Omina.* Romæ. 1696. Ed. Sext. Lugduni. 1704. Page 54.

[b] *Le Bulletin Médical.* Paris. Feb. 8, 1889; and Sajous' *Annual of the Universal Medical Sciences.* 1889. Vol. I., page H-55.

I. **Hygiene.**—The patient should take to bed, as soon as possible after symptoms show themselves, in a large, airy, quiet, well-warmed and well-ventilated room. Irreparable mischief is done by men struggling against their illness day after day, trying to "walk it off." In the Franco-German war of 1870–71, railroad travelling especially was proved to exert a very prostrating influence on enteric fever patients. Physicians, who should know better, when they are themselves attacked by this disease, are wont to fight against it and refuse to admit that they are sick. Liebermeister[a] has known physicians to make calls during the morning, when the evening before they had themselves seen that their own temperature was 104° F. in the axilla.

Not only should the patient take to bed as early as possible, but he should not be allowed to sit up again until the evening temperature has been perfectly normal for from three to six days at least. The hygiene of the sick room has been discussed at pages 44 and 49, and it only remains to emphasise the necessity for free ventilation. Even in winter, a window should be kept open during a great part of the twenty-four hours, at all events in the adjoining room to that occupied by the patient. "Even a strong draught of air, for a time, is harmless," for, adds Liebermeister,[b] "a patient with a high fever temperature cannot take cold."

Attention to personal hygiene is of the first importance in enteric fever. The bed and body-linen should be changed as often as they are soiled. The body of the patient should be kept scrupulously clean. The whole surface should be carefully sponged with vinegar (1 part) and tepid water (3 parts) several times a day. Particular care should be paid to the state of the teeth. A good antiseptic and detergent tooth-paste is the following :—

℞ Pulv. Saponis Duri, gr. 60;
Acid. Carbolic. Purissimi Liquefacti, ʒ ss;
Olei Eucalypti, ʒi;
Cretæ Præcipitatæ, ad ℥i.
M. ft. dentifricium.

[a] Von Ziemssen's *Cyclop. of the Pract. of Med.* Vol. I., page 229. 1875.
[b] *Loc. cit.* Page 231.

Should erythema show over the nates, or sacrum, or great trochanters, the affected part should be relieved of pressure by a water-cushion or an air-cushion, and, as a routine practice in all cases, the surface should be gently sponged with spirit of camphor. In this way, and by paying attention to cleanliness, the occurrence of bed-sores may be averted.

II. Diet.—The **dietetic treatment** of enteric fever is of the **first importance.** There can be little doubt that many patients are over-fed rather than under-fed in this fever, with the result that symptoms are aggravated, and diarrhœa in particular is increased. Let us reflect for a moment on the diseased state of a not inconsiderable portion of the digestive tract, and it will at once be evident that no solid food is admissible even if the patient did not turn from it with loathing. But, further, the powers of digestion are seriously impaired, and the assimilation of food is well-nigh at a stand still. Our object, in the face of such conditions, should be to give the patient just so much nutriment as will keep him alive and enable him to combat the disease which is making such inroads on his strength.

To secure these ends, the food should be **liquid**, very **nourishing**, easily **assimilable**, and exhibited in **moderate quantities** at rather **short intervals.**

Von Ziemssen, in his lectures on the treatment of enteric fever, points out that no pure albuminous food and still less fatty food should be given. Frequent change and variety of flavour and consistence of food is desirable. Starch, dextrin, and sugar may be exhibited. Milk is the most complete mixture of albumen, fat, and carbohydrates, and is an excellent food in enteric fever. Von Ziemssen does not give more than one pint as a rule in 24 hours. Freshly expressed meat juice, consisting of serum, lymph, and blood, forms an acceptable and highly digestible food. Frozen meat-juice is also well borne. If eggs are given in clear meat broths, not more than three should be administered during 24 hours. Jellies prepared from fresh calves' feet with white wine, are refreshing and grateful.

According to Stromeyer, the best thing to give to enteric fever patients is oaten grits, cooked for three hours, and given without sugar (Liebermeister).

The nearest approach to an ideal food for a fever patient is milk. In making this statement I do not wish to be misunderstood. There are some patients who cannot tolerate a milk diet. In other cases, patients are, as it were, poisoned with milk given in excessive quantities. Here, as in all other things, we must judge each case on its own merits. The fact remains that in an overwhelming majority of instances, a milk diet is the best and safest for a fever patient.

It is scarcely ever necessary to exceed the amount of **one quart** of milk in the 24 hours—that is, 40 fluid ounces. This quantity should be given in divided doses at regular intervals of one, two, or three hours, either plain, or, preferably, mixed with warm water. If it passes through the bowels in the form of lumpy, undigested curd, it should be peptonised, with *liquor pancreaticus*, or Fairchild's *zymine* peptonising powders. Or, it may be boiled and mixed with lime-water in the proportion of three parts of milk to one part of lime-water. The "*liquor calcis saccharatus*" of the Pharmacopœia may be substituted for the lime-water in proportionate parts, the lime-water containing half a grain of CaO in the ounce, whereas the saccharated solution contains 7·11 grains. Four ounces of milk for a meal would give 10 feedings a day, at intervals of nearly 2½ hours. Some of the milk, however, may be given in freshly-made tea in the morning, and some of it may be given as junket, or rennet, or in the form of custard, or blancmange (made with isinglass and milk), or cream. In the earlier stages of the fever, the feeding should be chiefly by day; afterwards, the meals must be given by night as well as by day, unless the patient is in a wholesome sleep (not somnolence or coma).

Besides milk, **animal broths** may be allowed to the extent of **one pint** in the 24 hours. Four such broths may be mentioned—beef-tea, chicken broth, or chicken jelly, veal broth, and mutton broth. Of these, the first and last are laxative, and, therefore, should not be given when diarrhœa is present. Beef-tea is particularly valuable because of its stimulating power, especially upon the heart. It may be flavoured with a little tomato juice or a stick of celery, I have often found it a good plan to mix beef-tea and chicken jelly in equal parts, when a patient tired of either singly.

Also, towards convalescence, tapioca, sago, or well-boiled rice, may be added to the broth in moderate quantities. The laxative effect of broth is lessened by adding a little isinglass (gelatin) or arrow-root to it.

In very prostrate conditions, Liebig's beef-tea, prepared by macerating lean meat, chopped very fine, with hydrochloric acid, and a little salt, will be found a valuable food. A receipt for this preparation will be found at page 513 of Dr. F. W. Pavy's "Treatise on Food and Dietetics" (1874).

If it is desired to give **stimulants and food together**, a teaspoonful of brandy or whisky may be added to each teacupful of warmed milk, or the excellent egg and brandy mixture of the Pharmacopœia (mistura spiritus vini gallici) may be prescribed.

As a **beverage, pure water** may be taken without stint. All through the fever cold water is the most grateful drink. When the tongue is furred and dry, a teaspoonful of glycerine or of glycerine of borax may be added to each tumblerful of drinking water; or the tongue, gums, and mouth generally may occasionally be painted with a linctus like the following, which will prevent or check the formation of sordes:—

℞ Potassii chloratis, gr. 30;
Acid. Borici, gr. 20;
Glycerini,
Succi Limonis, āā ℥ss.
Fiat Linctus.

Other **diet drinks** or *tisanes*, for occasional use according to circumstances are:—Toast and water, barley-water flavoured with oil of lemon (lemon rind), red or black currant jelly in hot or cold water—very grateful where sore throat is present, or strained tamarind tea (Hilton Fagge). Ice may be sucked, but it sometimes parches the mouth and lips rather than affords relief. Von Ziemssen recommends thin oat- and barley-water, the flavour being varied by the addition of sugar, cinnamon, wine, &c.

Seltzer water, or other similar mineral waters, the mineral acids in water, with or without sugar, thin milk of almonds (mistura amygdalæ), and a thin decoction of parched rice (especially where

there is a good deal of diarrhœa) are added to the diet drinks by Liebermeister. In advanced stages of this fever, or where tympanites is a troublesome symptom, aërated waters are contra-indicated.

III. **Stimulants.**—The rules already laid down for the administration of alcohol in acute disease apply in general to enteric fever (see pages 58 and 310). On the whole, stimulants are not so much required in enteric fever as in typhus, because the patients are often younger, and the heart is less affected. But it is often necessary to prescribe them—never as a matter of routine, but in definite doses and at specified intervals, just like *any other medicine*. **Failure of the circulation, pulmonary congestion,** and **general depression** are the indications for the giving of stimulants. Brandy in milk, or diluted and sweetened, agrees best with young children. Burgundy, port, and good claret suit adults, and old people are benefited by whisky, brandy, or champagne. The presence of albuminous urine, and, still more, of other evidences of nephritis, is an indication for the very cautious administration of alcoholic stimulants.

IV. **Antiseptic Drugs.**—*As yet*, we possess **no specific** for the prevention or cure of enteric fever. At the same time, certain drugs appear to have a distinctly beneficial effect in the disease where judiciously and properly administered.

Passing by the treatment by means of **iodide of potassium**, advocated by Sauer in 1840, and revived by Magonty in 1859, and von Willebrand in 1866—the last-named using a solution of one part iodine, two parts iodide of potassium, and ten parts water, giving three or four drops in a glass of water every two hours, we find **calomel** in large, or small and repeated, doses recommended by many distinguished authorities, such as Lesser (1830), Traube, Wunderlich, Liebermeister, and von Ziemssen in Germany; and Bouchard in France. Liebermeister has given this remedy, with but few exceptions, to every case of the fever (about 800 in number) admitted before the ninth day of the disease, in three or four eight-grain doses during the first twenty-four hours of treatment. In his hands, the death-rate fell from 18·3 per cent. among 377 patients treated non-specifically to 11·7 per cent. among 223 patients put on the calomel treatment. Further, he found that the use of calomel

materially shortened the duration of the disease and diminished its intensity. Bouchard considers that general antisepsis is secured by mercurial preparations. During four days, only at the beginning of the fever, the patient should take daily twenty pills of 2 centigrammes of calomel (6 grains in all).

On November 16, 1883, I reported to the Medical Section of the Academy of Medicine in Ireland the case of a man, aged twenty, a grocer's assistant, who was admitted into Cork-street Fever Hospital on September 28, 1883, on the eighth day of a severe attack of enteric fever. The fever ran a course of four weeks, and was characterised by a succession of high temperatures during the first twenty days, ataxic symptoms, obstinate constipation, hypostatic congestion of the lungs, and right basic pneumonia. Furthermore, treatment of a decidedly active kind was employed, and apparently with marked benefit to the patient. On five separate occasions twenty grains of quinine were given as an antipyretic in two quickly succeeding doses of ten grains each; twice the wet "pack" was employed for two hours at a time to control the pyrexia; and on three occasions, at intervals of forty-eight hours, ten-grain doses of calomel, guarded with a grain of opium, were administered as an antiseptic aperient.

The administration of calomel in large doses in enteric fever is nothing new, and is much thought of in Germany at all events.[a] "After the accurate observations of Wunderlich," says Niemeyer,[b] "we can scarcely doubt that by this remedy we may, in some few cases, cut short the disease (according to Wunderlich one or two five-grain doses are enough), and that in the great majority of cases where this remedy is given during the first week, and before the occurrence of much diarrhœa, the course of the disease is rendered milder and shorter. The experience of Pfeufer's clinic, as well as my own, perfectly agrees with Wunderlich's. We shall not attempt to say whether the calomel has a favourable influence on the typhous intestinal disease by opposing the sloughing and

[a] Cf. Von Ziemssen's *Cyclopædia of the Practice of Medicine.* Vol. I., page 200. "Acute Infectious Diseases."

[b] *Loc. cit.* P. 647.

ulceration, and whether, consequently, we can expect benefit from it only in the first weeks of the disease, when these changes have not yet taken place."

Niemeyer's observations have recently gained additional weight in an unexpected and interesting manner. Dr. Wassiljeff some years ago studied experimentally, in Hoppe-Seyler's laboratory, the influence of calomel on fermentation and bacteria. He found that the presence of one part of calomel in from twenty to one hundred parts of fibrin or fat, did not interfere with the action of the unorganised ferments of the saliva, gastric juice, and pancreatic juice; but the calomel did prevent the formation of certain decomposition products—as indol, phenol, skatol, creasote, and hydrogen sulphide. The calomel also prevented butyric acid fermentation. The action of calomel upon bacteria or micrococci was next studied, according to the Buchholtz-Wernick method. It was ascertained that the drug destroyed these organisms and prevented the appearance of new ones. From this it seems that calomel destroys organised ferments, but is without effect upon the unorganised ones. So far, all the experiments had been outside the body. Now, experiments were made on three dogs. They were given one grain of calomel each, and after some hours were killed. The intestines were ligated in the upper part of the duodenum and in the lower part of the colon, and the entire contents were carefully examined. In no case could the putrefactive products, indol, skatol, hydrogen sulphide, &c., be detected. It would seem from this that much of the good which calomel is known to accomplish in various intestinal troubles is due to its aseptic properties.[a]

Remembering, then, these aseptic or antiseptic properties of calomel, I felt no small degree of confidence in prescribing it in combination with opium—itself an antiseptic—in a case where persistent constipation during an attack of enteric fever, characterised by very high temperatures, was only too likely to lead to the formation of decomposition products in a diseased intestine.

Other antiseptics, among very many which have been recom-

[a] *The Physician and Surgeon*, and *Dublin Journal of Medical Science* for October, 1883. Page 327.

mended, are—arsenic, by Dr. Frederick Kirkpatrick[a]; antimony, by Surgeon-Major Edward Lawrie,[b] Residency Surgeon, Hyderabad; β-naphthol, by Prof. Bouchard, of Paris; hydronaphthol, by Michell Clarke;[c] carbolic acid, by Dr. F. Sidney Gramshaw,[d] of Easingwold, Yorkshire; the sulphites, strongly recommended by Professor Polli, of Milan; and, above all, turpentine—an old-fashioned and most valuable remedy, free chlorine, and quinine.

Arsenic usually controls diarrhœa; it is a heart-medicine, and helps the patient to combat the adynamia of the fever. It may be given with acids in the form of the hydrochloric solution—1 to 5 minims for a dose thrice daily.

Surgeon-Major Lawrie gives the **antimony** as recommended by Dr. Kent Spender,[e] of Bath, in frequently repeated small doses—one-sixteenth of a grain of tartar emetic every hour or every two hours. He finds that it cuts enteric fever short "with such certainty that it almost appears doubtful whether the lesion of typhoid is specific, or is not rather incidental or adventitious."

Professor Bouchard[f] uses and recommends **β-naphthol**, reduced to a fine powder and mixed with **salicylate of bismuth.** One hundred and fifty grains of β-naphthol are mixed with 75 grains of salicylate of bismuth, and this is divided into thirty powders. From three to twelve of these are given in the 24 hours, enclosed in a wafer, and swallowed with the food. The β-naphthol, insoluble in water, glycerine, or alcohol, reaches the intestine in consequence, and there acts as a powerful disinfectant, certainly deodorising the evacuations (J. Burney Yeo).

Dr. Leroux leaves out the bismuth from this combination when diarrhœa is not urgent; and if, on the contrary, there is constipation, he substitutes for it **salicylate of magnesium** until the bowels are free. It was Huchard, of Paris, who first recommended this

[a] *Dubl. Journ. of Med. Science.* Vol. XCI., page 420. 1891.

[b] *Report on the Civil Medical Department of His Highness the Nizam's Government for* 1888 (1279, *Fasli*). Hyderabad. 1889. Page 18.

[c] The *Practitioner*, December, 1888, and *Journal de Méd. et de Chir.*

[d] The *Lancet*, June, 23, 1888.

[e] The *Practitioner*, March, 1885.

[f] *Thérapeutique des Maladies Infectueuses.* Paris. 1889.

salt instead of bismuth salicylate. The drug acts both as an antipyretic and as an antiseptic in doses of 50 to 100 grains daily. Its laxative action is not great, so that its use is not contraindicated in cases where diarrhœa is copious. The formula of the salt is as follows :—

$$\left(C_6H_4\begin{matrix}COO\\OH\end{matrix}\right)_2 Mg + 4\,H_2O.$$

It contains 74·6 per cent. of salicylic acid (*Pharm. Post*, March 18, 1888).

Dr. J. Michell Clarke, of Bristol, recommends **hydronaphthol** in the treatment of enteric fever. It is a grayish-white crystalline powder, with a slight iodine odour, of somewhat indefinite composition, but yielding on repeated crystallisation a substance corresponding to β–naphthol. Dr. Clarke finds that it agrees with patients who are on an exclusively milk diet, although with other diets it is very apt to disagree. He prescribes 2 or 3 grains in gelatin capsules, or simply suspended in milk, or in a keratin-coated pill. This dose may be given, when diarrhœa is present, every two hours for the first three to six doses; afterwards every three or four hours. For children under one year the dose is half a grain, older children may take half a grain to one grain every hour, every two hours, or less often.

Carbolic acid is ordered in a mixture, of which this is the formula :—

℞ Acid. Carbol. Purissimi (Calvert) Liquefacti, min. xii;
Tincturæ Iodi. (B. P.), min. xvi;
Tincturæ Aurantii Cort., ʒiss;
Syrupi Simplicis, ʒiii;
Aquæ, ad ʒviii.

Dose: One ounce every four hours for the first fortnight, or until the urgent symptoms yield, and then three times a day.

If vomiting is excited, the dose of carbolic acid should be reduced, and a small quantity of dilute nitro-hydrochloric acid should be added to the mixture. Dr. Gramshaw rarely noted carboluria as a result of the treatment, which causes diarrhœa to cease.

The **sulphites**, recommended by Polli, seemed to Murchison to excite diarrhœa in some cases, and not to be of much use. Twenty

grains of sulphite of sodium, or from 1 to 2 drachms of sulphurous acid, largely diluted, may be given every four hours. Dr. Wilks, of Ashford, in a paper published in the *Brit. Med. Journal*, so long ago as 1870, claimed for sulphurous acid that it arrested the further development of the poison of enteric fever, and was, in fact, an antidote.

Turpentine is an excellent diffusible stimulant, and an antiseptic of great value. It relieves chest complications, controls diarrhœa, checks meteorism, and stays intestinal hæmorrhage. To secure the last-named object, it may be prescribed with ergot, according to the formula already given at page 124. In other cases, and for other purposes, 5 to 10, 15 or 20, minims of rectified spirit of turpentine may be given every second or third hour in capsule, or *perle*, or mixture. Should albuminuria, or nephritis be present, turpentine must be administered with caution.

Years ago this remedy was given with good results in adynamic fevers in the Meath Hospital in the form of "Turpentine Punch." It was introduced by Mr. Parr, the apothecary to the hospital.

A good way of prescribing it is with spirit of nitrous ether as follows:—

℞ Spt. Terebinth. Rectificati (or Olei Terebinth.), ʒii;
Spt. Ætheris Nitrosi, ʒi;
Spt. Chloroformi, ʒii;
Misturæ Amygdalæ, ad ℥vi.
Misce. Ft. mist. *Signa:* "Half an ounce for a dose."

The Confection of Turpentine of the British Pharmacopœia may be prescribed in 60 grain doses with peppermint water; or the official oil of turpentine emulsified with yolk of egg may be given with spirit of chloroform and peppermint water. According to Dr. W. Whitla,[a] Dr. E. Nelson prescribes the drug thus:—

℞ Olei Terebinth, ʒiii;
Ætheris, ʒiii;
Syrupi Tolutani, ℥i;
Mucilaginis Acaciæ, ℥i;
Aquæ Menthæ Piperitæ, ad ℥viii.
M. ft. mist. *Signa;* "Half an ounce for a dose."

[a] *Elements of Pharmacy, Materia Medica, and Therapeutics.* By William Whitla, M.D., J.P. 1889. Page 520.

Professor Horatio C. Wood, of New York, bears eloquent testimony to the usefulness of oil of turpentine in enteric fever. He gives his uncle, Dr. George B. Wood, the credit of introducing this drug into the treatment of enteric fever. Professor Wood adopts the following formula:—

℞ Olei caryophylli, min. vi;
Olei Terebinthinæ, ʒiss;
Glycerini,
Mucilaginis acaciæ, āā, ʒss;
Syrupi et aquæ, āā ad ʒiii.

M. ft. mist. *Signa*: "A dessertspoonful to be given every two hours during the day."

In intestinal hæmorrhage, 20 minim doses of oil of turpentine every hour or every second hour is an efficient remedy.

Both Sir Thomas Watson and Murchison, and quite recently Burney Yeo,[a] highly recommend free chlorine as a useful antiseptic in this fever.

The liquor chlori of the Pharmacopœia may be prescribed in 20 minim doses with the mineral acids, or we may prepare a fresh solution of chlorine gas as recommended by Dr. Yeo.

"Into a twelve-ounce bottle put thirty grains of powdered potassic chlorate, and pour on it 40 minims of strong hydrochloric acid. Chlorine gas is at once rapidly liberated. Fit a cork into the mouth of the bottle, and keep it closed until it has become filled with the greenish yellow gas. Then pour water into the bottle, little by little, closing the bottle, and well shaking at each addition until the bottle is filled." . . . "To twelve ounces of this solution for an adult, I add twenty-four or thirty-six grains of quinine, and an ounce of syrup of orange peel, and I give an ounce every two, three, or four hours, according to the severity of the case."

Dr. Yeo says that this is pleasanter to take than the official liquor chlori. The tongue cleans quickly, and the fœtor of the evacuations subsides. Dr. Yeo thinks that we obtain not only an intestinal but also a general antisepsis by this treatment.

[a] *Treatment of Typhoid Fever.* London. 1891. Page 29.

Eberth, in experimenting with the *Bacillus typhosus*, found that quinine checked its culture. This observation enhanced the opinion long entertained that this substance was not only an antipyretic but an antiseptic in enteric fever. W. Vogt, of Berne, Liebermeister, of Tübingen, Bouchard, of Paris, and Clement Cleveland, of New York, all recommend that the system should be rapidly saturated with quinine early in the fever.

To adults Liebermeister usually gives from 22½ to 45 grains of the sulphate or hydrochlorate of quinine, in powders of 7½ grains each, *every ten minutes*. He does not repeat these large doses for 48 hours. Quinism is generally produced to an extreme degree.

Bouchard gives 30 grains every third evening, in four equal doses of 7½ grains each every half hour during the first fortnight. In the third week, the 30 grains are reduced to 22½ grains, and in the fourth week to 15 grains every third day.

Dr. Clement Cleveland[a] gives from five to ten grain doses every fifteen minutes for two hours. At other times he gives from 5 to 10 grains every two or three hours. When this treatment agrees, the patients seemed to him to recover more quickly and perfectly, and with fewer sequelæ. He was also impressed with the fact that the death-rate is much lower with the quinine treatment than with the expectant plan.

Professor Grancher, of Paris, gives a child, aged five, from 15 to 22½ grains, divided into three doses, every half hour at about 5 or 6 p.m. The child usually sleeps after these large doses. Dr. Grancher believes that quinine has a specific antiseptic action in enteric fever.

Burney Yeo, as already mentioned, gives moderate doses in combination with chlorine.

The foregoing by no means exhausts the list of antiseptics recommended in the treatment of enteric fever. Dr. Burney Yeo quotes the following additional remedies :—

Mr. Kesteven, of Brisbane, in the *Practitioner* of May, 1885, and of April, 1887, wrote in warm praise of the value of oil of eucalyptus in this disease. He gave it in 220 cases, many of whom

[a] *New York Medical Record.* Nov. 20th, 1886.

had a "bad start," and he had only four deaths. The dose employed was from 5 to 10 minims, in emulsion with mucilage every four hours. He combined with each dose half a drachm of sal volatile, half a drachm of spirit of chloroform, and half a drachm of glycerine.

Dr. Janeway, of New York, highly recommends **camphor**, not only as an excellent antiseptic, but also as a cardiac stimulant. In the *Dublin Journal of Medical Science* for November, 1879 (Vol. LXVIII, page 411), Mr. George B. White, F.R.C.S.I., reported a case of enteric fever of nine weeks' duration complicated with acute cerebral symptoms, phlegmasia dolens of the left leg, and rigors, in which a very large dose of camphor (more than 40 grains), given by mistake, was followed almost immediately by good results. In dispensing a mixture ordered to be compounded with camphor water, spirit of camphor was substituted, and the patient, a lady aged 21, swallowed 40 grains of camphor in nearly one ounce of rectified spirit.

Pécholier, of Montpellier, is an advocate for the administration of **creasote** both by the mouth and in enemata, while **thymol** has been given by Dr. F. Henry, in doses of a grain and a half, or two grains, made into a pill with soap, every six hours. The Italian physician, Testi,[a] has also given thymol in 150 cases of enteric fever with good results.

L. Wolff,[b] in a recent paper, speaks highly of naphthalin as an antiseptic remedy. His opinion is based on its employment in one hundred consecutive cases. This remedy was proposed by L. Natanson in a communication, entitled "La Naphtaline dans la Fièvre Typhoïde."[c]

Edgar Hirtz prescribes **salol**, in combination with salicylate of bismuth, in doses of 60 grains (4 grammes) a day. It appears to act like naphthol. The stools lose their fœtid odour, the tongue cleans rapidly. It is valuable from two points of view: it effects first an intestinal antisepsis, next an urinary antisepsis, for it is

[a] *Allgem. Wien. med. Zeitung.* No. 9, page 90. 1889.
[b] *Medical News.* Philadelphia. 1891. LVIII., pages 569–572.
[c] *Bulletin de la Soc. de Méd. prat. de Paris.* 1891. Pages 161–163.

broken up in the system into salicylic acid and carbolic acid, which are eliminated in the urine.[a]

A combination of β-naphthol with salicylic acid, known as the **salicylate of naphthol**, or briefly **betol**, has been in use for some time as a substitute for β-naphthol. The composition of this preparation is, however, not constant, and it sometimes injures diseased kidneys.

MM. Yvon and Berlioz, of Paris, have recently drawn attention[b] to the value as an intestinal antiseptic of benzoate of β-naphthol, or, as they term it, benzonaphthol. It is obtained by acting on β-naphthol with benzoyl chloride. When purified by repeated solution in and crystallisation from boiling alcohol, and, finally, by means of a dilute solution of caustic soda, it appears as a white crystalline powder without taste or smell. Its formula is $C_{10}H_7O$ (C_7H_5O). When introduced into the intestinal canal it breaks up into β-naphthol, which remains in the intestine, and benzoic acid, which passes off in the urine in combination with alkaline bases, or is converted in greater or less amount into alkaline hippurates.

Benzonaphthol should be given in small doses frequently repeated—4 to 8 grains in wafer paper, or suspended in syrup and water every three or four hours. An adult may take up to 5 grammes (75 grains), a child up to 2 grammes (30 grains) a day.

Although I have felt it a duty to bring forward the foregoing long list of drugs which are now employed in the treatment of enteric fever, I thoroughly agree with Dr. Hilton Fagge when he says that "at present the **most rational** and **successful treatment** of this, as of most other fevers, is to help the patient by **rest, suitable food**, and **good nursing**; to watch carefully and intelligently, and to interfere when complications arise but not before."

In fact, the attitude of the physician who is in attendance upon a patient in enteric fever should be that of **watchful, intelligent,** and **armed expectancy.**

[a] *La Pratique Journalière des Hôpitaux de Paris.* 1891. Page 166.
[b] *Le Progrès Médical*, Nov. 14, 1891, and *The Practitioner*, Dec., 1891.

CHAPTER XLVI.

Curative Treatment of Enteric Fever (*Continued*).

Antipyretic Treatment: (1). The Water-treatment (hydrotherapy, or balneotherapy), cold, cool, and warm baths; immersion treatment (Dr. James Barr)—Description of Apparatus—(2). Reduction of Temperature by means of the Ambient Air (de Souza).—(3). Antipyretic Drugs: Quinine, salicin, salicylic acid, the salicylates, salol, phenazone, acetanilide, kairin, thallin, digitalis, veratria, resorcin or thymic acid with acetanilide—Hilton Fagge's *Placebo*.

V. The **antipyretic treatment** of enteric fever is based on the clinical experience that a continuous and protracted high temperature inflicts grave injury upon the nervous system, the muscular tissues, and particularly the heart. It is not the mere height of the thermometer at a given moment which is harmful, but a temperature which is persistently above normal. A temperature ranging from 98° or 100° at one time of the day to 106° at another time will do less harm than a temperature which keeps continuously high at 102° or 103°. Another noteworthy fact is that children, as compared with adults, not only become feverish more easily and quickly, but also bear high temperatures much better. Hence, it is not necessary to take the same active steps to reduce pyrexia in the very young which we must adopt if adults are to be saved when stricken by fever.

(A.) Far in advance of all other means for reducing temperature in enteric fever stands the **water treatment.** The reasons for this pre-eminence are given in Cantani's Address on "Antipyresis," to the Tenth International Medical Congress at Berlin. To this classical oration reference has already been made at page 53, and in the same place the reader will find a full account of the different ways in which this method of treatment may be carried into effect.

First adopted by Currie, of Liverpool, in 1787, the cold water treatment of fever fell into abeyance for many years. Its revival

was due to Ernst Brand,[a] of Stettin (1861) and Jürgensen,[b] of Kiel (1866); and since the publication of the results obtained by these writers, this method of treatment has been widely adopted in Germany, France, America, Australia, and England. In 1880, Dr. Cayley, of the London Fever Hospital, discussed the subject at length in the Croonian Lectures, and in the third edition of Murchison's "Treatise on the Continued Fevers of Great Britain," he gives a full account of his practical experience of the cold bath treatment, both at the London Fever Hospital and at the Middlesex Hospital.

Dr. Hilton Fagge[c] describes the practical application of the water treatment very succinctly as follows:—

"When we have decided on bringing down the temperature, there are several ways of accomplishing it. One is to place the patient at once in a cold bath of 60° or 65° F. The shock may sometimes serve as a useful stimulus; but it is almost always better to use a tepid bath of 90° to 85° F., and rapidly cool the water with lumps of ice. The temperature is best watched by means of a thermometer *in recto*, and it must be remembered that it will most likely fall considerably after removal from the bath. Another important precaution is to give brandy immediately after, or even before, the bath, so as to stimulate the heart, and further the cutaneous circulation. Currie's original plan of cold affusion is best adapted to relieving headache and delirium with pyrexia by directing a douche upon the head. The practical difficulties of a bath in the case of adult patients, and the serious disadvantage of lifting and moving them—it may be many times in a few hours—speak strongly for applying cold to the surface as they lie in bed. For this purpose the 'wet pack' has been often used with good success. It is usually soothing and sedative as well as antipyretic, but it is less effectual than the bath, and sometimes is resented. Another plan is placing bladders of ice in the axilla and over the

[a] *Hydrotherapie des Typhus.* Stettin. 1861.

[b] *Klinische Studien über die Behandlung des Abdominal-typhus mittelst des kalten Wassers.* Leipzig. 1866.

[c] *Principles and Practice of Medicine.* Third Edition. 1891. Vol. I., page 172.

great vessels of the neck and thighs, or fixing a coil of Leiter's tubes in the same regions and feeding them from a receptacle of iced water placed over the patient's bed. But in many—perhaps in most—cases, the easiest and safest is also the most efficient method, namely, sponging the surface with cold water, or rubbing the trunk and limbs with pieces of ice, as the patient lies on a blanket with a waterproof sheet under it. Even when there does not appear any call for active interference, sponging the face, arms, and legs with cold water or spirit lotion, is grateful to the patients, and is often followed by tranquil sleep."

According to Prof. Paul Lefort,[a] Parisian physicians are now practically unanimous in recommending "**la Balneothérapie**" in enteric fever. He quotes, as joining in a chorus of approval of this method of treatment, Bouchard, Hayem, Millard, Chauffard, Gérin-Rose, Juhel-Rénoy, and Josias. Gérin-Rose advises that the patients, in addition to having baths at 30° C. (86° Fahr.), for twenty minutes at a time, four times a day, should drink as much [water] as possible; and Debove says: "Il ne suffit pas de le *laisser boire*, il faut le *faire boire*," adding: "Perhaps, indeed, if cold water baths have any beneficial effect, they owe it simply to the diuresis which they induce."

Josias is rapturous in his praise of cool baths, given at 18° C. (64·4° F.) every three hours and for 15 minutes at a time. These baths should be suspended only in cases of **intestinal hæmorrhages.** Menstruation, affections of the respiratory organs (bronchitis, pulmonary congestion, pneumonia, emphysema), or of the renal organs (albuminuria), offer no contra-indication to the employment of baths.

"Thanks to cold baths," he says, "a typhoid fever is no longer 'typhoid' except in name; patients thus treated are no longer prostrate, present no stupor, but remain lively and clear-headed; their tongues are moist, their thirst is extreme, which permits of an allowance of some 4 to 5 litres (7 to 9 pints) of liquids, nourishing or otherwise. Excessive diarrhœa and polyuria are observed; this diarrhœa, but particularly this polyuria, are such that the patient

[a] *La Pratique Journalière des Hôpitaux de Paris.* 1891. Pages 163, *et seq.*

may be considered as daily washing out his intestines and kidneys. But, in an infective disease like typhoid fever, such a flushing, carrying away all the waste of the organism, should not be regarded save as a positive advantage."

An interesting discussion on the treatment of enteric fever took place before the Berlin Medical Society in the Session of 1884–85.[a] Senator, in opening the discussion, observed that at the General Hospital at Hamburg, between 1874 and 1877, 937 cases were treated without cold baths and 568 by means of them, the mortality for the two systems of treatment being identical, viz., 7·2 per cent. In his opinion, as a mere antipyretic, phenazone (antipyrin) and such like drugs, act better than a cold bath, but the latter is most beneficial as a cutaneous stimulus.

Goltdammer, who advocated cold baths, stated that of 3,600 cases treated at the Bethaneum the mortality was 12·8 per cent., but that if the cases which were admitted later than the second week (of which the mortality was 36 per cent. and which, therefore, had no baths during the most important period) were excluded, the mortality fell to 9 per cent. The mortality of cases over forty years of age was 41 per cent.

During the years 1849–64 the typhoid mortality in the Second Army Corps at Stettin was 25·9 per cent.; after the introduction in 1865 of cold bathing it fell to 8 per cent. The mortality among the cases in the Prussian Army is 10 per cent., in the Austrian 26·8, in the Italian 28·36, and in the French 36·5 per cent. The expectant method, Goltdammer considered, gave very bad results.

Sajous,[b] who may be regarded as a recognised exponent of American medical opinion, writing in 1891, says that "the Brand treatment seems at last to be winning its long-deferred recognition as the method *par excellence* of managing enteric fever." He quotes the favourable opinion of the method expressed by Dr. F. E. Hare, Resident Medical Officer in the Brisbane Hospital, Australia, in two

[a] *Verhandlungen der Berliner medicinischen Gesellschaft*, 1884-85, and *London Medical Record*, December 15, 1886, page 557.

[b] Annual of the Universal Medical Sciences. 1891. Vol. I. H.-54.

communications on the subject. In the latter of these,[a] Dr. Hare compares the mortality in 1,828 cases during the period of expectant treatment (271 deaths, or 14·8 per cent.) with that in 171 cases during a period of incomplete bath-treatment (21 deaths, or 12·3 per cent.) and in 797 cases during the period of strict bath treatment (56 deaths, or 7·0 per cent.). In the last group, 15 of the fatal cases should be deducted. Excluding these, 782 cases gave a death-rate of 5·2 per cent.

In *The Lancet* for 1890 (Vol. I., page 690) Dr. James Barr, Physician to the Northern Hospital, Liverpool, speaks highly of prolonged immersion in what he calls the "**tank bath.**" He illustrates his communication with notes of a series of cases—nine in number—in which he employed this method. In addition to these, three other cases were treated by him in the same way. All the twelve patients recovered. They were immersed for periods varying from six to thirty-one days.

The "**tank**" used at the Northern Hospital, Liverpool, consists of a well-made wooden box 6ft. long, 2ft. 10 inches wide, and 12 inches deep. It is lined with lead, which is painted white, and coated with a thick layer of shellac varnish. The shellac makes the tank, on other occasions, a convenient medium for administering electric baths. Each tank is provided with a large discharge pipe, which in the case of these tanks communicates with a soil-pipe, which leads down to the sewer: the tank, containing 70 gallons of water, can thus be emptied in three minutes. To the tanks hot and cold water are plentifully supplied. Each tank is provided with a sheet of bed-ticking, which would almost allow the patient to be submerged, but at the head there is a strip, about a foot wide, which does not sink so deeply, and on which rests an air-pillow so as to keep the head above water.

The patient is wrapped up in a blanket and completely immersed, except the head. The tank is covered with a half lid, which prevents the weight of the bed-clothing resting on the patient, a waterproof sheet, and bed-clothing to keep in the heat of the water. A thermometer is kept constantly in the tank. As long as the

[a] *Australasian Medical Gazette.* July, 1889. Sydney.

patient's temperature is over 100°, the temperature of the tank need not rise above 90° to 93°; but as the body temperature approaches the normal, so also should the tank temperature. Dr. Barr did not find it necessary to lower the temperature of the water below 90° or to raise it above 98°. By regulating the heat of the water in the tank, there is no fear of any collapse, as the temperature of the body cannot fall below that of the surrounding medium.

Anxious that the patients while immersed should be as little disturbed as possible, Dr. Barr enjoined them to pass their urine and fæces into the tank. But he himself admits that there are grave objections to this, and that so far he has been unable to devise satisfactory means of rendering the water in the tank aseptic. Hence he thinks it will probably be best in most cases to raise the patient above the water while the bowels are being moved.

The patients subjected to the immersion treatment were kept chiefly on a **milk diet**, but Dr. Barr found that in the tank they digested boiled bread and milk very well, notwithstanding the opinion of Dr. Lauder Brunton that farinaceous food in enteric fever on several occasions seemed to him "to afford a more favourable nutrient medium to the bacilli." **None** of the patients **had any alcohol** during their residence in hospital. Also, they had **very little physic.** When there is constipation **calomel** in small doses appears to Dr. Barr to be perhaps the **best purgative. Naphthaline**, as recommended by Rosenbach, was prescribed to procure **intestinal antisepsis**, chiefly on account of its very slight solubility.

The **effects of the immersion treatment** were briefly the following:—

1. **Temperature.**—Dr. Barr considers that the tank effects a true antipyretic action, where the thermogenesis is diminished, the thermolysis regulated, and the thermotaxic mechanism is improved.

2. **Circulatory System.**—There is a marked improvement in the vaso-motor tone: the blood-vessels become smaller and firmer; the pulse slower, fuller, and of improved tension; the heart maintains its vigour. There was no intestinal hæmorrhage in any of the cases, and Dr. Barr is inclined to think that the tank lessens the liability to this complication, because, without doubt, the improved

vaso-motor tone extends to the abdominal vessels, as shown by the lessened diarrhœa, the diminution in the distension of the abdomen, and the rise in the arterial tension.

3. **Respiratory System.**—The rate of breathing lessens in frequency, the bronchitis and congestion of the lungs improve and soon disappear.

4. **Digestive System.**—The improvement in the digestive tract is, perhaps, more marked than anywhere else. The tongue becomes moist and clean, the salivary secretion increases, the appetite and digestion improve, and the diarrhœa not only lessens, but the character of the motions changes for the better.

5. **Nervous System.**—The delirium disappears, and the general well-being of the patient greatly improves.

6. **Skin**—The horny layers of the palms of the hands and soles of the feet get quite macerated; but on the skin of the body generally there is very little effect, with the exception of a slight roughness and elevation of the papillæ.

7. The **Tissues in general.**—There is marked diminution in dehydration of the tissues, which takes place in all febrile conditions. This is very apparent in the case of the tongue, which maintains its proper size and keeps moist.

While he advocates the treatment of enteric fever by baths, Dr. Cayley impartially discusses three chief objections which have been advanced to the adoption of this method of treatment. These are:—

First—It is dangerous to the patient, from its being likely to cause collapse, intestinal hæmorrhage, congestion, and inflammation of the lungs.

Secondly—The great mechanical difficulty in carrying it out.

Thirdly—The discomfort and pain it causes the patients, and their consequent repugnance to submit to it.

Dr. Cayley shows very clearly that the first of these objections has no foundation in fact. In 2,068 cases reported by Dr. Goltdammer, there was only one instance of fatal collapse. This accident can occur only when the treatment has been deferred until the later or adynamic stages of the fever, and may be obviated by

raising the temperature of the water in the bath. Dr. Barr also pointed this out. The frequency of intestinal hæmorrhage, again, is not increased by the bath, rather the reverse. At the same time, Cayley admits that, should hæmorrhage or peritonitis occur, the bathing must be at once intermitted, as perfect quiet is requisite.

With regard to lung complications, Dr. Cayley thinks it cannot be doubted but that, when the bathing treatment is begun early enough, it has a marked effect in preventing them.

The mechanical difficulties are. no doubt, hard to get over in private practice; but they are not insuperable, and in every properly equipped General or Private Hospital suitable apparatus for the purpose should be available. Dr. Cayley, in his edition of Murchison's "Treatise on the Continued Fevers," describes such an apparatus, constructed by Mr. Hawksley, the surgical instrument maker, of Oxford-street, London, and planned by Mr. E. A. Fardon, at the time Resident Medical Officer of the Middlesex Hospital.

It consists of two uprights, which move on wheels, and a cross-bar. A kind of hammock made of strips of webbing is placed under the patient, and then attached by a suspender to the cross-bar. By means of pulleys and an endless chain the hammock is lifted from the bed, then slid along the bar till it is over the bath, and let down into it and drawn up again in the same manner. Not only does this entirely relieve the nurses of any strain, but it is both safer and more comfortable for the patient. It takes, however, longer to bathe a patient in this way than by simply lifting him into the bath.

In reference to the possible repugnance of the patient to the bath, Cayley observes that with judicious management he has met with very little difficulty from this cause. In a large number of cases the relief given to the febrile oppression is so great that the bath is grateful; in other cases, though disagreeable, the patients are quite willing to submit in view of the after-relief. In all cases the temperature and duration of the bath must be adapted to the condition and feelings of the patient and the effect it produces. Other

means of reducing temperature must be employed where the bath is objected to or produces unfavourable symptoms.

(B.) Dr. A. de Souza[a] recommends lowering of the temperature in fever by means of the **ambient air**. This method of reducing bodily temperature is especially applicable, according to him, in enteric, hectic, and tubercular fevers. In winter he keeps the room in which the patients are from 8° to 10° C. (46·4° to 50° Fahr.), the only bed-covering being a sheet and one blanket; if towards the end of the night the temperature of the room or of the patients falls considerably, something extra is thrown over the feet. The higher the fever, the lower is the temperature of the room to be kept. If the air of the room be gradually cooled, the patients do not notice or soon become accustomed to the fresher atmosphere. In summer even, much may be done by proper ventilation and by diminishing the bed-clothes. Cold lotions and baths may also assist.

(C.) A third way of reducing temperature is by the **action of drugs**, called in consequence "**antipyretics.**" Never very partial to this method, I confess that since I read Cantani's Address on "Antipyresis," already quoted on more than one occasion in this book, I have become more chary than ever in the use of antipyretics in fever. The views of this enlightened physician have been stated at pages 7 and 53, and need not be recapitulated here.

At the same time, it is necessary to allude to some of the remedies included under the heading "Antipyretics."

1. **Quinine** deservedly occupies the first place in the list. It may be given in suspension in water, or in milk with 3 to 5 grains of camphor, in 5-grain doses every third hour, or in still larger doses (10 grains) every ten minutes until the desired quantity—20 to 40 grains—has been taken. This remedy is particularly useful in the later stages of the disease, when the fever has a remittent type. Children bear quinine well (Barthez and Rilliet, 1853).

2. **Salicin, salicylic acid,** the **salicylates,** and **salol** are not trustworthy antipyretics in enteric fever. Salicylate of sodium in repeated doses of 10 to 20 grains has, at the Meath Hospital, pro-

[a] *El Monitor Medico.* September, 1886.

duced toxic symptoms, with temporary depression of the heart's action and delirium. Albuminuria has also been observed elsewhere after its free administration.

3. The great group of **coal-tar derivatives** may next be considered. Of these, the best known is **phenazone (antipyrin)**, now official, but objectionable as an antipyretic in fever because of its depressant action. To children it should be given with the greatest caution. **Acetanilide (antifebrin)**, also official, is an excellent febrifuge, particularly when prescribed with quinine—3 grains of antifebrin and 2 grains of quinine, repeated every third or fourth hour, while necessary. **Kairin** ($C_{10}H_{13}NO$) was introduced into practice as an antipyretic by Dr. Wilhelm Filehne,[a] in 1883. He recommended doses of from 7 to 15 grains, repeated hourly until temperature falls. Its taste is horrible—saline, bitter, and persistently nauseous—so it should be prescribed either in pills, with glycerine of tragacanth, or in cachets (Martindale). It is efficient, but its effects are evanescent, and it is apt to depress the heart and cause collapse with cyanosis. **Thallin** (the sulphate of tetrahydroparamethyloxychinolin!) in 3 or 4 grain doses, reduces temperature readily; but this remedy, also, is not free from danger, and is contraindicated in cardiac weakness or kidney disease.

4. **Digitalis** in large doses has a powerful action in reducing temperature, and is highly recommended by Liebermeister,[b] Wunderlich,[c] Thomas, and other German physicians. Liebermeister prescribes it in powder or in pills, giving from 11 to 22 grains in 36 hours, and following it up by a "full dose" (30 to 45 grains) of quinine. "Its use in these large doses," says Cayley, "can hardly be regarded as quite free from danger." Even Liebermeister states that it is to be used only where there is no considerable degree of cardiac weakness, and where the pulse is not yet extremely frequent. He adds—hardly for our encouragement—"The impending paralysis of the heart is not prevented by the use of this drug, but seems rather to be favoured thereby.

[a] *Berlin klin. Wochensch.* 1883. Nos. 6, 16.

[b] Von Ziemssen's *Cyclopædia of Pract. Med.* 1875. Vol. I., page 217.

[c] *Ueber den Nutzen der Digitalisanwendung beim enterischen Typhus.* Archiv. der Heilkunde. 1862. Page 97.

5. **Veratria**, in comparatively large doses ($\frac{1}{12}$th of a grain in pill every second hour until decided nausea or vomiting ensues), was recommended by W. Vogt, and employed by Liebermeister. It is a remedy which will not commend itself to British physicians in the treatment of enteric fever.

6. The antiseptic and antipyretic methods of treatment have recently been combined by Dr. E. Tordeus, of Brussels,[a] who gives **resorcin** or **thymic acid** as an antiseptic together with acetanilide as an antipyretic. The dose usually employed by Dr. Tordeus was three-quarters of a grain of thymic acid and from one and a half to two grains of acetanilide, repeated not too frequently, say every two hours. Resorcin is best administered, according to Martindale, well diluted with water and flavoured with syrup of orange or glycerine. By the doses mentioned, the temperature was reduced in enteric fever from 104° to nearly normal, without any unfavourable symptoms accruing.

This long list of antipyretics may appropriately be closed with a quotation from Dr. Hilton Fagge. "It seems to be grateful to most patients," he observes, "to take what is called '**a simple febrifuge**,' such as ten drops of dilute hydrochloric acid in infusion of orange, of calumba, or of serpentary. It is probably a mere *placebo*, but there is every reason to please as well as to cure our patients."

[a] *Nouveau Traitement de la Fièvre Typhoïde. Journal de méd., chir., et pharmacologie.* Bruxelles. 1891. Vol. XCII., pages 325–332.

CHAPTER XLVII.

Curative Treatment of Enteric Fever (*Continued*).

Treatment of certain Complications and Sequelæ.

Strong Decoction of Coffee and Caffeïn in Ataxia and Adynamia.—Inhalation of Oxygen in Broncho-pneumonia.—Prevention of Bedsores.—Treatment of Epistaxis.—Treatment of Intestinal Symptoms, Complications, and Sequelæ: Constipation, diarrhœa, tympanites or meteorism, vomiting, abdominal pain, hæmorrhage from the bowels, peritonitis, perforation, medical and surgical measures.

VI. Little need be said in this Chapter about the treatment of the complications and sequelæ of enteric fever which engage the systems of the body other than the digestive tract. For the rules already laid down for the management of the cerebral and cardiac affections of typhus, the pulmonary affections of measles, and the anginal and renal affections of scarlatina, apply equally to those cases of enteric fever in which similar complications may happen to arise.

The great subject of the treatment of the intestinal complications which are almost peculiar to enteric fever, however, remains to be discussed.

Before I take up this topic, I would like to allude to a few troublesome complications and to certain remedies which have not hitherto been mentioned in connection with the special lesions to combat which their employment is indicated.

In well-marked **ataxia** and **adynamia** of fever, the administration of a strong decoction of **coffee** (*café noir*) is often followed by highly beneficial results. And, in accordance with its physiological action, the active principle of coffee called caffeïn is employed as a cerebral and cardiac stimulant, particularly in bad fevers. According to Huchard, of Paris, **caffeïn**, exhibited endermically, gives excellent results in ataxo-adynamic enteric fever. It is a general tonic in

adynamic states. Caffeïn is very soluble in aqueous solutions of benzoate, cinnamate, and salicylate of sodium. These dissolve it in chemically equivalent quantities. Martindale says that the following salicylate of sodium solution of it forms an unirritating hypodermic injection :—

℞ Caffeinæ, gr. 20 ;
Sodii Salicylatis, gr. 17½ ;
Aquæ destillatæ, ad ʒi.

Dose : 1 to 6 minims. This solution contains one grain in three minims.

Huchard employs two solutions—a weak and a strong. The formulæ for these are as follows :—

No. 1. *Weak*—

℞ Sodii Benzoatis, gr. 45 ;
Caffeinæ, gr. 30 ;
Aquæ destillatæ, ʒiss.

Make a solution by warming.

No. 2. *Strong*—

℞ Sodii salicylatis, gr. 47 ;
Caffeinæ, gr. 60 ;
Aquæ destillatæ, ʒiss.

Make a solution by warming.

Another remedy worth noting is **inhalation of oxygen.** In the "Medical and Surgical Reports of Cook County Hospital, 1890,"[a] will be found an account of inhalation of oxygen in a case of typhoid fever complicated by broncho-pneumonia.

Bedsores may be prevented by scrupulous cleanliness and the use of the water-bed. The parts should be kept dry and freely dusted with some emollient powder. Daily friction with spirit of camphor, eau de Cologne, whisky, or brandy, is a good preventive.

Epistaxis, or **nose-bleeding,** is sometimes so profuse and so obstinate as to call for special treatment. The drugs afterwards recommended in intestinal hæmorrhage may be prescribed. A bladder of ice should be applied over the forehead and nose ; while "hazeline" (an aqueous solution of hamamelis), infusion of matico

[a] Chicago. 1891. Pages 125–127.

or of rhatany, or a solution of alum or of tannin should be injected into the nostrils. Hot water injections may also be tried, and these measures failing, the posterior nares should be plugged.

The symptoms and complications connected with the digestive system in enteric fever which call for special treatment are the following:—(1). Constipation; (2). Diarrhœa; (3). Tympanites, or Meteorism; (4). Vomiting; (5). Abdominal Pain; (6). Hæmorrhage from the Bowels; (7). Peritonitis; (8). Perforation.

1. In treating **constipation**, we should never forget the advice tendered by Baglivi,[a] two centuries ago, that in the treatment of "Febris mesenterica maligna," all drastic purgatives are "**to be shunned like the plague.**" In the early stage of enteric fever, an experience of many years has satisfied me that a moderate dose of calomel (from 3 to 5 grains) is often beneficial, and effectually overcomes constipation. After the first week or ten days, calomel should be given with caution. Three other plans may then be tried either in rotation or alternately. First, diet may be so arranged as to act as a laxative—beef-tea, or strained mutton broth may be allowed; or ripe orange juice, or a few grapes carefully deprived of their skin and seeds. Secondly, a simple enema may be administered every thirty-six or forty-eight hours, according to circumstances. Trousseau used an enema of chamomile infusion. On no account, however, should a glycerine enema be ordered after the first few days of enteric fever—the increased peristalsis on which its efficacy depends might lead to dangerous consequences—inflammation, or perforation. Thirdly, an excellent remedy for constipation is castor oil in small doses, as both Trousseau and Murchison recommend. My favourite prescription is to mix a teaspoonful of glycerine with a wineglassful of warm milk, and then to add a teaspoonful of castor oil. This dose, or draught, may be repeated in six or eight hours, until the bowels are freed. It is a specially useful remedy in the constipation of convalescence.

Hilton Fagge discountenances any interference with constipation in enteric fever. He thinks it better to err on the side of

[a] Baglivi. Opera omnia. Romæ. 1696. Editio sexta. Lugduni. 1704.

caution, and to abstain from meddling with the bowels until convalescence is established.

2. **Diarrhœa.**—This is not only a common symptom, but sometimes a serious complication of enteric fever, and if unchecked may run down the patient's strength, and so endanger his life. Our object should be—not, as recommended by the late Dr. Todd, in 1860, to lock up the bowels, and keep them so; but to **check diarrhœa**, limiting the motions to two in the twenty-four hours, if possible.

An excellent remedy is a small starch enema, containing ten to twenty drops of laudanum. This stays peristaltic action and generally controls the milder forms of diarrhœa.

Much, too, may be effected by a change of diet. The milk should be boiled and mixed with lime-water in the proportion of 3, or only 2, parts of milk to 1 of lime-water. Dr. T. W. Grimshaw[a] used to employ the saccharated solution of lime in proportional doses—this preparation being about 14 times stronger than plain lime-water. Beef-tea should be withheld, and to each teacupful of chicken broth a teaspoonful of stiff isinglass jelly (gelatine) should be added. Thin arrowroot may also be allowed, or rice-milk. Above all, the quantity as well as the quality of the food should be controlled.

Local applications to the abdomen often do good. For example: light linseed meal poultices, turpentine epithems, mustard fomentations, and wet-compresses, may be tried in turn or singly. As regards poulticing in this fever, I long since gave up the application to the abdomen of bulky linseed meal poultices (which are positively dangerous from a mechanical point of view) in favour of the following: A piece of lint of suitable size is moistened with a warmed mixture of laudanum (one fluid drachm), glycerine (seven fluid drachms), and water (seven fluid ounces), and laid over the abdomen. Oiled silk or gutta-percha tissue is then applied, over which is placed a sheet of French wadding, or medicated cotton-wool—the whole being kept in position by a soft flannel roller or

[a] "On the Management of the Bowels in Enteric Fever." *Dubl. Journ. of Med. Science.* Vol. LXIII., page 132. 1877.

bandage. This "glycerine poultice," or "glycerine compress," as it may be called, is at once comfortable and efficient.

Trousseau[a] used to treat diarrhœa in *dothiénentérie* by ordering in the first instance a saline purge, a neutral salt, 25 to 30 grammes of sulphate of sodium or a seidlitz powder, as an alterative or modifier of the intestinal secretions. This treatment he considered to be especially applicable when the diarrhœa was accompanied by a certain degree of meteorism. If the desired result was not obtained, he used to prescribe so-called "absorbent powders," a mixture of $7\frac{1}{2}$ grains of subnitrate of bismuth with an equal quantity of prepared chalk—this dose being repeated three, four, six, eight times in the twenty-four hours, or even oftener according to circumstances. He also frequently gave "la mixture anglaise," namely,

℞. Cretæ Præparatæ, ℥j ;
Syrupi Aurantii Corticis, ℥j ;
Aquæ, ℥iij.

Powdered calumba root in doses of from $7\frac{1}{2}$ to 15 grains was another favourite remedy with him. If these means failed, he had recourse to pills containing about one-tenth of a grain of nitrate of silver in washed bread-crumb (*mie de pain*). Of these one was to be taken every hour in the course of the day.

If the "**Mistura Cretæ**" of the British Pharmacopœia is prescribed, it should be ordered to be prepared afresh, as in my experience it is a mixture which does not keep well. Instead of ordering it I much prefer to prescribe a mixture containing aromatic chalk powder, with or without opium, compound tincture of chloroform, powdered gum acacia, and cinnamon water. "Chlorodyne," or its pharmacopœial equivalent—the tincture of chloroform and morphin—may be substituted in this mixture for the compound tincture of chloroform.

Murchison gives the following favourite prescription :—

℞. Acid. Sulphurici Aromat., min. xx ;
Liquor. Opii Sedativi (Battley) min. iij ;
Tincturæ Catechu, ℥ss ;
Aquæ Menthæ Piperitæ, ℥j.

[a] *Clinique Médicale de l'Hôtel Dieu de Paris.* 1865. Tome I., pages 258, 259.

M. ft. haustus. *Signa*: "The draught to be taken every third or sixth hour."

Other remedies employed by Murchison were—Vegetable charcoal in teaspoonful doses every fourth hour (especially useful in diarrhœa accompanied by tympanites); powders of equal parts of Dover's powder and gray powder; or acetate of lead in solution in doses of 2 or 3 grains every four or six hours, with or without one-twelfth of a grain of acetate of morphin.

Dr. Grimshaw gave dilute sulphuric acid in the proportion of 3 drachms in an eight-ounce mixture, of which an ounce was to be taken every three hours. Sometimes he added to the mixture small quantities of solution of morphin, or of tincture of opium. In more urgent cases he employed the lead and opium pills of the Pharmacopœia, in four-grain doses every fourth hour, with great benefit.

Dr. Samuel Fenwick[a] speaks highly of sulphate of copper—one quarter of a grain with one quarter of a grain of opium every three hours. Murchison has an alternative prescription, according to which a quarter of a grain of sulphate of copper may be given in solution with sulphuric acid, quinine, and a few drops of laudanum every four or six hours.

Liebermeister lets moderate diarrhœa alone; but in severe cases he administers opium in small and repeated doses, sometimes in combination with ipecacuanha or nux vomica, and sometimes with tannin, alum, or other astringents.

Twenty years ago, Dr. James Little asked Dr. Alfred Hudson to see a lady suffering from enteric fever in whom looseness of the bowels was the chief trouble, and he suggested a pill consisting of one-sixth of a grain of carbolic acid, one-sixth of a grain of extract of opium, and three grains of trisnitrate of bismuth, to be taken after each loose stool. Dr. Little informs me that he found this pill so useful that he has commonly used it since, employing during the past two years the salicylate of bismuth instead of the trisnitrate. As, however, it often happens that patients in enteric fever are too ill to swallow a pill, Dr. Little has, when this was the

[a] *Outlines of Medical Treatment.* Third Edition. London. 1891. Page 454.

case, given the same ingredients in a mixture of which the formula approximately is—

℞. Bismuthi Salicylatis, gr. 30;
Pulv. Acaciæ, ʒii;
Tinct. Opii, min. xviii;
Glycerini Carbolici, min. xxx;
Tinct. Lavand. Co., ʒiii;
Aquæ, ad ℥vi.

Signa: A sixth part two, three, or four times in 24 hours, as required.

I may state, as a matter of personal observation, that small doses of turpentine given systematically every few hours from an early stage of the fever seem to control the diarrhœa in a remarkable and satisfactory manner.

3. **Tympanites**, or **Meteorism.**—In the first place, I am strongly of opinion that the occurrence of this troublesome and often dangerous symptom may be prevented by the turpentine treatment, commenced early and persevered in. No heroic doses are required, but the remedy should be given steadily through the fever. Dr. George B. Wood,[a] of America, long ago recommended turpentine in doses of from 5 to 20 minims every hour or every second hour, in all cases of enteric fever accompanied by tympanites and a dry tongue. In atonic ulceration and protracted convalescence, with a dry, red, and smooth tongue, he regarded turpentine almost in the light of a specific.

Another remedy, which controls tympanites, is **nux vomica**, or its active alkaloid, **strychnin**. These keep up intestinal "tone" and check undue distension of the bowel. Some years ago I was called to the Curragh in consultation to see a young officer ill of enteric fever with much tympanites. I advised 3 minim doses of liquor strychninæ to be exhibited three times a day, because his heart was weak, and with a view of controlling tympanites. A few days afterwards intestinal hæmorrhage occurred, whereupon my treatment was, I believe, somewhat unfavourably criticised.

[a] *Practice of Medicine*. Second Edition. 1849. Vol. I., page 328.

But surely both intestinal hæmorrhage and perforation would be more likely caused by over-distension of a diseased intestine than by any increased peristalsis due to moderate doses of nux vomica or of strychnin.

As the gas collects c efly in the colon, Murchison recommends enemata containing carbolic acid glycerine (half a drachm), or six minims of creasote with half an ounce of glycerine, or an ounce of vegetable charcoal with two ounces of mucilage, or two drachms of spirit of turpentine with two ounces of olive oil, or two drachms of asafœtida with 10 minims of oil of rue—any one of the foregoing in a pint and a half of barley water (decoctum hordei). Of these formulæ, the only one I object to is that containing charcoal, for this substance loses its absorbent properties when saturated with fluid, and so is practically inert and therefore useless.

The abdomen should be *gently* rubbed in the direction of the colon. Turpentine fomentations may be applied from time to time, or cold compresses as recommended by Liebermeister. Dr. Peter,[a] of La Pitié, Paris, found ice poultices—made by scattering small fragments of ice over a thick layer of dry linseed meal—most effectual for subduing the tympanitic distension. As the ice thaws, a poultice is formed which, in consequence of the slow melting of the ice, is kept at the temperature of melting ice. To Professor Monneret belongs the credit of devising and introducing to the profession this very valuable method of treating tympanites.

In Dr. Cayley's opinion,[b] the application of ice to the surface of the abdomen is by far the best way of treating great distension. It may be conveniently applied by putting small pieces between two folds of flannel. *It is especially indicated in hæmorrhage.* By this means a considerable general reduction of temperature may be effected.

Should tympanites not yield to the foregoing measures, O'Beirne's long enema tube should be cautiously passed up the rectum, when flatus will probably escape freely through it owing to reflex contraction of the bowel and increased peristaltic action. Puncturing

[a] *Brit. Med Journal.* Vol. II., page 450. 1869.
[b] Murchison. 1884. Third Edition. Page 673.

the colon with a capillary trochar and cannula has been suggested, but such a procedure is not to be recommended.

4. **Vomiting.**—The clinical significance of this symptom has been already discussed (see page 376). In all cases where vomiting occurs, inquiry should be made as to the quality and quantity of the food, and as to the times and frequency of feeding. Attention to these details may cause vomiting to cease without further trouble.

At the beginning of enteric fever, Murchison states that vomiting is often relieved by an **emetic** of ipecacuanha (20 grains) and antimony (one grain), or of carbonate of ammonia (40 grains). An emetic should **never** be administered after the tenth day of enteric fever.

Other remedies are—a sinapism or turpentine epithem to the pit of the stomach. Ice may be sucked—the patient being enjoined to swallow small fragments of it while still unmelted. The patient should take milk and lime-water in equal parts. Effervescing draughts or a mixture containing bismuth and dilute hydrocyanic acid should be prescribed. A hypodermic injection of 3 to 5 minim of liquor strychninæ is often of use. So also is "ingluvin" in 5-grain doses.

5. **Abdominal Pain.**—This troublesome symptom is often independent of peritonitis. It is best treated by warm fomentations, gentle massage, the application of a light poultice or compress, with a dessertspoonful of laudanum sprinkled over it. Occasionally a single leech applied to the right iliac region gives great relief. Murchison says that from 2 to 6 leeches may be applied in this way in a young and robust patient, early in the fever.

6. **Hæmorrhage from the Bowels.**—This alarming occurrence, if profuse in an advanced stage of the disease, indicates that the patient is in extreme danger.

Should hæmorrhage occur, the treatment by baths must be intermitted. Complete rest must be insisted upon, and a hypodermic injection of morphin should be given. Ice should be sucked, and an ice-poultice, or a bladder containing broken pieces of ice, should be laid over the right side of the abdomen (Murchison).

Patients sometimes object to the ice-poultice, and then of course it must be removed.

As for internal remedies, those in most request are—for acute hæmophilia, perchloride or pernitrate of iron in large doses; for local intestinal hæmorrhage, gallic or tannic acid, turpentine, rhatany, hamamelis, acetate of lead, opium, and ergot. During many years Murchison found the following mixture almost invariably effectual for stopping the bleeding:—

℞. Acidi Tannici, gr. 10;
Tincturæ Opii, min. x;
Spt. Terebinthinæ, min. xv;
Mucilaginis, ʒii;
Tinct. Chloroformi Comp., min. xx;
Aquæ Menthæ Piperitæ, ad ʒi.

M. ft. haustus, secundâ quâque horâ sumendus.

Dr. J. B. Russell,[a] of Glasgow, gives the tincture of ergot in drachm doses every hour, and has never known it fail. Murchison also speaks highly of ergot, which he considers to be "a most efficacious styptic, even in the most profuse hæmorrhage." From 3 to 5 grains of "ergotin" dissolved in 10 minims of distilled water, or in equal parts of glycerine and rectified spirit, may be injected beneath the skin. The drug may also be exhibited in combination with turpentine, in the form prescribed at page 124.

J. H. Jenkins,[b] in the *Medical World*, Philadelphia, for September, 1890, reports a case of repeated profuse intestinal hæmorrhages in the fourth week of enteric fever, in which one quart of blood was transfused directly from the common carotid artery of a lamb into the brachial vein of the patient at the bend of the elbow. Immediate improvement was noticed, and sixteen days after the operation the patient was able to sit up.

When the hæmorrhage is so profuse as to endanger life, von Ziemssen[c] recommends enemata of ice-water and injections of blood or of salt-water.

[a] *Glasgow Med. Journal.* May, 1869.
[b] Sajous' *Annual of the Univ. Med. Sciences.* 1891. Vol. I. H.-54.
[c] *Loc. cit.* 1888. Vol. I., page 261.

Opinions are not unanimously in favour of ergot as a styptic in hæmorrhage from the bowels. Dr. James Little, who was probably the first physician in Ireland to recommend it, has long since abandoned its use; and Dr. Walter G. Smith also considers that its efficacy is much over-estimated and its employment is of questionable advantage.

All authorities are agreed that the first and chief indication in hæmorrhage is **absolute rest**, and this is secured by withholding food for some hours, and by the free exhibition of opium, internally or as a hypodermic injection of morphin.

7. **Peritonitis.**—When this serious complication is localised, much benefit often is derived from the application of even a single leech over the ileo-cæcal valve.

In all forms of peritonitis the great object to be secured is absolute rest, and to secure this **opium** must be given with a free hand. It is our "**sheet-anchor**" in both peritonitis and perforation. To an adult two grains of solid opium may be given at once on the supervention of symptoms of peritonitis, and this may be followed by a grain every second or third hour, until slight stupor ensues. Nothing is more remarkable than the *tolerance* of opium displayed by the unhappy subject of peritonitis. Murchison states that as much as 60 grains has been taken in three days with benefit. In one case at Cork-street Fever Hospital, I gave quite a young girl no less than 13 grains in 24 hours without any sign of narcotism. In some of these cases, part of the opium may, no doubt, be rejected by vomiting. When the stomach is irritable, hypodermic injections of morphin should be substituted for doses of opium by the mouth. Murchison insisted that the opium should be given alone, and not in combination with calomel, which brings down more bile into the lower bowel and so excites peristaltic action. By far the best plan is simply to give a tiny pilule consisting solely of one grain of opium.

The accompanying pain and tension of the abdomen must be relieved by warm fomentations, bran poultices, and turpentine epithems; or, still better, by the use of the ice-bladder or ice-poultice.

The only food and stimulant should be a tablespoonful of milk and of iced brandy and water, given alternately every half hour.

Should the patient's life be spared, we must beware of interfering with the constipation induced by the opium. Far better to leave the bowels to themselves than to run the terrible risk of setting up another, and probably fatal, attack of peritonitis. "No harm will be done," says Liebermeister, "if a week, or even two or three weeks, pass without any movement of the bowels. Not until later, when all evidences of peritonitis have disappeared, will it be permissible to use a small lukewarm enema for the purpose of emptying the rectum of the hard fæcal matters it often contains."

8. **Perforation.**—Although all treatment in this deadly accident is only a forlorn hope, we are bound to save life if possible, and so the measures just recommended in peritonitis should be carried out. In addition, when the symptoms of perforation are followed by great abdominal distension, E. Friedrich,[a] Stein,[b] and others have recommended that the intestinal and peritoneal gases should be drawn off by **paracentesis.**

Shall **laparotomy** *be performed for intestinal perforation in enteric fever?* This question was answered in the affirmative by Leyden, of Berlin, on May 24, 1884.

At the Annual Meeting of the American Surgical Association, 1888, Dr. J. Ewing Mears,[c] of Philadelphia, read a paper on the **surgical treatment** of perforation of the intestine in typhoid fever. He had carefully searched current medical literature up to date and found only **four cases** of surgical interference in perforating typhoid ulcer, in all of which a fatal result had, unfortunately, occurred. The cases were operated on by Prof. Kussmaul, of Strassburg; Mr. T. H. Bartleet, of Birmingham; Dr. R. B. Bontecou, of Troy, N. Y.; and Dr. T. G. Morton, of Philadelphia. The following propositions and suggestions were offered:—1. Surgical interference

[a] *Prag. Vierteljahrs. für prakt. Heilkunde.* 1868. c. 11.

[b] *Deutsch. Arch. für klin. Med.* 1869. VI., page 454.

[c] *Philadelphia Medical News.* September 29, 1889, page 358; and Braithwaite's *Retrospect of Medicine.* Vol. XCIX., page 24. January–June, 1889.

is not justifiable and should not be instituted in cases of typhoid fever in which perforation occurs when the infective process is at its height. 2. In mild cases of the disease in which the pyrexia has not been of high grade, and in which perforation occurs at the end of the third week, or later when the stage of convalescence is fully pronounced, laparotomy may be performed. Surgical interference in cases of this character is advocated with the hope that if the method of operation suggested by Lücke—laparotomy with the creation of an artificial anus—be adhered to, success may be attained. 3. Rapidity of operation will be an essential factor in the achievement of success, through which prolonged exposure of the cavity will be avoided and shock greatly lessened.

The **statistics** of the operation are as follows:—

1884 (Sept.)—Mikulicz: 4 cases, with one recovery, though, unfortunately, the diagnosis is doubtful.

1885.—Lücke: resection; death.

1886.—Escher: 1 case; recovery; but the case is regarded by Louis as one of appendicitis.

1886.—Greig Smith: 1 case; doubtful diagnosis; death.

1886.—Bartleet: 1 case; death.

1887.—Bontecou: 1 case: death.

1887.—Morton: 1 case; death.

1889.—Bontecou: 1 case; death.

1889.—Senn: volvulus and perforation, 1 case; death.

1889.—Hahn: 2 cases; death.

1890.—Kimura: 1 case; death.

To these fifteen cases are to be added that of Taylor, 1891, and three reported in the *Medical News* for Saturday, November 21, 1891, by Weller Van Hook, A.B., M.D., Professor of Surgical Pathology and Bacteriology, College of Physicians and Surgeons; Professor of Surgery, Chicago Post-Graduate Medical College; Surgeon to Cook County and Chicago Charity Hospitals. One of Dr. Van Hook's cases recovered, making in all **19 recorded laparotomies, with 4 recoveries.**

Dr. Van Hook, in his paper, to which I owe many of the foregoing particulars, arrives at the following conclusions:—

1. There is no rational treatment for perforation in the course of typhoid fever, except laparotomy.

2. The indication for laparotomy when perforation occurs in typhoid fever is imperative.

3. The only contra-indication is a moribund condition of the patient.

4. Collapse is often at least temporarily relievable by hot peritoneal flushing.

5. The stage of the fever is not to be considered as an indication, or as a contra-indication, for laparotomy.

6. The severity of the typhoid fever alone is not a contra-indication.

7. Early laparotomy offers the most hope.

8. The symptoms of peritonitis should not be awaited before operating.

9. In taking charge of all typhoid fever patients, it is the physician's duty to be ready, in case of perforation, to perform laparotomy.

10. The published statistics of laparotomy for this condition are strongly in favour of operation.

11. The *technique*, though not complicated, demands much thoughtfulness, acquired dexterity, great rapidity, and thoroughness.

Viewing the hopelessness of treating perforation successfully by either medical or surgical methods, we may well say "Prevention is better than cure." "Recollect," said Sir Dominic Corrigan,[a] "with these ulcers or diseased follicles, how thin a layer of serous membrane literally stands as the barrier between life and death in this disease, whether the death result from hæmorrhage or from perforation; and that a purgative or an article of diet injudiciously exhibited may at any moment cause the one or the other. There is, perhaps, no other disease in which recovery, and even life, depend so much on attention to details of treatment, and on steadiness in withholding officious interference, no less than in judicious management."

[a] *Lectures on the Nature and Treatment of Fever.* 1853. Lect. X. Page 99.

CHAPTER XLVIII.

The Curative Treatment of Enteric Fever—*Concluded.*

Management in Convalescence.

Unwonted Exertion, exposure to cold, indiscretion in diet are all to be avoided.—Treatment of: Constipation, atonic diarrhœa.—Reduce stimulants day by day.—Solid Food: anecdote narrated by Dr. Stokes.—Rules for giving Solid Food: Fish, boiled or broiled—not fried.—Dietaries recommended by Hilton Fagge, Murchison, von Ziemssen, Niemeyer, F. Woodbury, Hutchinson, Lauder Brunton.—Medicines in Convalescence.—Change of Air.—Prolonged Rest.

In probably no other acute disease—scarlatina and rheumatic fever not even excepted—is there more need than in enteric fever for continued watching and skilful handling on the part of the physician far on into the period of convalescence.

Each step in the management of the patient in his progress towards health demands most anxious consideration, and should be taken only at the instance, or with the sanction, of the physician.

Long after the febrile movement of enteric fever has drawn to a close, unwonted exertion, exposure to cold, or indiscretion in diet may again place life in jeopardy. Hence it is that the time for leaving the sick-bed, and then the sick-chamber, for going out of doors, and for taking solid food, must all be arranged *for* the patient—not *by* the patient.

Remember how indefinite is the duration of the process known as cicatrisation. No one can tell in a given case to what an extent Peyer's patches are involved, and again—as we have seen—cicatrisation may be completed in one portion of the bowel ere it has well-nigh begun in another. Under these circumstances, it is obvious that the strictest caution should be observed in allowing a convalescent from enteric fever to exert himself, in the administration of aperients, and in ordering a change of diet.

In the first place, evening observations on the temperature should be continued for at least two weeks after convalescence has commenced. In this way, we can usually gauge with approximate certainty the favourable or unfavourable nature of the patient's progress; while timely warning is given that the diet is disagreeing or that a relapse is impending.

Constipation, again, is frequently most troublesome even after attacks in which diarrhœa has been a prominent symptom. It may sometimes be overcome by varying the food, or the times at which food is given. In other cases, it will be sufficient to give the glycerine and castor oil draught already mentioned (see page 463), or to administer a simple enema every day or every second day. Another *safe* remedy is the **sipping** of a tumblerful of cold, luke-warm, or hot water in the morning fasting. Apart from its diluent and laxative effects, water sipped in this way stimulates both the heart and the liver. Dr. Lauder Brunton, F.R.S., in his very interesting work on "Disorders of Digestion,"[a] says that Kronecker has discovered that the act of swallowing seems to remove entirely the inhibitory action of the vagus upon the heart, for the time being, so that the pulse becomes exceedingly rapid. The extent to which this occurs will hardly be credited by anyone who has not tried the experiment. In Dr. Brunton's own case, he found that sipping half a wineglassful of water would raise his pulse from 76 to considerably over 100. So that, in fact, **a glass of cold water**, slowly sipped, **will stimulate the heart** as much as, or more than, a glass of brandy swallowed at a draught.

Dr. Brunton proceeds to show[b] that sipping not only has this powerful effect upon the heart, but that it has also a similar effect upon **the liver**. It has been shown by Zawilski[c] that water, slowly sipped, not only increases the amount of bile secreted, but causes it to be secreted under higher pressure, so that if any slight obstruction should be present in the bile-ducts, it will be overcome, and the bile will flow freely into the bowel.

[a] London: Macmillan & Co. 1886. Page 11.
[b] *Loc. cit.* Page 73.
[c] Sitzungsbericht der wiener Akad. 1877. Mat. nat. Abtg. Bd. IV., p. 73.

When, on the other hand, **diarrhœa** persists through convalescence, the patient should be kept in bed. Murchison advises that this atonic diarrhœa should be treated by astringents and tonics such as acetate of lead, sulphate of copper, or nitrate of silver. At the same time he draws attention, with approval, to a remark of Trousseau[a], that when there is great emaciation, especially in cases which have been treated on too lowering principles, vomiting and purging during convalescence may be of a purely nervous character, and sometimes are at once relieved by solid food.

Hilton Fagge wisely observes that "**stimulants** should be at once **diminished** on the subsidence of fever, and in many cases it is desirable to substitute an ounce or two of wine twice or thrice a day for brandy at frequent intervals. With young patients, however, after a favourable attack there is often no need for stimulants or drugs."

I consider that this is sound advice. A grave responsibility rests on a physician who does not **daily revise** his patients' allowance of stimulants when recovering from acute illness. There can be no doubt that habits of intemperance in too many instances date back to a time when a good-natured or a neglectful physician allowed his patient to remain day after day or week after week on an allowance of wine or spirit, for the giving of which there was no warrant.

Another rule to be stringently carried out is, that on no account should alcoholic stimulants be taken **without food**—it may be only a biscuit. I hold that most fever convalescents are better without alcoholic stimulants—they have had poison enough in their bodies—but, if stimulants are given, they should be prescribed as medicine and not *à la carte blanche*.

All modern authorities are agreed that the greatest caution must be exercised in permitting the typhoid convalescent to partake of **solid food**, and particularly butcher's meat. Stokes, indeed, tells a graphic and amusing story, in his "Lectures on Fever,"[b] of a lady,

[a] *Clinique Méd. de l'Hôtel Dieu de Paris.* 1865. Tome I., page 264. See above, page 399.

[b] Edition of 1874. Pages 332, *et seq.*

recently married, who was violently delirious on the 12th or 13th day of "extremely severe petechial fever"—that is, be it noted, typhus, not typhoid—and in her delirium craved for "a rump of beef and cabbage," which were being cooked in the kitchen, and the odour of which filled the house. Her sister, who was nursing her, believing she was dying, resolved to gratify her wish from the feeling that it was right to accede to the request of a dying person. She proceeded to the kitchen, and brought up a large mess, steaming hot, to the lady's bedside, when the patient devoured it with great avidity. Shortly afterwards her husband came in, and was told what had happened. He became terrified, and sent for physicians in every direction. Four or five assembled, each of whom had his own suggestion to offer. At length the late Dr. Harvey, then Physician-General, joined in the consultation. On proceeding to the lady's bedside, the consultant-in-chief, Dr. Harvey, found the patient asleep. She was reported to him to be comatose. His advice was: "*I*'d let her sleep it out." She did sleep it out, and in the course of some hours awoke much better. Her recovery was perfect. Dr. Stokes, in narrating this anecdote, was careful to show that he did not do so with the object of inducing physicians to feed their patients with salt beef and cabbage in fever.

"**No solid food,**" says Hilton Fagge, "should be given **for a fortnight** after fever and diarrhœa have ceased. The patient will bitterly complain of the restriction; but if the physician has once lost a patient from perforation during convalescence he will be inexorable ever after. Progress to health after this disease must be slow if it is to be sure."

To the same effect Murchison writes: "Notwithstanding the cravings of the patient's appetite, the diet must be at first restricted to such articles as milk, eggs, farinacea, custards, light puddings, beef-tea, chicken-tea, or calf's foot jelly. **Meat** ought **not to be allowed for at least seven days** after the cessation of pyrexia, and not even then if there be any signs of intestinal disturbance; and before meat is given it is well to try for a day or two a piece of boiled sole, smelt, or whiting."

In reference to the last-mentioned recommendation, I may state that a fish diet does not always agree with a fever convalescent. Indeed, it sometimes appears to produce poisonous effects, perhaps owing to the formation of one of the alkaloids produced by the decomposition of proteid substances, to which Professor Selmi, of Bologna, gave the name of ptomaïns, by which they are now known. The result may be a recrudescence of the pyrexia. I have known this to occur in more than one case after enteric fever—the fish eaten being a sole apparently in good condition. "Of all fish," writes F. W. Pavy,[a] "the **whiting** may be regarded as the most delicate, tender, easy of digestion, and least likely to disagree with a weak stomach. It is sometimes styled the chicken of the fish tribe. The haddock is somewhat closely allied, but has a firmer texture, and is inferior in flavour and digestibility. The **sole** is a tender and digestible fish. It also has a delicate flavour, and deservedly enjoys a high reputation as an article of food for the invalid. The **flounder** is light and easy of digestion, but insipid. In all cases where fish is required for a weak stomach, **either boiling or broiling** should constitute the process of cooking. Frying is objectionable on account of the fatty matter used rendering the fish rich and more indigestible."

Von Ziemssen, in his "Lectures on the Treatment of Typhoid Fever," to which reference has before been made, expresses the opinion that the diet of convalescence should follow the usual diet of health with certain modifications. The return to solid food should be as follows:—First breakfast: tea, with biscuit and one soft-boiled egg. Second breakfast: 100 grammes (3½ ozs.) of finely minced raw bacon with wheat bread crumbs. Noon: 150 grammes (5¼ ozs.) of pigeon, young chicken, or partridge finely minced in soup. Later on this may be given in combination with a mild sauce and mashed potato, wine or beer being taken as a beverage.

In the afternoon: tea with biscuit or cakes. For supper: "mush,"—that is, Indian corn porridge—and milk, two soft eggs and some raw bacon. This may soon be followed by calves' feet for breakfast, then an English broiled beefsteak, mutton and pre-

[a] *A Treatise on Food and Dietetics.* 1874. Page 156.

serves, and in the evening some strengthening soup and some beer. At this stage, when the patient thinks of nothing but eating, something new in the way of food must be given daily.

"It is best," says Niemeyer,[a] "to let the patient eat frequently, but only a little food at a time, so that the slight amount of gastric juice secreted by the convalescents may suffice for its complete digestion. All indigestible food, which forms large amounts of fæces, should be strictly forbidden. An insignificant indigestion, a moderate diarrhœa, or slight vomiting, should be regarded as a very dangerous occurrence, because it may induce perforation of an ulcer that has not yet cicatrised."

F. Woodbury[b] considers the different preparations of coca especially valuable in convalescence from enteric fever, being superior to digitalis, for instance, in not having a tendency to cause diarrhœa.

Hutchinson[c] finds a too early return to solid food prejudicial to the patient. He continues the exclusive milk diet for three or four days after complete defervescence, or adds only animal broths. Then he gives soft-boiled eggs, the juice of underdone meat, with milk toast and other farinaceous articles. At the end of a week the soft parts of oysters and fish are added to the dietary; at the end of ten days, the light meat of broiled chicken; at the end of two weeks, butcher's meat. All these articles of food are given in small quantities at first.

I close this subject of diet in convalescence after enteric fever with a quotation from Dr. Lauder Brunton's Lettsomian Lectures on "Disorders of Digestion," delivered before the Medical Society of London in January and February, 1885:—"The palate," he says, "like the appetite, sometimes makes demands which are apt to be misconstrued. As the late Professor Laycock observed, patients recovering from a severe illness not unfrequently have a strong desire for salt herrings, pork, or ham, things which would be almost certain to disagree with them if their appetite were

[a] *A Text-book of Practical Medicine.* Revised Edition. 1880. Vol. II., page 655.

[b] Sajous' *Annual of the Universal Medical Sciences.* 1890. Vol. I. H.-52.

[c] *Loc. cit.*

indulged. But the fact is that the patients do not want the pork or herring; what they really desire is salt, and they crave for these articles because they contain salt. If salt be given to them in the form of a mixture, their appetite is appeased, and the harm is avoided which the herring or ham might have caused."

Few, if any, medicines are required in convalescence. But if the patient's state remains unsatisfactory—if his progress towards recovery "hangs fire"—when, in a word, convalescence is slow, Murchison recommends "quinine, the mineral acids, iron, cod-oil, and change of air." The last is, to my mind, by far the most important of all—particularly, should the patient have passed through his illness in the house which gave it to him owing to structural faults and defective drainage. Some years ago a young lady, almost dying of asthenia and anæmia after a protracted typhoid fever, was—by the advice of one of the first consulting physicians in Dublin—sent away in a carriage from one of the squares in Dublin only as far as the suburb of Sandymount, two miles distant. The result was that she immediately began to improve, and ultimately made a rapid and complete recovery.

Lastly, several months should be allowed to elapse before the convalescent resumes his usual avocations—at least, if they are such as to put a strain upon either his bodily or his mental powers. It often takes six months or even a year before these are thoroughly restored. "There is no disease," observes Hilton Fagge, "not even rheumatic fever, in which it is so important for the patient to have a long period to recover his strength before returning to his ordinary duties."

Should these rules be carried out, one would be justified in anticipating for the convalescent a "new lease of life," with a braced and invigorated muscular system, and a healthy, clear, and active brain.

CHAPTER XLIX.

Infection and Immunity.

Klemperer and Klemperer's researches on Infection and Immunity.—Case of acute fibrinous pneumonia.—*Pneumotoxins* and *anti-pneumotoxins*.—Dr. A. C. Abbott's conclusions.

I have stated, on page 22, that the question of **immunity** is still undecided. The most recent and valuable contribution to the literature of the subject is a paper by Klemperer and Klemperer, in Nos. 34 and 35 of the *Berliner klin. Wochenschrift* for 1891, on the nature of the infection in acute fibrinous pneumonia. For the following abstract of their researches I am indebted to an excellent review (by Dr. A. C. Abbott, First Assistant in the Department of Hygiene of the University of Pennsylvania[a]), of the experimental work in connection with immunity and infection carried on during the past few years. This review was contributed to the number of the *Practitioner* for December, 1891.

Klemperer and Klemperer's experiments bore entirely upon the nature of the infection in **acute fibrinous pneumonia** and have shed much light upon some of the obscure features of that disease. They found but little difficulty in affording immunity to animals that are otherwise susceptible to the pathogenic action of the organisms concerned in the production of this disease,[b] by the introduction into their tissues of the products of growth of the organisms from which the latter had been separated. The immunity thus produced is seen in some cases to last as long as six months; again it is seen to disappear suddenly in a way not to be explained. It was seen in one case to be hereditary.

[a] See also *Medical News*, Philadelphia, November 7, 1891.

[b] Animals do not, as a rule, present the pneumonic changes seen in human beings. The introduction of the diplococcus of pneumonia into their tissues results, in the case of susceptible animals, in the production of septicæmia.

The energy of the substance which has the power of affording immunity was seen to be very much increased by subjecting it to temperatures somewhat higher than that at which it was produced by the bacteria—37·5° C. (99·4° F.) Klemperer and Klemperer found that if this substance was heated to a temperature of from 41° to 42° C. (105·8° to 107·6° F.) for three or four days, or to 60° C. (140° F.) for from one or two hours, intravenous injection was followed by complete immunity in from three to four days; whereas, if the unwarmed material was used, immunity did not appear until fourteen days, and then only after the employment of relatively large amounts. Moreover, when the previously heated products are introduced into the circulation of the animal, the systemic reaction is of but short duration, but if the unwarmed substance is employed, immunity is manifest only after the appearance of considerable elevation of temperature, which lasts for a long time.

In explanation of these differences, they suggest that, in the latter case, the high fever that is seen to occur in the animal may serve to replace the warming to which the product had not previously been subjected, and which is necessary before the products of the bacteria are in a position to bring about the condition of immunity. They claim that the bacterial products employed in producing immunity in this case are not, in reality, the immunity-affording substance, but that they are only the agents that bring about in the tissues of the animals alterations that result in the production of *another body* that protects the animal.

In support of this, their argument is that several days are necessary for the production of immunity by the introduction into the animal of the bacterial products; whereas, if the blood-serum of this animal, which is now protected, be introduced into the circulation of another animal, no such delay is seen, but instead, the animal is forthwith protected. In the former case the actual protecting body must first be manufactured by the tissues; whereas, in the second it is already prepared, and is introduced as such into the second animal.

The serum of immunified animals is not only capable of rendering

other animals immune, but moreover **possesses curative powers** when the disease is already in progress. The serum of immunified animals, when injected into the circulation of animals in which this form of infection was in progress, and in which there was a body-temperature of from 40·4° to 41° C., reduced this temperature to normal (37·5° C.) in twelve consecutive experiments during the first twenty-four hours following its employment.

Klemperer and Klemperer explain that the crisis seen in pneumonia in human beings occurs at the moment when the poisonous products, manufactured by the bacteria located in the lungs, are present in the circulation in amounts sufficient to call forth in the tissues the **reactive change** that results in the production of the **antidotal substance** which has the power of rendering the poisons inert.

At the time of the crisis in pneumonia the bacteria themselves are in no way affected. They remain in the lungs, and can be detected in full vigour and virulence, in the sputum of patients a long time after the disease is cured. They have lost none of their power of producing poisonous products, and still possess their original pathogenic relations toward susceptible animals. It is only after the crisis that their **poisons are neutralised** by this antidotal proteid which has been eliminated by the cells of the tissues, and as this occurs the systemic manifestations gradually disappear. Klemperer and Klemperer isolated, from the cultures of the diplococcus of pneumonia, a **proteid body** which is the agent concerned in producing the tissue-changes that result in the formation of the protecting substance. They likewise isolated from the serum of immunified animals a proteid which possesses the same powers as the serum itself—of affording immunity and of curing the disease. The poisonous bacterial product they propose to call **pneumotoxin**; the protecting body, **anti-pneumotoxin.**

After obtaining these results upon the lower animals they directed their attention to human beings, and found that by the subcutaneous application of the serum of immunified animals to patients suffering from acute fibrinous pneumonia the results were in the main promising. They found that while healthy individuals

and those suffering from other forms of disease presented no systemic reaction after the injection of the serum, in six cases of pneumonia in which the serum was employed there was a remarkable fall of temperature and slowing of the pulse within the first twelve hours after it was injected. In four of these cases the temperature fell to normal, but rose again after six hours. In two cases it fell to normal, and remained at that point.

It would appear from the results obtained by these two observers that immunity against this disease, and the processes concerned in its cure, are **of a chemical nature**, the active poisons of the organisms, the pneumotoxins, being neutralised by the tissue-products, the anti-pneumotoxins.

Results upon animals in general analogous to those of Klemperer and Klemperer have been obtained by Emmerich and Fowitzky.[a]

In the light of these experiments the hypothesis advanced by Buchner, that immunity was to be explained by reactive changes in the integral cells of the body, receives considerable support.

As a result of his very critical and comprehensive review of the modern literature of the subject, Dr. A. C. Abbott arrives at the following conclusions:—

"1. That, of the hypotheses that exist for the explanation of immunity, that which assumes acquired immunity to be due to **reactive changes on the part of the tissues** has received the greatest support.

"2. That immunity is most frequently seen to follow the introduction into the body of the **products of growth of bacteria** that in some way or other have been **modified.** This modification may be artificially produced from the products of virulent organisms and then introduced into the tissues of the animal; or the organisms themselves may be so treated that they are no longer virulent, so that when introduced into the body of the animal they eliminate poisons of a much less vigorous nature than is the case when they possess their full virulence.

"3. That immunity following the introduction of bacterial

[a] Emmerich and Fowitzky: *Münch. med. Wochenschr.*, 1891, No. 32.

products into the tissues is not the result of the permanent presence of these substances *per se* in the tissues, or to a tolerance acquired by the tissues to the poison, but is probably due to the formation in the tissues of another body that acts as an antidote to the poisonous substance.

"4. That this protective proteid that is eliminated by the cells of the tissues need not of necessity be antagonistic to the life of the organisms themselves, but in some cases must be looked upon more as an antidote to their poisonous products.

"5. That in the serum of the normal circulating blood of many animals there exists a body that is capable, outside of the body, of rendering inert bacteria that, if introduced into the body of the animal, would prove infective.

"6. That, in many instances, infection may be looked upon as a contest between the bacteria and the tissues, carried on on the part of the former by the aid of the poisonous products of their growth, and resisted by the latter through the agency of proteid bodies normally present in their integral cells.

"7. That when infection occurs it may be explained either by the excess of vigour of the bacterial products over the antidotal or protective proteids eliminated by the tissues, or to some cause that has interfered with the normal activity and production of these bodies by the tissues.

"8. That phagocytosis, though frequently seen, is not essential to the existence of immunity, but is more probably a secondary process; the bacteria being taken up by the leucocytes only after having been rendered inert through the normal germicidal activity of the serum of the blood and other fluids of the body."

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